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**DEPARTEMENT: MEDICINE AND SURGERY**

**COURSE: RENAL PHYSIOLOGY**

**COURSE CODE: PHS 303**

**QUESTION 1: Discuss the role of kidney in glucose homeostasis.**

**Answer:**

The kidneys synthesize glucose from amino acids and other precursors during prolonged fasting, a process called gluconeogenesis. The kidneys’ capacity to add glucose to the blood during prolonged periods of fasting rivals that of the liver. With chronic kidney disease or acute failure of the kidneys, these homeostatic functions are disrupted and severe abnormalities of body fluids volumes and composition rapidly occurs. With complete renal failure, enough potassium, acids, fluid, and other substances accumulate in the body to cause death within a few days, unless clinical interventions such as hemodialysis are initiated to restore, at least partially, the body fluid and electrolyte balances.

**QUESTION 2: Discuss the process of micturition.**

**Answer:**

Micturition is the process by which the urinary bladder empties when it becomes filled. This process involves two main steps; firstly, the bladder fills progressively until the tension in its walls rises above a threshold level. This tension elicits the second step, which is a nervous reflex called the micturition reflex that empties the bladder or, if this fail, at least causes a conscious desire to urinate. Although the micturition reflex is an autonomic spinal cord reflex, it can also be inhibited or facilitated by centers in the cerebral cortex or brain stem.

***Micturition reflex:*** As the bladder fills, many superimposed micturition contractions begin to appear. They are the result of a stretch reflex initiated by sensory stretch receptors in the bladder wall, especially by the receptors in the posterior urethra when this area begins to fill with urine at the higher bladder pressures. Sensory signals from the bladder stretch receptors are conducted to the sacral segments of the cord through the pelvic nerves and then reflexively back again to the bladder through the parasympathetic nerve fibers by way of these same nerves. When the bladder is only partially filled, these micturition contractions usually relax spontaneously after a fraction of a minute, the detrusor muscles stop contracting, and pressure falls back to the baseline. As the bladder continues to fill, the micturition reflexes become more frequent and cause greater contractions of the detrusor muscle. Once a micturition reflex begins, it is “self-regenerative.” That is, initial contraction of the bladder activates the stretch receptors to cause a greater increase in sensory impulses from the bladder and posterior urethra, which causes a further increase in reflex contraction of the bladder; thus, the cycle is repeated again and again until the bladder has reached a strong degree of contraction. Then, after a few seconds to more than a minute, the self regenerative reflex begins to fatigue and the regenerative cycle of the micturition reflex ceases, permitting the bladder to relax.

***Facilitation or inhibition of micturition by the brain***

Spinal centers for micturition are present in sacral and lumbar segment. But these spinal centers are regulated by higher centers. The higher centers, which control micturition, are of two types, inhibitory and facilitatory centers.

Inhibitory centers for micturition: centres in the midbrain and cerebral cortex inhibit the micturition by suppressing spinal micturition centers.

Facilitatory centers for micturition: centers in the Pons facilitate micturition via spinal centers. Some centers in cerebral cortex also facilitate micturition.

**QUESTION 3: Explain Juxtaglomerular apparatus.**

**Answer:**

Juxtaglomerular apparatus is a specialized organ situated near the glomerulus of each nephron. It is formed by three different structures which are: 1) Macula densa 2) Extraglomerular mesangial cells 3) Juxtaglomerular cells

1) ***Macula densa***: is the end potion of the thick ascending segment before it opens into distal convoluted tubule. It is situated between afferent and efferent arterioles of the same nephron. It is very close to afferent arteriole. Macula densa is formed by tightly packed cuboidal cells. It plays an important role in tubuloglomerular feedback mechanism which regulates the renal blood flow and glomerular filtration rate. Macula densa cells also secretes thromboxane A

Characteristic features of macula densa cells are:

i) They are not well adapted for reabsorption. ii) They are not innervated. iii) These cells are in direct contact with the mesangial cells and in close contact with the Juxtaglomerular cells. iv) They act as chemoreceptors and are stimulated by decreased NaCl concentration and thereby causing increased renin release.

2) ***Extraglomerular mesangial cells***: they are situated in the triangular region bound by the afferent arteriole, efferent arteriole and macula densa. These cells are also called agranular cells, lacis cells or Goormaghtigh cells. Extraglomerular mesanglial cells secrete prostaglandin and cytokines like interleukin-2 and tumor necrosis factor.

Characteristic features of these cells are:

i) They are in contact with both the macula densa cells (on one side) and Juxtaglomerular cells (on the other side). ii) Functionally, these cells possibly relay the signals from macula densa to the granular cells after modulating the signals. In this way, a decreased intraluminal Na+ load, Cl– load, or both, in the region of macula densa stimulates the Juxtaglomerular cells to secrete renin. iii) They also show granulation to secrete renin in conditions of extreme hyperactivity.

