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1. 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 (T2DM), providing potential targets for novel therapies. Recent studies have indicated that the kidney is responsible for up to 20% of all glucose production via gluconeogenesis. In patients with T2DM, overall glucose production increases by as much as 300%, 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 (SGLT) proteins. SGLT2, situated in the S1 segment, is a low-affinity high-capacity transporter reabsorbing up to 90% of filtered glucose. SGLT1, situated in the S3 segment, is a high-affinity low-capacity transporter reabsorbing the remaining 10%. In patients with T2DM, renal reabsorptive capacity maladaptively increases from a normal level of 19.5 to 23.3 mmol/l/min. Once glucose has been reabsorbed into the tubular epithelial cells, it diffuses into the interstitium across specific facilitative glucose transporters (GLUTs). GLUT1 and GLUT2 are associated with SGLT1 and SGLT2, respectively.

The effect of long-term ethanol feeding on the activity of (Na + K)-ATPase in cortex and outer medulla and fractional excretion of electrolytes in remnant kidney of adult rats after unilateral nephrectomy were studied. Wistar adult rats were fed 20% (v/v) aqueous ethanol solution as sole drinking fluid for 8-10 weeks. Right kidney was removed under ether anaesthesia. The animals were subjected to an acute NaCl loading by means of a continuous infusion given 2, 7 and 14 days after nephrectomy. Renal handling of electrolytes was estimated from fractional excretion of sodium and potassium. After the infusion the animals were killed and (Na + K)-ATPase and Mg2+-ATPase activities were measured in the cortex and outer medulla of the remnant kidney. Two days after nephrectomy both groups showed a gradual increase of renal (Na + K)-ATPase activity reaching 60 percent at day 14. Mg2+-ATPase activity did not change with respect to basal values. Compared to basal values the fractional excretion of sodium after nephrectomy, dropped in both groups but more significantly in the ethanol-fed rats than in the control group. Fractional excretion of potassium did not change in the control group after nephrectomy while the ethanol-fed group displayed a significative decrease at days 7 and 14. According to our results the rise in renal (Na + K)-ATPase activity is consistent with the renal sodium retention found in ethanol-fed rats.

1. 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. The urinary bladder can store around 350-400ml of urine before it expels it out.

The urinary bladder has two distinct stages or phases:

Resting or filling stage

* 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.

 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.

As mentioned earlier, the process of micturition is governed by both the nervous and muscular systems. 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.