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ASSIGNMENT

Questions: 1. Discuss the role of the kidney in glucose homeostasis.

2. Discuss the process of micturition.

3. Explain juxtaglomerular apparatus.

4. Discuss the role of the kidney in the regulation of blood pressure.

5. Discuss the role of kidney in calcium homeostasis.

1.

 The kidneys are essentially designed to filter large quantities of plasma, reabsorb substances that the body must conserve, and secrete substances that must be eliminated. These basic functions are critical to regulation of fluid and electrolyte balance, body fluid osmolality, acid-based balance, excretion of metabolic waste and foreign chemicals, arterial pressure, hormone secretion, and, glucose balance in the body.

 The maintenance of glucose homeostasis is crucial in preventing pathological consequences that may result from hyperglycemia or hypoglycemia. Chronically uncontrolled hyperglycemia leads to a higher risk of macrovascular and microvascular complications, such as cardiovascular disease, nephropathy, neuropathy, and retinopathy. Hypoglycemia, on the other hand, may lead to a myriad of central nervous system complications (e.g, confusion, behavioral changes, seizures, loss of consciousness, and even death), since the brain is the body’s largest consumer of glucose in the fasting or postabsorptive state.

Maintenance of glucose homeostasis by the kidneys is by several complementary physiologic processes which includes:

1. Glucose reabsorption
2. Gluconeogenesis
3. Glucose excretion

1. Glucose reabsorption: The kidneys contribute to glucose homeostasis by filtering and reabsorbing glucose. Under normal conditions, the kidneys retrieve as much glucose as possible, rendering the urine virtually glucose free. The glomeruli filter from plasma approximately 180 grams of D-glucose per day, all of which is reabsorbed through glucose transporter proteins that are present in cell membranes within the proximal tubules.

2. Gluconeogenesis: The kidney is considered as two separate organs; the renal medulla is characterized mainly by glucose utilization and the renal cortex is responsible for glucose release. The separation of these activities represents the consequence of differences in the distribution of numerous enzymes along the nephron. The cells in the renal medulla can use only glucose for their needs (like the brain) and they have enzymes capable of glucose-phosphorylation and glycolysis. They can therefore phosphorylate important amounts of glucose and accumulate glycogen but, because these cells do not have glucose-6-phosphatase or any other gluconeogenic enzymes, they are unable to release glucose into the bloodstream. Moreover, the cells in the renal cortex have gluconeogenic enzymes and they can produce and release glucose into the circulation. However these cells cannot synthesize glycogen because they have little phosphorylating capacity.

2.

Micturition is the process of discharging urine from the urinary bladder. Micturition (urination) is needed to eliminate organic waste products, which are produced as a result of cell metabolism in the body.

 When the volume of urine in the bladder reaches about 250ml, stretch receptors in the bladder walls are stimulated and excite sensory parasympathetic fibres which relay information to the sacral area of the spine. This information is integrated in the spine and relayed to two different sets of neurons. Parasympathetic motor neurons are excited and act to contract the detrusor muscles in the bladder so that bladder pressure increases and the internal sphincter opens. At the same time, somatic motor neurons supplying the external sphincter via the pudendal nerve are inhibited, allowing the external sphincter to open and urine to flow out, assisted by gravity.

Control of micturition

Children and adults have considerable control over when and where they pass urine. They can also increase or decrease the rate of flow and even stop and start again. This control is learnt in infancy and involves other sensory fibres in the bladder wall. These fibres convey information on the degree of bladder fullness via the spine to the higher centres of the brain, the thalamus and cerebral cortex. This causes us to become aware that we need to pass urine and of the urgency of the situation. These links between the spine and cerebral cortex are not established until about two years of age and it is suggested that toilet-training is therefore not physiologically possible until that time.

 The brain is able to override the micturition reflex by inhibiting the parasympathetic motor nerve fibres to the bladder and reinforcing contraction of the external sphincter. The internal sphincter will not open until the external sphincter does. The increase in bladder volume increases stretch receptor and nerve activity, making the sensation of pressure more acute. When it is convenient, the brain centres remove the inhibition and permit micturition under our conscious control. When the bladder contains about 500ml, pressure may force open the internal sphincter; this in turn forces open the external sphincter and urination occurs whether it is convenient or not.

