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Q1. Discuss the role of kidney in glucose homeostasis?

The human kidney is involved in the regulation of glucose homeostasis and in abnormalities found in diabetes mellitus via three different mechanisms: (i) release of glucose into the circulation via gluconeogenesis; (ii) uptake of glucose from the circulation to satisfy its energy needs; and (iii) reabsorption into the circulation of glucose from glomerular filtrate to conserve glucose carbon.

Plasma glucose concentrations are determined by the relative rates of glucose entry into, and removal from, the circulation. Normally, despite wide daily fluctuations in the rate of delivery of glucose into the circulation (e.g. meal ingestion) and in the demands of tissues for glucose (e.g. during exercise), plasma levels are maintained within a relatively narrow range throughout the day. Maximal plasma concentrations following meal ingestion are usually < 9.0 mmol/l and minimal concentrations, after moderate fast or exercise, are usually > 3.0 mmol/l . This is in contrast to other substrates such as glycerol, lactate, free fatty acids (FFAs) and ketone bodies, for which daily fluctuation is much greater. Teleologically, this can be explained by the fact that, on the one hand, the body must defend itself from hyperglycaemia, which is associated with both chronic effects (including retinopathy, neuropathy, nephropathy and premature atherosclerosis) and acute effects (including diabetic ketoacidosis and hyperosmolar hyperglycaemic state, which have significant associated morbidity and mortality); on the other hand, the body must also defend itself against hypoglycaemia, which can cause cardiac arrhythmias, neurological dysfunction, coma, seizures and death. Brain function is particularly dependent on having adequate levels of plasma glucose because the brain is unable to either store or produce glucose and alternative sources of energy are either in short supply (e.g. ketone bodies) or are unable to pass the blood–brain barrier (e.g. FFAs).

The precise regulation of plasma glucose concentrations is mainly determined by hormonal and neural factors, which regulate endogenous production of glucose. Acute glucoregulatory mechanisms involve insulin, glucagon and catecholamines, which can effect changes in plasma glucose levels over a matter of minutes. Insulin suppresses glucose release in both the liver and kidney by direct enzyme activation/deactivation, as well as by reducing the availability of gluconeogenic substrates and actions on gluconeogenic activators. Glucagon has no effect on the kidney, but increases both gluconeogenesis and glycogenolysis in the liver. Catecholamines have multiple acute actions, including stimulation of renal glucose release, inhibition of insulin secretion, stimulation of glucagon secretion, and increases in gluconeogenic substrate supply, stimulation of lipolysis and reduced tissue glucose uptake.

Growth hormone, thyroid hormone and cortisol influence glucose levels over a period of hours by altering the sensitivity of the liver, kidney, adipose tissue and muscle to insulin, glucagon and catecholamines, and by altering the activity of key enzymes, which effect glycogen stores and availability of gluconeogenic precursors (lactate, glycogen and amino acids). In the post-absorptive state, glucose uptake by tissues is largely dependent on tissue needs and the mass-action effects of the ambient plasma glucose concentration and, to a lesser extent, on the permissive actions of insulin and counter-regulatory hormones (e.g. thyroid hormones, growth hormone, catecholamines and cortisol). In these circumstances, most uptake of glucose occurs in tissues that do not require insulin (e.g. brain, gastrointestinal tract, renal medulla). However, in the postprandial state, although insulin and other hormones exert greater influence on tissue uptake of glucose, changes in hepatic and renal glucose release into the circulation are still quite important

Q2. Discuss the process of micturition?

**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.

**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.

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 (Martini, 2002).

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 (Martini, 2002). 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.

We can increase the rate of urine flow by contraction of the abdominal muscles and by the performance of Valsalva’s manoeuvre (forced expiration against a closed glottis) (McLaren, 1996). Contraction of the strong pelvic floor muscles can stop urine in mid-flow. The sound of running water also encourages micturition (Silverthorn, 2003) but some people cannot urinate in the presence of others, no matter how great their need.

After micturition, less than 10ml of urine remains in the bladder (Martini, 2002) and the cycle begins again.

