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QUESTIONS

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

ANSWERS

1. The plasma glucose concentration is determined by the amount of glucose synthesized, and the one removed from the circulation and metabolized. This concentration must be maintained within a relatively narrow range despite the wide daily fluctuations in glucose ingestion and glucose demands in various tissues. Other substrate such as free fatty acids(FFA), glycerol, lactate and ketone bodies have greater daily fluctuations. This can be explained by the need of the body to protect himself against hyper- and hypoglycemia. Hyperglycemia is associated with both chronic effects (such as nephropathy, retinopathy, neuropathy and premature atherosclerosis) and also acute complications (including diabetic ketoacidosis and hyperosmolar hyperglycemia state that are associated with higher morbidity and mortality).

The regulation of endogenous production of glucose is determined by hormonal and neural factors. In the acute phase, glucoregulatory mechanism involve insulin, glucagon and catecholamine and they can effect changes in plasma glucose levels in a matter of minutes. Insulin is able to suppress glucose release in both the kidney and liver by direct enzyme activation/deactivation and by reducing the availability of gluconeogenesis substrates. Glucagon has no effect on the kidneys, but its stimulates glycogenolysis and gluconeogenesis in the liver. Catecholamine also have multiple acute actions. They can stimulate renal glucose release and glucagon secretion and inhibit insulin secretion.

The kidney is involved in maintaining glucose homeostasis through three different mechanisms: gluconeogenesis; glucose uptake from blood for its own energy requests and reabsorption into the general circulation of glucose from glomerular filtrate in order to preserve energy.

 2. we depend on micturition to eliminate organic waste products, which are produced as a result of cell metabolism in the body. The urinary system also regulates the concentration of sodium, potassium, chloride and other ions in the blood as well as helping to maintain normal blood Ph., blood pressure and blood volume. This article will concentrate on how urine is produced stored in the bladder and excreted from the body, and will summarize some of the problems that may cause urinary incontinence.

Micturition is a simple reflex which is displayed by infants who are not toilet-trained. When the volume of urine in the bladder reaches about 250ml, stretches 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.

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, so micturition is clearly more than just a simple reflex. 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.

 3. the juxtaglomerular apparatus (also known as the juxtaglomerular complex) is a structure in the kidney that regulates the function of each nephron, the function units of the kidney. The juxtaglomerular apparatus is named because it is next to (juxta-) the glomerulus.

STRUCTURE

The juxtaglomerular apparatus is a part of the kidney nephron, next to the glomerulus. It is found between the afferent arteriole and the distant convoluted tubule of the same nephron. This location is critical to its function in regulating renal blood flow and glomerular filtration rate.

FUNCTION

Renin is produced by juxtaglomerular cells. These cells are similar to epithelium and are located in the tunica media of the afferent arterioles as they enter the glomeruli. The juxtaglomerular cells secrete renin in response to:

* Stimulation of the beta-1 adrenergic receptor
* Decrease in renal perfusion pressure (detected directly by the granular cells)
* Decrease in NaCl concentration at the macula densa, often due to decrease in glomerular filtration rate.

Extraglomerular mesangial cells

Extraglomerular mesangial cells are located in the junction between the afferent and efferent arterioles. These cells have a contractile property similar to vascular smooth muscles and thus play a role in “regulating GFR” by altering the vessel diameter. Renin is also found in these cells.

MACULA DENSA

At the point where the afferent arteriole enters the glomerulus and the efferent arteriole leaves it, the tubule of the nephron the arterioles of the glomerulus from which it rose. At this location, in the wall of the distal convoluted tubule, there is a modified region of the tubular epithelium called the macula densa. Cells in the macula densa respond to changes in the sodium chloride levels in the distal tubule of the nephron via the tubuloglomerular feedback (TGF) loop.

The macula densa’s detection of elevated sodium chloride, which leads to an increase in GFR, is based on the concept of purinergic signaling. An increase in the salt concentration causes several cell signals to eventually the cause the adjacent afferent.

CLINICAL SIGNIFICANCE

Excess secretion of renin by the juxtaglomerular cells can lead to excess activity of the renin-angiotensin system, hypertension and an increase in blood volume. This is not responsive to the usual treatment for essential hypertension, namely medications and lifestyle modification.

One cause of this can be increased renin production due to narrowing of the renal artery, or a tumor of juxtaglomerular cells that produces renin. These will lead to secondary hyperaldosteronism, which will cause hypertension, high blood sodium, low blood potassium, and metabolic alkalosis.

**4. The kidneys and their influence on blood pressure**

The kidneys play a central role in the regulation of arterial blood pressure. A large body of experimental and physiological evidence indicates that renal control of extracellular volume and renal perfusion pressure are closely involved in maintaining the arterial circulation and blood pressure. Renal artery perfusion pressure directly regulates sodium excretion; a process known as pressure natriuresis, and influences the activity of various vasoactive systems such as the renin-angiotensin-aldosterone (RAS) system. Along with vessel morphology, blood viscosity is one of the key factors influencing resistance and hence blood pressure. A key modulator of blood viscosity is the renin-angiotensin system (RAS) or the renin-angiotensin-aldosterone system(RAAS), a hormone system that regulates blood pressure and water balance.

The blood pressure in the body depend upon:

* The force by which the heart pumps out blood from ventricles of the heart – and this dependent on how well the heart muscles gets stretched by the inflowing blood into the ventricles.
* The degree to which the arteries and arterioles constrict- increases the resistance to blood flow, thus requiring a higher blood pressure
* The volume of the blood circulating round the body; if the volume is high, the ventricles get more filled, and the heart muscle gets more stretched.

The kidney influences blood pressure by:

* Causing the arteries and veins to constrict
* Increasing the circulating blood volume

**How the kidneys increase circulating blood volume?**

Angiotensin-2 also stimulates the adrenal gland to secrete a hormone called aldosterone. Aldosterone stimulates more Na reabsorption in the distal tubule, and water gets reabsorbed along with the Na. the increased Na and water reabsorption from the distal tubule reduces urine output and increases the circulating blood volume. The increased blood volume helps stretch the heart muscle and causes it to generate more pressure with each beat, thereby increasing the blood pressure. The circulating blood volume is directly proportional to the stretch of the heart muscle.

5. chronic kidney disease causes imbalance in bone metabolism and increases the risk of a type of bone disease called renal osteodystrophy. These imbalances also can cause calcium to deposit in the blood vessel and contribute to heart disease. To determine calcium status, your doctor will measure and evaluate calcium, phosphorus and PTH levels. If calcium levels are low, a calcium supplement may be prescribed. Sometimes, calcium-based phosphorus binders are prescribed to treat both low calcium and high phosphorus levels.

If calcium levels are high then high calcium food, calcium supplements and calcium-based phosphorus binders may be limited or avoided to help control calcium levels.