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17/MHS01/114

RENAL PHYSIOLOGY ASSIGNMENT.

1. DISCUSS THE ROLE OF KIDNEY IN GLUCOSE HOMEOSTATIS?

The kidney contributes to glucose homeostasis by filtering and reabsorbing glucose, gluconeogenesis during fasting and excretion when there is excess of glucose in the blood. Under normal conditions the kidney retrieves as much glucose as possible, rendering the urine virtually glucose free. But during periods of hypoglycaemia or hyperglycaemia the kidney undergoes processes to maintain homeostasis.

In a hypoglycaemic condition, probably during starvation or when the glucose level is just too low due to other factors the kidney would undergo gluconeogenesis to release glucose into the system.

In glucose reabsorption, the capacity of the glucose transporters which is known as the tubular maximum for glucose ranges from 260 to 350mg/min/1.73m^2 in healthy adults and children corresponds to plasm glucose level of approximately 200mg/dL. Once this threshold is reached and the transporters are unable to reabsorb all the glucose, glucosuria occurs. Glucosuria is mostly connected with hyperglycaemia, but there are special cases where glucosuria occurs before the threshold is reached. Glucosuria can also occur in cases of hyperfiltration (pregnancy).

2. DISCUSS THE PROCESS OF MICTURITION?

Micturition is the process of expelling urine from the urinary bladder. This act is also known as voiding of the bladder. It is a reflex process. However, in grown up children and adults it can be controlled voluntarily to some extent. The process of micturition is regulated by the nervous system and the muscles of the bladder and urethra. The urinary bladder can store around 350-400ml of urine before it expels it out.

 The muscles involved are the detrusor muscles of the urinary bladder and the internal and external urethral sphincters. The muscles of the abdomen also play a role by putting pressure on the bladder wall.

 Within the nervous system, the process is governed by the autonomous nervous system and the somatic system. Stimuli to the sympathetic nerve causes the detrusor muscle to relax and the internal sphincter to contract during filling of the bladder. Stimuli to the parasympathetic nerve causes the detrusor muscle to contract and the internal sphincter to contract during emptying of the bladder. Stimuli to the somatic nerve gives the external sphincter the ability to contract for voluntary control of micturition.

Once the urinary bladder reaches its maximum capacity, the stretch receptors in the walls of the bladder send an impulse through the pelvic nerve to the brain through the spinal cord. The micturition reflex is ultimately generated from the level of spinal cord after it receives reflexes from the pontine region of the brain. When the bladder receives signal from the brain the detrusor muscle contracts and the internal sphincter relaxes. When urine enters the urethra and the stretch receptors are stimulated, they send impulses through the pelvic nerve to the spinal cord which leads to the inhibition of the pudendal nerve which causes the relaxation of the internal sphincter.

There are higher centres which control micturition apart from the spinal centre; the inhibitory centre (in the midbrain and cerebral cortex) and facilitatory centre (pons and cerebral cortex).

3. EXPLAIN JUXTAGLOMERULAR APPARATUS?

The juxtaglomerular apparatus is a specialized structure formed by the distal convoluted tubule and the glomerular afferent arteriole. It is located near the vascular pole of the glomerulus and its main function is to regulate blood pressure and filtration rate of the glomerulus. The primary role of the juxtaglomerular apparatus is the secretion of hormones, these hormones include; Renin and Prostaglandin.

 Structures that make up the juxtaglomerular apparatus include;

a. Macula densa; this are modified region of tubular epithelium located in the wall of the distal convulated tubule (of the same nephron) at the point where the afferent arterioles enter the glomerulus and the efferent arteriole leaves it. Macula densa cells stimulate the juxtaglomerular cells to release renin when they detect a drop in sodium concentration in tubular fluid.

b. Extraglomerular mesangial cells; these are located in the junction between the afferent and efferent arterioles. These cells are also called agranular cells, lacis cells or goormaghtigh cells. They have a contractile property similar to vascular smooth muscles and thus play a role in regulating the glomerular filtration rate (GFR) by altering the vessel diameter.

c. Juxtaglomerular cells; these cells are also called granular cells, they synthesize, store and secret the enzyme renin. They are specialized smooth muscles cells mainly in the walls of the efferent arterioles that deliver blood to the glomerulus. Juxtaglomerular cells secrete renin in response to drop in pressure detected by stretch receptors in the vascular walls or when stimulated by macula densa cells.

4. DISCUSS THE ROLE OF KIDNEY IN REGULTION OF BLOOD PRESSURE?

Kidneys play a role an important role in the long-term regulation of arterial blood pressure by, regulating the volume of extracellular fluid and through renin-angiotensin metabolism.

Renin is a peptide with 340 amino acids. Along with angiotensin, renin forms the renin-angiotensin system which is a hormone system that plays an important role in the Maintenace of blood pressure. Renin is stimulated by; fall in the arterial blood pressure, reduction of the ECF volume, increased sympathetic activity and decreased load of sodium and chloride in macula densa.

When Renin is released into the blood, it acts on a specific plasma protein called angiotensinogen or renin substrate. By the activity of renin, the angiotensinogen is converted into a decapeptide called angiotensin I. Angiotensin I is converted into angiotensin II, which is an octapeptide by the activity of angiotensin-converting enzyme (ACE) secreted from the lungs. Angiotensin I is physiologically inactive and only acts as a precursor of angiotensin II.

Angiotensin II causes blood vessels to constrict and blood pressure to increase. Angiotensin II regulates glomerular filtration rate by two ways;

i. it constricts the efferent arteriole which causes decrease in filtration after an initial increase.

ii. it contracts the glomerular mesangial cells leading to decrease in surface area of glomerular capillaries and filtration.

Angiotensin II stimulates the release of hormone aldosterone in the adrenal glands, which causes the renal tubules to retain sodium, potassium, and fluids. If the renin-angiotensin system becomes overactive, consistently high blood pressure results. Over time uncontrolled high blood pressure can cause arteries around the kidneys to narrow, weaken or harden. In regulating high blood pressure, the rate of excreted sodium and water from the body is increased.

5. DISCUSS THE ROLE OF THE KIDNEY IN CALCIUM HOMEOSTATIS?

Kidneys play a role in the regulation of blood calcium level by activating 25-hydroxycholecalciferol into vitamin D. This hormone 25-hydroxycholecalciferol is formed from cholecalciferol, which is present in the skin and intestines. The cholecalciferol (vitamin D3) is then converted in the liver into 25-hydroxycholecalciferol. Vitamin D is necessary for the absorption of calcium from the intestine.

 Parathormone (PTH) secreted by the parathyroid glands maintains the blood calcium level within the critical range of 9 to 11mg/dL. On the kidney, the PTH increases the reabsorption of calcium from the renal tubules along with magnesium ions and hydrogen ions. PTH increases calcium intake mainly from the distal convulated tubules, vitamin D increases reabsorption in the collecting duct and calcitonin decreases reabsorption.

On the Gastrointestinal tract, PTH increases the absorption of calcium ions from the GI Tract indirectly, it increases the formation of 1,25-dihyroxycholecalciferol in the kidneys. This activated vitamin D acts on the intestinal epithelium and enhances absorption of calcium from intestine into the blood.

When blood calcium level increases, it inhibits the formation of 1,25-dihydroxycholecalciferol by either suppressing the conversion of 25-hydroxycholecalciferol into 1,25-dihydroxycholecalciferol or a decrease in the PTH secretion. Also 25-hydroxycholecalciferol is inhibited by itself as a feedback mechanism, i.e. an excess of 25-hydroxycholecalciferol in the blood plasma prevents more production of it.