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Course : renal physiology

1] renal handling of glucose and electrolyte ;

The kidney contributes to glucose homeostasis through processes of gluconeogenesis, glucose filtration, glucose reabsorption, and glucose consumption. Each of these processes can be altered in patients with type-2 diabetes , providing potential targets for novel therapies. Recent studies have indicated that the kidney is responsible for up to twenty percent of all glucose production via gluconeogenesis. In patients with, overall glucose production increases by as much as three hundred per cent , with equal contributions from hepatic and renal sources. This increased production contributes not only to increased fasting glucose in T2DM patients but also to raised postprandial glucose because, in contrast to the liver, glucose ingestion increases renal gluconeogenesis. Under normal circumstances, up to 180 g/day of glucose is filtered by the renal glomerulus and virtually all of it is subsequently reabsorbed in the proximal convoluted tubule. This reabsorption is effected by two sodium-dependent glucose cotransporter.

The process of renal glucose reabsorption is mediated by active (sodium-coupled glucose cotransporters) and passive (glucose transporters) transporters. In hyperglycemia, the kidneys may play an exacerbating role by reabsorbing excess

glucose, ultimately contributing to chronic hyperglycemia, which in turn contributes to chronic glycemic burden and the risk of microvascular consequences. This article provides an extensive review of the kidneys' role in normal human physiology, the mechanisms by which they contribute to glucose regulation, and the potential impact of glucose imbalance on the kidneys.

Renal release of glucose into the circulation is the result of glycogenolysis and gluconeogenesis. Glycogenolysis involves the breakdown of glycogen to glucose-6-phosphate from precursors. Conversely, gluconeogenesis involves formation of glucose-6-phosphate from those same precursors and subsequent conversion to free glucose. Interestingly, the liver and skeletal muscles contain most of the body's glycogen stores, but only the liver contains glucose-6-phosphatase. The basic urine-forming unit of the kidney is the nephron, which serves to filter water and small solutes from plasma and reabsorb electrolytes, amino acids, glucose, and protein.

The nephron, of which there are approximately 1 million in each kidney, consists of a filtering apparatus (the glomerulus) that is connected to a long tubular portion that reabsorbs and conditions the glomerular ultrafiltrate. Fluid filtered from the glomerular capillaries flows into the tubular portion, which is made up of a proximal tubule, the Loop of Henle, and the distal tubule, all of which assist in reabsorbing essential substances and converting filtered fluid into urine.

2] physiology of micturition :

Micturition or urination is the process of expelling urine from the bladder. This act is also known as voiding of the bladder. The excretory system in humans includes a

pair of kidneys, two ureters, a urinary bladder and a urethra. The kidneys filter the urine and it is transported to the urinary bladder via the ureters where it is stored till its expulsion. The process of micturition is regulated by the nervous system and the muscles of the bladder and urethra.

Stages of Micturition

The urinary bladder has two stages;

1. Resting or filling stage
2. Voiding stage

Resting or Filling Stage

It is in this phase of the bladder that the urine is transported from the kidneys via the ureters into the bladder. The ureters are thin muscular tubes that arise from each of the kidneys and extend downwards where they enter the bladder obliquely. The oblique placement of the ureters in the bladder wall serves a very important function. The opening of the ureter into the urinary bladder is not guarded by any sphincter or muscle. Therefore, this oblique nature of opening prevents the urine from re-entering the ureters. At the same time, the main muscle of the urinary bladder, the detrusor muscle, is relaxing allowing the bladder to distend and accommodate more urine. Action potentials carried by sensory neurons from stretch receptors in the urinary bladder wall travel to the sacral segments of the spinal cord through the pelvic nerves. Since bladder wall stretch is low during the storage phase, these afferent neurons fire at low frequencies. Low-frequency afferent signals cause relaxation of the bladder by inhibiting sacral parasympathetic preganglionic neurons and exciting lumbar sympathetic

preganglionic neurons. Conversely, afferent input causes contraction of the sphincter through excitation of Onuf's nucleus, and contraction of the bladder neck and urethra through excitation of the sympathetic preganglionic neurons.

Voiding Stage

During this stage, both the urinary bladder and the urethra come into play together. The detrusor muscle of the urinary bladder which was relaxing so far starts to contract once the bladder's storage capacity is reached. The urethra is controlled by two sets of muscles: The internal and external urethral sphincters. The internal sphincter is a smooth muscle whereas the external one is skeletal. Both these sphincters are in a contracted state during the filling stage.

Within the nervous system, the process is governed by the autonomous nervous system and the somatic system. Once the urinary bladder reaches its maximum capacity, the stretch receptors in the walls of the bladder send an impulse via the pelvic nerve to the brain via the spinal cord. The micturition reflex is ultimately generated from the level of the spinal cord after it receives reflexes from the pontine region in the brain. Once the bladder and the urethra receive the signals to empty the bladder, the two sphincters relax and the detrusor muscle causes the contractions of the bladder.

Along with these muscles, the muscles of the abdomen also play a role by putting pressure on the bladder wall. This leads to complete emptying of the bladder. The bladder has a dual role: to store urine for much of the time without excess leakage; to void the bladder content completely and rapidly at a time that is under the control of the individual. An inability to achieve these objectives will result in

incontinence, ineffective voiding or retention of urine, and is a cause of a considerable reduction in the quality of life, and significant medical problems