After micturition, less than 10ml of urine remains in the bladder and the cycle begins again.

Problems Associated With Micturition

There are several factors which affect the process of micturition. Some of these can be due to physical trauma or disease; others are psychological in nature. Following are a few disorders that affect micturition:

* Detrusor Instability – This is a condition where the detrusor muscle contracts without any apparent reason. This muscle is responsible for contracting the bladder and help with the micturition process. As a result, detrusor instability results in urinary incontinence.
* Urinary Retention – This condition is characterized by the inability to empty the bladder completely. The onset may be gradual or sudden. The causes can range from a blockage in the urethra, nerve problems and weak bladder muscles.
* Spinal Cord Trauma – Injuries to the spinal cord, specifically the tenth thoracic vertebra (T10) can cause the bladder to be overactive or cause urinary incontinence.

3.

The juxtaglomerular apparatus of the kidney serves as an intrarenal baroreceptor that is composed of four basic elements: the terminal portion of the afferent arteriole, the macula densa (a segment of the distal tubule), the extraglomerular mesangial region, and the efferent arteriole at the glomerulus. Because of its location in the nephron, it is highly sensitive to changes in volume as induced by various diuretic classes, and thus it is sensitive to changes in kidney perfusion pressure. The juxtaglomerular apparatus is also known to be adrenergically innervated, and has β-1 adrenoreceptors.

 Sympathetic stimulation or decreases in volume or perfusion pressure can all stimulate the juxtaglomerular apparatus response to release renin into the afferent arteriole. Renin then converts angiotensinogen, which is formed in the liver, to angiotensin I. Angiotensin-converting enzyme converts angiotensin I to angiotensin II, which is a potent vasoconstrictor and stimulates secretion of aldosterone from the adrenal cortex.

4.

The kidneys help in the regulation of the blood pressure by the Renin-Angiotensin aldosterone mechanism. Decrease in blood pressure leads to generation of impulses that travel to the macula densa. The macula densa is a structure found along the renal tubules, it regulates the concentration of sodium chloride. The impulses reaching the macula densa leads to activation of the juxtaglomerular apparatus (a structure found within the renal corpuscles that controls glomerular filtration) and the juxtaglomerular cells produce renin. Renin works on angiotensinogens which are proproteins, it activates them and converts them to angiotensin I. Angiotensin I, to a great extent is inactive so angiotensin converting enzyme (ACE) converts angiotensin I to angiotensin II.

1. Angiotensin II then constricts blood vessels to increase total peripheral resistance and then increase blood pressure.
2. Angiotensin can also act on the adrenal gland to produce aldosterone, which acts on the renal tubules, causing the absorption of electrolytes especially sodium alongside water to increase blood volume, increase venous output, increase cardiac output and finally increase blood pressure.
3. Angiotensin II can stimulate the thirst center, leading to an urge to take water which increases blood volume, increases cardiac output and then increases blood pressure.
4. Finally, angiotensin stimulates the release of antidiuretic hormone/vasopressin from the posterior pituitary gland which acts on collecting ducts to cause the absorption of water, thereby increasing blood volume, increases blood volume, increases venous return, increases cardiac output and finally increases blood pressure.

5.

 The kidney is critically important in calcium homeostasis. Under normal blood calcium concentrations, the parathyroid hormone stimulates production of the biologically-active form of vitamin D within the kidney, which acts also to increase blood concentrations of calcium. The parathyroid hormone then maximizes tubular reabsorption of calcium within the kidney. Almost all of the calcium that enters glomerular filtrate is reabsorbed from the tubular system back into blood, which preserves blood calcium levels. This activity results in minimal losses of calcium in urine. If tubular reabsorption of calcium decreases, calcium is lost by excretion into urine.

 It is critical to maintain blood calcium concentrations within a tight normal range. Deviations above or below the normal range frequently lead to serious diseases:

* Hypocalcemia refers to low blood calcium concentration. Clinical signs of this disorder reflect increased neuromuscular excitability and include muscle spasms, tetany and cardiac dysfunction.
* Hypercalcemia indicates a concentration of blood calcium higher than normal. The normal concentration of calcium and phosphate in blood and extracellular fluid is near the saturation point; elevations can lead to diffuse precipitation of calcium phosphate in tissues, leading to widespread organ dysfunction and damage.