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QUESTION 1: DISCUSS THE ROLE OF KIDNEY IN GLUCOSE HOMEOSTASIS

The kidney reduces blood glucose level by reabsorption in proximal convoluted tubule of the nephron. The glucose is transported by secondary active transport (sodium cotransport) mechanism. Glucose and sodium bind to a common carrier protein in the luminal membrane of tubular epithelium and enter the cell. The carrier protein is called sodium-dependant glucose cotransporter 2 (SGLT2). From tubular cell glucose is transported into medullary interstitium by another carrier protein called glucose transporter 2 (GLUT2). The renal threshold for glucose is 180 mg/dL in venous blood. When the blood level reaches 180 mg/dL glucose is not reabsorbed completely and appears in urine.

The kidney increase blood glucose level through the process of gluconeogenesis. This is the synthesis of glucose from amino acids and other precursors during prolonged fasting. It aids the liver in this process.

QUESTION 2: THE PROCESS OF MICTURITION

The process is called micturition reflex. This reflex is caused by the stimulation of stretch receptors situated on the wall of urinary bladder and urethra. When about 300 to 400 mL of urine is collected in the bladder, intravesical pressure increases. This makes the bladder wall stretch resulting in stimulation of stretch receptors and generation of sensory impulses. Sensory impulses from the receptors reach the sacral segments of spinal cord via the sensory fibers of pelvic (parasympathetic) nerve. Motor impulses produced in spinal cord, travel through motor fibers of pelvic nerve towards bladder and internal sphincter to cause contraction of detrusor muscle and relaxation of internal sphincter. This makes the urine enter the urethra from the bladder. In the urethra, the stretch receptors here are stimulated and send sensory impulses to spinal cord via pelvic nerve fibers. Now the impulses generated from the spinal centers inhibit pudendal nerve. This causes the external sphincter to relax leading to micturition. The micturition reflex is described as self regenerative seeing as the initial contraction of bladder further activates the receptors to cause further increase in sensory impulses from the bladder and urethra. These impulses, in turn cause further increase in reflex contraction of bladder. The cycle continues repeatedly until the force of contraction of bladder

reaches the maximum and the urine is voided out completely. It should be noted that during micturition, the flow of urine is facilitated by the increase in the abdominal pressure due to the voluntary contraction of abdominal muscles.

Micturition can be summed up with the following steps moving from to to bottom:

1. Filling of the bladder and stimulation of stretch receptors
2. Sensory impulses pass via pelvic nerve
3. Sacral segment of spinal cord
4. Motor impulses pass via pelvic nerve
5. Contraction of detrusor muscle and relaxation of internal sphincter
6. Flow into urethra and stimulation of stretch receptors
7. Sensory impulses pass via pelvic nerve
8. Inhibition of pudendal nerve
9. Relaxation of external sphincter
10. Voiding of bladder

QUESTION 3: EXPLAIN THE JUXTAGLOMERULAR APPARATUS

The juxtaglomerular apparatus is found near the glomerulus of the nephron. It is formed by 3 different structures which include: the macula densa, extraglomerular mesangial cells and juxtaglomerular cells.

The macula densa is the end portion of thick ascending segment before it opens into distal convoluted tubule. It is formed by tightly packed cuboidal epithelial cells and it is found between afferent and efferent arterioles of the same nephron.

The extraglomerular mesangial cells, which can also be called agranular cells, lacis cells or Goormaghtigh cells, are situated in the triangular region bound by afferent arteriole, efferent arteriole and macula densa. There are also glomerular mesangial/glomerular mesangial cells found between the glomerular capillaries which play an important role in regulating the glomerular filtration by their contractile property.

The juxtaglomerular cells are specialized smooth muscle cells situated in the walls of the efferent and afferent arterioles just before the Bowman capsule. These smooth muscle cells are mostly present in tunica media and tunica adventitia of the wall of the arterioles. They are also called granular cells because of the presence of secretary granules in their cytoplasm. Juxtaglomerular cells form a thick cuff called polar cushion or polkissen around the afferent arteriole just before it enters the Bowman capsule.

The primary function of juxtaglomerular apparatus is the secretion of hormones. It also regulates the glomerular blood flow and glomerular filtration rate.

The juxtaglomerular apparatus secretes renin (by the juxtaglomerular cells) and prostaglandin (by the extraglomerular mesangial cells). Renin functions in reduction in arterial blood pressure, reduction in ECF volume, increases sympathetic activity. Prostaglandin decreases blood pressure by systemic vasodilation, diuresis and natriuresis.

The extraglomerular mesangial cells of juxtaglomerular apparatus secrete cytokines like interleukin-2 and tumor necrosis factor. Macula densa secretes thromboxane A2.

QUESTION 4: DISCUSS THE ROLE OF KIDNEY IN THE REGULATION OF BLOOD PRESSURE

When blood pressure alters slowly in several days/months/years, the nervous mechanism adapts to the altered pressure and looses the sensitivity for the changes. It cannot regulate the pressure any more. In such conditions, the renal mechanism operates efficiently to regulate the blood pressure. Therefore, it is called long-term regulation. The kidneys play an important role in the long-term regulation of arterial blood pressure either by regulating the volume of extracellular fluid or through renin-angiotensin mechanism.

Through regulation of extracellular fluid: 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 due to an increament in blood pressure. Pressure natriuresis is excretion of sodium in urine to to increased blood pressure. Even a slight increase in blood pressure doubles the water excretion. Pressure diuresis and natriuresis leads to decrease in extracellular fluid volume and blood volume, which in turn brings the arterial blood pressure back to normal level. When blood pressure decreases, the reabsorption of water from renal tubules is increased. This in turn, increases ECF volume, blood volume and cardiac output, resulting in restoration of blood pressure.

Through renin-angiotensin mechanism: When blood pressure and ECF volume decrease, renin secretion from kidneys is increased. It converts angiotensinogen into angiotensin I. This is converted into angiotensin II by angiotensin-converting enzyme. The angiotensin II acts in two ways to restore the blood pressure.

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 the kidneys, so that glomerular filtration reduces. This results in retention of water and salts, increases ECF volume to normal level. This in turn increases the blood pressure to normal level.

Angiotensin II stimulates the adrenal cortex to secrete aldosterone. This hormone increases reabsorption of sodium from renal tubules. Sodium reabsorption is followed by water

reabsorption followed by increase in ECF volume and blood volume. It increases the blood pressure to normal level.

QUESTION 5: DISCUSS THE ROLE OF THE KIDNEY IN CALCIUM HOMEOSTASIS

Calcitriol (1,25-dihydroxycholecalciferol) is a steroid hormone synthesized in the kidney. It is the activated form of vitamin D. Its production is stimulated when there is a drop in blod calcium level. Its main action is to increase the blood calcium level by acting on the small intestine to increase calcium absorption.