3) ***Juxtaglomerular cells***: are specialized smooth muscle cells situated in the wall of afferent arteriole just before it enters the Bowman’s capsule. These smooth muscle cells are mostly present in tunica media and tunica adventitia of the wall of the afferent arteriole. Juxtaglomerular cells are also called granular cells because of the presence of secretory granules in their cytoplasm.

Characteristic features of Juxtaglomerular cells are:

i) They have well-developed Golgi apparatus and endoplasmic reticulum, abundant mitochondria and ribosomes. ii) They synthesize, store and release an enzyme called renin. Renin is stored in the secretory granules of Juxtaglomerular cells and therefore, these are also called granular cells. iii) They act as baroreceptors (tension receptors) and respond to changes in the transmural pressure gradient between the afferent arterioles and the interstitium. iv) They are densely innervated by the sympathetic nerve fibres and release their renin content in response to the sympathetic discharge. v) As these cells act as vascular volume receptors, they monitor renal perfusion pressure and are stimulated by hypovolaemia or decreased renal perfusion pressure.

**QUESTION 4: Discuss the role of kidney in regulation of blood pressure.**

**Answer:**

Kidneys play an important role in the long-term regulation of arterial blood pressure. When blood pressure alters slowly in several days/months/years, the nervous mechanism adapts to the altered pressure and loses the sensitivity for the changes. It cannot regulate the pressure anymore. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long-term regulation. Kidneys regulate arterial blood pressure by two ways: 1) By regulation of Extracellular Fluid volume. 2) Through renin-angiotensin mechanism.

1) ***Regulation of Extracellular fluid volume***: when the blood pressure increases, kidneys excrete large amounts of water and salt, particularly sodium, by means of pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantity of water in urine because of increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure natriuresis is the excretion of large quantity of sodium in urine. Because of diuresis and natriuresis, there is a decrease in ECF volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When the blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood pressure and cardiac output, resulting in restoration of blood pressure.

***2) Renin-angiotensin mechanism***: when blood pressure and ECF volume decreases, renin secretion from kidneys is increased. It converts Angiotensinogen to Angiotensin I. Angiotensin I is converted to Angiotensin II by Angiotensin-converting enzyme produced by the lungs. Angiotensin II acts in 2 ways to restore the blood pressure:

i) It causes constriction of arterioles in the body so that the peripheral resistance is increased and blood pressure rises. In addition, angiotensin II causes constriction of afferent arterioles in kidney, so that glomerular filtration reduces. This results in retention of water and salts and this increases the ECF volume to normal level. Thus blood pressure is increased to the normal level.

ii) Simultaneously, angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases the reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water reabsorption, resulting in increased ECF volume and blood volume. It increases the blood pressure to normal level.

**QUESTION 5: Discuss the role of kidney in Calcium homeostasis.**

**Answer:**

Plasma calcium concentration is maintained within a narrow range (8.5-10.5 mg/dL) by the coordinated action of parathyroid hormone (PTH), calcitonin, and ionized calcium. The kidney plays a key role in this process by the regulation of calcium excretion. Approximately 10 percent (100 mg/day) of the ingested calcium is excreted in the urine. About 41 percent of the plasma calcium is bound to plasma proteins and is therefore not filtered by the glomerular capillaries. The remainder is combined with anions such as phosphate (9 percent) or ionized (50 percent) and filtered through the glomeruli into the renal tubules. Normally, the renal tubules reabsorb 99 percent of the filtered calcium, and about 100 mg/day are excreted in the urine. Approximately 90 percent of the calcium in the glomerular filtrate is reabsorbed in the proximal tubules, loops of Henle, and early distal tubules. In the late distal tubules and early collecting ducts, reabsorption of the remaining 10 percent is more variable, depending on the calcium ion concentration in the blood. When calcium concentration is low, this reabsorption is great, and thus almost no calcium is lost in the urine. Conversely, even a minute increase in blood calcium ion concentration above normal increases calcium excretion. Several factors are involved in the regulation of calcium in renal tubules. PTH and activated vitamin D enhance calcium reabsorption in the thick ascending limb, distal convoluted tubule and collecting tubule. Estrogen promotes calcium absorption in the distal convoluted tubule and collecting tubule. Acidosis contributes to hypercalciuria by reducing calcium reabsorption in the proximal tubule and distal tubule, and alkalosis vice versa. Diuretics like thiazide and furosemide also alter calcium absorption in the renal tubules; thiazide promotes calcium reabsorption and furosemide inhibits it. The most important factor controlling the reabsorption of calcium in the distal portions of the nephrons, and therefore controlling the rate of excretion, is Parathyroid hormone (PTH).