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DEPT: MEDICINE & SURGERY

ASSIGNMENT

1. Discuss the role of the kidney in glucose homeostasis.
2. Discuss the process of micturition.
3. Explain the juxtaglomerular apparatus.
4. Discuss the role of the kidney in regulation of blood pressure.
5. Discuss the role of the kidney in calcium homeostasis.

NUMBER 1

THE ROLE OF THE KIDNEY IN GLUCOSE HOMEOSTASIS

The kidney aids glucose homeostasis by significantly utilizing glucose (primarily in the renal medulla). Apart drom this, the kidney has other ways of regulating glucose, which include;

- Gluconeogenesis; the kidney and the liver are the only organs in the body which have glucose-6-phosphatase enzyme which is involved in gluconeogenesis. Gluconeogenesis is the synthesis of glucose from other non-carbohydrate sources such as lactate and glycerol.
- Removal of water and solutes from plasma and returning of desirably substances into blood.

The kidney reabsorbs glucose which is filtered from plasma. The total glomerular filtration rate GFR in a healthy human is about 180L/day, and the average plasma glucose level is 90mg/dL, hence, in a day the total filtered glucose is 102g. But with the aid of sodium glucose transporters (SGLT 1 and 2) is reabsorbed from the renal tubule lumen. Even in chronic hyperglycemia the kidney reabsorbs glucose.

NUMBER 2

MICTURITION

This is the process by which urine is expelled by the urinary bladder.

This involves two main steps:

Resting/filling phase; First, the bladder fills progressively until the tension in its walls rises above a threshold level

The voiding phase; this is the second step, which is a nervous reflex called the micturition reflex that empties the bladder or,if this fails, at least causes a conscious desire to urinate.

Micturition is regulated by the nervous system and the muscles of the bladder and urethra.

FILLING PHASE

During the filling phase, the bladder accumulates increasing volumes of urine while the pressure inside the bladder remains low. The pressure within the bladder must be lower than the urethral pressure during the filling phase. If the bladder pressure is greater than the urethral pressure (resistance), urine will leak out.

The filling of the urinary bladder depends on the intrinsic viscoelastic properties of the bladder and the inhibition of the parasympathetic nerves. Thus, bladder filling primarily is a passive event.

Sympathetic nerves also facilitate urine storage in the following ways:

- Sympathetic nerves inhibit the parasympathetic nerves from triggering bladder contractions.
- Sympathetic nerves directly cause relaxation and expansion of the detrusor muscle.
- Sympathetic nerves close the bladder neck by constricting the internal urethral sphincter. This sympathetic input to the lower urinary tract is constantly active during bladder filling.

As the bladder fills, the pudendal nerve becomes excited. Stimulation of the pudendal nerve results in contraction of the external urethral sphincter. Contraction of the external sphincter, coupled with that of the internal sphincter, maintains

urethral pressure (resistance) higher than normal bladder pressure. This increase in urethral pressure with filling is the continence reflex.

The pressure gradients within the bladder and urethra play an important functional role in normal micturition. As long as the urethral pressure is higher than that of the bladder, the person will remain continent. If the urethral pressure is abnormally low or if the intravesical pressure is abnormally high, urinary incontinence will result.

During some physical activities and with coughing, sneezing, or laughing, the pressure within the abdomen rises sharply. This rise is transmitted to the bladder, and in response the urethra both anatomically and functionally is designed to increase its pressure and maintain continence. When the pressure transmitted to the bladder is greater than that within the urethra, urine will leak out, resulting in stress incontinence.

EMPTYING PHASE

The storage phase of the urinary bladder can be switched to the voiding phase either involuntarily (reflexively) or voluntarily. Involuntary reflex voiding occurs in an infant when the volume of urine exceeds the voiding threshold. When the bladder is filled to capacity, the stretch receptors within the bladder wall signal the sacral cord. The sacral cord, in turn, sends a message back to the bladder to initiate urination.

At this point, the pudendal nerve causes relaxation of the urethral sphincter, which is also accompanied by broader pelvic floor relaxation. The sympathetic nerves send a message to the internal sphincter to relax and open, resulting in a lower urethral resistance. When the urethral sphincters relax and open, the parasympathetic nerves trigger contraction of the detrusor. When the bladder contracts, the pressure generated by the bladder overcomes the urethral pressure, resulting in urinary flow. These coordinated series of events allow unimpeded, automatic release of the stored urine. While conscious control of this reflex develops after infancy, the primitive voiding reflex may reappear with spinal cord injuries.

DELAYING VOIDING OR VOLUNTARY VOIDING

Bladder function is automatic but completely governed by the brain, which makes the final decision on whether or not to void. The normal function of urination

means that an individual has the ability to stop and start urination on command. In addition, the individual has the ability to delay urination until a socially acceptable time and place. The healthy adult is aware of bladder filling and can willfully initiate or delay voiding.

In a healthy adult, the PMC functions as an on-off switch that is signalled by stretch receptors in the bladder wall and is, in turn, modulated by inhibitory and excitatory neurologic influences from the brain. When the bladder is full, the stretch receptors are activated. The individual perceives the activation of the stretch receptors as the bladder being full, which signals a need to void or the sensation of urinary urge.

