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Physiology assignment.

1. The role of the kidney in glucose homeostasis.

The kidneys control the glucose level by three main mechanisms; glycogenolysis, gluconeogenesis and glucose reabsorption. Glycogenolysis is the break down of glycogen to glucose -6-phosphate and its subsequent hydrolysis to free glucose. Gluconeogenesis is the formation of glucose-6-phosphate which is eventually hydrolyzed to glucose from amino acid precursors. In relation to its glucose function, the kidney can be seen as *two* separate organs; the renal medulla and the renal cortex. The renal medulla functions in utilizing glucose because it has phosphorylating enzymes which are able to phosphorylate glycogen. However, the cells of the renal medulla lack glucose -6- phosphatase so they aren't able to release free glucose into the system. The renal cortex on the other hand, functions in glucose production because of it's glucose-6-phosphatase activity. It produces free glucose during prolonged periods of fasting through a process called gluconeogenesis. Lastly, glucose reabsorption is the process of absorbing excess glucose again and its done through the glucose transporter proteins that are present in cell membranes within the proximal tubules. If the capacity of these transporters is exceeded, glucose is found in the urine, a condition called glucosuria.

1. The mechanism of micturition

At its most basic level, 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, 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 neurones. Parasympathetic motor neurones 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 neurones supplying the external sphincter via the pudendal nerve are inhibited, allowing the external sphincter to open and urine to flow out, assisted by gravity.

1. Explain the juxtaglomerular apparatus

The juxtaglomerular apparatus (JGA) is a specialized structure situated at the vascular pole of the glomerulus of each nephron which functions mainly in the regulation of blood pressure and filtration rate of the glomerulus. It is formed by three different structures; the Macula Densa, Extraglomerular mesangial cells and juxtaglomerular cells. The Macula Densa is made up of tightly packed cuboidal epithelial cells. It is found at the end portion of the thick ascending tubule just before it opens into the distal convoluted tubule. Between afferent and efferent arteriole but closer to the afferent arteriole. It functions in the tubuloglomerular feedback mechanism and secretes thromboxane A2.

The extraglomerular mesangial cells or lacis cells or a granular cells. Cells having the capability to secrete prostaglandins and cytokines. They are located in the triangular region bounded by the afferent arteriole, afferent arteriole and Macula Densa.

The juxtaglomerular cells or granular cell : they are specialized smooth muscle cells in the wall of the afferent arteriole just before it enters the Bowman’s capsule. They are mainly in the tunica media and tunica adventitia of the afferent arteriole wall.

The juxtaglomerular apparatus has the juxtaglomerular cells which secrete renin, a hormone that couples with angiontensin forming the renin-angiontensin system which is a hormone system playing an important role in blood pressure homeostasis. The extraglomerular mesangial cells secrete prostaglandins. Macula Densa secretes Thromboxane A2 and plays an important role in the tubuloglomerular feedback mechanism which regulates blood flow and glomerular filtration rate.

1. Regulation of Blood Pressure (BP).

The kidneys participate in the long term regulation of arterial pressure in two ways; through the Extracellular fluid volume (ECF) and The Renin-angiotensin system.

Regulation through the Extracellular fluid volume (ECF): an increase in BP will result in the excretion of large quantities of water in urine condition referred to as Pressure Diuresis. It will also result in an excretion of large quantities of sodium in urine, a condition referred to as Pressure Natriuresis. As a result of diuresis and natriuresis, the ECF volume and blood volume (BV)decrease, bringing arterial pressure back to normal.

Conversely, a decrease in BP will cause more reabsorption of water and sodium from the renal tubules increasing ECF volume, blood volume and cardiac output resulting in the restoration of BP.

Regulation through Renin-angiotensin: Renin is secreted by the juxtaglomerular apparatus and it’s secretion is stimulated by four main factors; a fall in arterial BP, a reduction in ECF volume, increased sympathetic activity and decreased load of sodium and chloride in the macula densa. Resulting from these factors, as earlier stated, Renin is produced. It converts angiotensinogen into angiotensin I which is converted to angiotensin II by angiotensin-converting-enzyme (ACE). Angiotensin II acts in several ways to bring the BP back to normal. It causes the constriction of arterioles of the body which increases peripheral resistance, hence, BP rises. It all also causes constriction of the arterioles of the Kidney so that glomerular filtration can decrease, a decrease in glomerular filtration translates to more water retention, raising the ECF volume and by extension, the blood pressure. Simultaneously, it stimulates the release of aldosterone from the adrenaline cortex which will help increase the reabsorption of sodium by the renal tubules, this increases ECF volume which results in the blood pressure to rise.

1. Calcium homeostasis by the kidney: when the level of calcium in the body is high, the Parathyroid hormone (PTH) is stimulated. Increased levels of PTH will stimulate calcium reabsorption in the thick ascending loop of Henle and distal tubules, this decreases the amount of calcium being excreted in the urine. Conversely, reduction of PTH causes calcium excretion by decreasing the reabsorption in the loops of Henle and distal tubules. In the proximal tubule, calcium reabsorption is independent of PTH. An increase in ECF volume will cause an increase in the excretion of sodium and water in the urine in addition to calcium. Conversely, a decrease in blood pressure will lead to the reabsorption of sodium, water and calcium reducing the amount of calcium being excreted.