**Potential problems associated with micturition**

For normal micturition to occur we need:

- Intact nerve pathways to the urinary tract;

- Normal muscle tone in the detrusors, sphincters and pelvic floor muscles;

- Absence of any obstruction to urine flow in any part of the urinary tract;

- Normal bladder capacity;

- Absence of environmental or psychological factors which may inhibit micturition.

Loss of any of these normal functions may result in incontinence or urgency to micturate.

Neurological disorders may include stroke, Alzheimer’s disease or any condition where nerve pathways to and from the spine and brain are blocked or injured. The neurotransmitter acetylcholine (ACh) is involved in the relaying of nerve signals in micturition. ACh can be blocked with the drug atropine, so the detrusor muscle will not contract and retention of urine will occur.

Stress incontinence can occur at any age. It occurs when abdominal pressure rises, for example when sneezing or coughing. The normally acute angle between the bladder and urethra is lost when abdominal pressure rises slightly, causing pressure in the bladder to rise.

Laxity and weakness of muscles at the bladder neck, around the urethra and in the pelvic floor will mean that incontinence occurs with relatively small pressure changes. Stress incontinence can occur in men following prostatectomy, and in women after childbirth and during the menopause due to decreased oestrogen secretions.

Renal stones, inflammation and an enlarged prostate gland may all obstruct the flow of urine and may result in frequency of micturition and retention of urine. Bladder tumours and pregnancy also reduce normal bladder capacity. Environmental and psychological factors can also affect a patient’s ability to pass urine.

**Conclusion**

Micturition requires the coordinated activity of sympathetic, parasympathetic and somatic nerves. It also requires normal muscle tone and freedom from physical obstruction and psychological inhibition. Control from our higher brain centres allow us to determine the right time and place to allow this important physiological function to occur

Q3. Explain juxtaglomerular apparatus?

**Juxtaglomerular apparatus (JGA)** A region of tissue found in each [nephron](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/nephron#1O6nephron) in the kidney that is important is regulating [blood pressure](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/blood-pressure) and body fluid and electrolytes. It is located where the distal convoluted tubule passes close to the afferent arteriole supplying the Bowman's capsule, near to the glomerulus, and contains two types of specialized cells. Large [smooth muscle](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/smooth-muscle) cells in the wall of the afferent arteriole form the *juxtaglomerular cells*, or granular cells; these contain granules of the proteolytic enzyme [renin](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/renin#1O6renin), which are released when the juxtaglomerular cells detect decreased [blood pressure](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/blood-pressure) in the arteriole. The JGA also includes chemoreceptor cells of the adjacent region of the distal tubule, which form a tightly packed array called the *macula densa*. This detects low concentrations of sodium ions in the filtrate inside the kidney tubule (indicative of reduced plasma sodium levels) and triggers release of renin from the juxtaglomerular cells. Release of renin into the bloodstream leads to increased levels of [angiotensins](https://www.encyclopedia.com/medicine/anatomy-and-physiology/anatomy-and-physiology/angiotensin#1O6angiotensin), which raise blood pressure and also stimulate the secretion of [aldosterone](https://www.encyclopedia.com/science-and-technology/biochemistry/biochemistry/aldosterone#1O6aldosterone) from the adrenal cortex and [antidiuretic hormone](https://www.encyclopedia.com/science-and-technology/biochemistry/biochemistry/antidiuretic-hormone#1O6antidiuretichormone) from the posterior pituitary. Aldosterone promotes reabsorption of sodium ions from the distal tubule, and [antidiuretic hormone](https://www.encyclopedia.com/science-and-technology/biochemistry/biochemistry/antidiuretic-hormone) promotes water reabsorption.

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

Calcium homeostasis is a complex process involving the following 4 key components: serum calcium, serum phosphate, 1,25-dihydroxyvitamin D-3, and parathyroid hormone (PTH). More than 99% of the total body calcium is stored in bone in the form of phosphate and hydroxide salts, predominantly as hydroxyapatite. Normally, a very small portion of this calcium is available for exchange in the serum.