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**Q1. Discuss the role of kidney in glucose homeostasis?**

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 the filtration rate of the glomerulus. The macula densa is a collection of specialized epithelial cells in the distal convoluted tubule that detect sodium concentration of the fluid in the tubule. In response to elevated sodium, the macula densa cells trigger contraction of the afferent arteriole, reducing flow of blood to the glomerulus and the glomerular filtration rate. The juxtaglomerular cells, derived from smooth muscle cells, of the afferent arteriole secrete renin when blood pressure in the arteriole falls. Renin increases blood pressure via the renin-angiotensin-aldosterone system. Lacis cells, also called extraglomerular mesangial cells, are flat and elongated cells located near the macula densa. Their function remains unclear

Q2. Discuss the process of micturition?

Micturition is a process where urine is expelled from the body. Animals and humans have a specialised system of organs known as the excretory system to eliminate the waste products from the body. In other words, the process of expelling urine from the body is called micturition. It is brought about by reflex contraction of a special muscle called the **detrusor muscle** after voluntary relaxation of the sphincter muscle.

**Micturition Process**

Micturition process consists of two phases:

**Storage phase**

**Voiding phase**

**Storage Phase**

The urinary bladder is a balloon-shaped, hollow, muscular, organ that acts as the storage organ for urine. The urinary bladder in a healthy urinary system can store up to 16 ounces of urine for 2 to 5 hours easily. The circular sphincter muscles prevent leakage of urine. They close tightly around the opening of the bladder into the tube (urethra) that allows the passage of urine outside the body.

**Voiding Phase**

When the bladder is filled with urine, the nerves in it are triggered, which in turn stimulates the need to urinate. The brain signals urinary bladder to contract. The receptors of the urinary bladder send a signal to the central nervous system, in response to which the nervous system sends a signal that incites the contraction of the urinary bladder. Through the urinary opening at the urethra, the urine is eliminated, and the process is called micturition. The neural mechanism involved is called the micturition reflex.

**Q3. 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 the filtration rate of the glomerulus. The macula densa is a collection of specialized epithelial cells in the distal convoluted tubule that detect sodium concentration of the fluid in the tubule. In response to elevated sodium, the macula densa cells trigger contraction of the afferent arteriole, reducing flow of blood to the glomerulus and the glomerular filtration rate. The juxtaglomerular cells, derived from smooth muscle cells, of the afferent arteriole secrete renin when blood pressure in the arteriole falls. Renin increases blood pressure via the renin-angiotensin-aldosterone system. Lacis cells, also called extraglomerular mesangial cells, are flat and elongated cells located near the macula densa. Their function remains unclear

**Q4 Discuss the role of kidney in regulation of blood pressure?**

The kidney plays 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 system. As a result, many researchers argue that identifying any marked rise in blood pressure requires resetting of the relationship between arterial blood pressure and urinary sodium excretion, which can occur by an array of systemic or local mechanisms. Almost all of the monogenic forms of hypertension affect sites in the kidney associated with sodium handling and transport. Experimental models of spontaneous hypertension, such as the Dahl salt-sensitive rat, have been used to study the effects of kidney transplantation on blood pressure. Results from studies of kidney transplantation indicate that pressure sensitivity to sodium intake 'follows' the kidney, meaning that the recipient of a 'salt-resistant kidney' acquires sodium resistance, and that the recipient of a 'salt-sensitive kidney' acquires pressure sensitivity. The examples above and discussed in this Review demonstrate that it should come as no surprise that most disorders that affect the kidney or the renal vasculature commonly lead to secondary forms of hypertension.

**Q5. Discuss the role of Kidney in Calcium homeostasis?**

The role of the kidney in calcium homeostasis has been reshaped from a classic view in which the kidney was regulated by systemic calcitropic hormones such as vitamin D3 or parathyroid hormone to an organ actively taking part in the regulation of calcium handling. With the identification of the intrinsic renal calcium-sensing receptor feedback system, the regulation of paracellular calcium transport involving claudins, and new paracrine regulators such as klotho, the kidney has emerged as a crucial modulator not only of calciuria but also of calcium homeostasis. kidneys are major regulators of calcium homeostasis. This is illustrated by the profound and complex dysregulation of mineral metabolism appearing during chronic kidney disease (CKD) recognized as mineral and bone disorders in chronic kidney disease (MBD-CKD).Fluxes of calcium between the small intestine (the place for calcium absorption), the bone (the main storage place for calcium), and the kidney (the main place of elimination of the absorbed calcium) are highly controlled by numerous transport mechanisms, hormones, and interconnected feedback loops. This is an absolute requirement to prevent unwanted biomineralization in tissues. Indeed, calcium is a highly reactive ion which has high propensity to form microcrystals in fluids and tissues. In mammals, the complex process of biomineralization takes place in a controlled manner in teeth and bones, in which the matrix is calcified with hydroxyapatite, a calcium-phosphate salt. As the calcification process necessitates interactions between matrix proteins and high local calcium and phosphate concentrations at a specific pH, keeping calcium in solution also demands significant effort. With plasma concentrations of ∼2.4 mmol/l for calcium and 1 mmol/l for phosphate, crystallization would spontaneously occur, if inhibitors of calcification, such as magnesium, fetuin A, osteoprotegerin, or matrix gla protein were not present in plasma. Thus the control of calcification in bone on the one hand and the preserved solubility of calcium salts in plasma on the other hand are both dependent on the tight control of plasma levels of calcium and phosphate and on the presence/absence of strong inhibitors.The same reasoning can be applied to urine in which calcium has a concentration of ∼3 mmol/l and phosphate 10 mmol/l. Inhibitors and promoters of urine crystallization and stone formation have been extensively described, and calciuria appears to be a major promoter of crystallization and stone formation. Hypercalciuria, as defined either as excretion rate or as concentration, is contributing to Randall's plaque formation and to kidney stone formation. Thus control of calciuria is instrumental in reducing the risk of intrarenal biomineralization and kidney stone formation. In plasma, only ∼50% of calcium is freely available, the rest being bound to proteins or forming complexed salts. The concentration of free ionized calcium depends on plasma pH and plasma protein content and constitutes the calcium that is sensed and defended by the organism. It can be measured as ionized calcium.In the kidney, the only source of calcium reaching the tubules is ultrafiltrated calcium, consisting of ionized calcium and other calcium-containing salts filtered through the glomerulus. It represents ∼50% of the total plasma calcium, but it is impossible to be precisely measured in a clinical setting. This constitutes a major caveat when it comes to evaluating the fractional excretion of calcium. No secretion or backleak of calcium contributes to the calcium delivered to the tubular system and, consequently, the load of calcium filtered is the unique and major contributor of calcium reaching the proximal tubule (PT). Along the tubular system, complex transepithelial transport mechanisms allow a highly regulated reabsorption of ∼98% of filtrated calcium. In certain circumstances though, the tubular reabsorption system may be overwhelmed by the filtrated load, as seen in primary hyperparathyroidism or in vitamin D intoxication. In these two conditions, the calcium reabsorption machinery of the kidney is maximally stimulated but cannot counterbalance the filtered calcium load, leading to hypercalciuria.