When an individual cannot find a bathroom nearby, the brain bombards the PMC with a multitude of inhibitory signals, via the periaqueductal gray matter, to prevent detrusor contractions. At the same time, an individual may actively contract the levator muscles to keep the external sphincter closed or initiate distracting techniques to suppress urination.

NUMBER 3

THE JUXTAGLOMERULAR APPARATUS

The juxtaglomerular apparatus is a specialized structure formed by the junction of the distal convoluted tubule and the glomerular afferent arteriole. The juxtaglomerular apparatus is involved in maintaining blood pressure and volume by the production of the hormone rennin.

The juxtaglomerular apparatus is composed of 3 types of cells

1. Juxtaglomerular cells
2. Macula densa cells
3. Extra glomerular mesangial cells

JUXTAGLOMERULAR CELLS

They are specialized smooth muscle cells located mainly in the walls of the afferent arterioles, that deliver blood to the glomerulus. They are referred to as granular cells because of the granules present in their cytoplasm

MACULA DENSA CELLS

The macula densa is an area of closely packed specialized cells lining the wall of the distal tubule, at the point where the thick ascending limb of the loop of henle meets the distal convoluted tubule. The macula densa is the thickening where the distal tubule touches the glomerulus.

EXTRA GLOMERULAR MESANGIAL CELLS

Extra glomerular mesangial cells are a form of smooth muscle cells. They are also known as lacis cells. They are located in the space between the afferent and efferent arterioles, and the glomerular capillaries. They may be involved in autoregulation of blood flow to the kidney.

FUNCTION OF THE JUXTAGLOMERULAR APPARATUS

1. Secretion of hormones.
2. Regulation of glomerular blood flow and glomerular filtration rate.

SECRETION OF HORMONES

The juxtaglomerulus apparatus secretes 2 hormones

1. Renin
2. Prostaglandin

Renin; renin is a peptide hormone secreted by the juxtaglomerular apparatus. It main function is to cause an increase in blood pressure.

The secretion of rennin is stimulated by 3 factors;

- When a fall in arterial blood pressure is detected by pressure sensitive receptors (baroreceptors) in the arterial vessels.
- When a decrease in sodium chloride is detected in the kidney by the macula densa in the juxtaglomerular apparatus.
- When sympathetic nervous system activity is detected through beta1 adrenergic receptors.

Prostaglandin; Prostaglandins secreted from kidney are PGA2 and PGE2. These hormones are secreted by juxtaglomerular cells. Prostaglandins decrease the blood pressure by systemic vasodilatation, diuresis and natriuresis.

REGULATION OF GLOMERULAR BLOOD FLOW AND GLOMERULAR FILTRATION RATE

Glomerular blood flow and glomerular filtration rate are controlled by the Tubuloglomerular feedback mechanism. Macula densa of juxtaglomerular apparatus in the terminal portion of thick ascending limb is sensitive to the sodium chloride in the tubular fluid. When the glomerular filtrate passes through the terminal portion of thick ascending segment, macula densa acts like a sensor. It detects the concentration of sodium chloride in the tubular fluid and accordingly alters the glomerular blood flow and GFR. Macula densa detects the sodium chloride concentration via Na+-K+2Cl– cotransporter (NKCC2).

NUMBER 4

REGULATION OF BLOOD PRESSURE BY THE KIDNEY

The kidney is only involved in long term response to chronic changes in 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.

Kidneys regulate arterial blood pressure by two ways:

1. By regulation of ECF volume

2. The renin-angiotensin mechanism

Regulation of ECF volume: the kidney regulates blood pressure by changing the ECF volume. It does this by altering the amount of sodium and water excreted.

When blood pressure increases the kidney secretes large amounts of water and sodium by means of pressure diuresis and pressure natriuresis. Pressure diuresis is the excretion of large quantities of water in urine in response to an increase in blood pressure. Natriuresis is the excretion of large quantity of sodium in urine in response to an increase in blood pressure.

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 kidney increases reabsorption of water at the renal tubules. This causes an increase in ECF volume, blood volume and cardiac output, hence, blood pressure increases.

The 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 ACE (angiotensin-converting enzyme).

Angiotensin II acts in two 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 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.

ii. ii. Simultaneously, 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, resulting in increased ECF volume and blood volume. It increases the blood pressure to normal level.

NUMBER 5

THE ROLE OF THE KIDNEY IN CALCIUM HOMEOSTASIS

The kidney is an important organ in regulating calcium homeostasis. It does this by activating vitamin D3 (25-hydroxycholecalciferol) via parathyroid hormone to its active form 1,25dihydroxycholecalciferol (calcitriol).

Actions of 1, 25-Dihydroxycholecalciferol

1. It increases the absorption of calcium from the intestine, by increasing the formation of calciumbinding proteins in the intestinal epithelial cells. These proteins act as carrier proteins for facilitated diffusion, by which the calcium ions are transported. The proteins remain in the cells for several weeks after 1,25-dihydroxycholecalciferol has been removed from the body, thus causing a prolonged effect on calcium absorption
2. It increases the synthesis of calcium-induced ATPase in the intestinal epithelium
3. It increases the synthesis of alkaline phophatase in the intestinal epithelium
4. It increases the absorption of phosphate from intestine along with calcium.