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Pharmacology

PHS 212

Renal glucose reabsorption is the part of kidney (renal) physiology that deals with the retrieval of filtered glucose, preventing it from disappearing from the body through the urine.

If glucose is not reabsorbed by the kidney, it appears in the urine, in a condition known as glycosuria. This is associated with diabetes mellitus.

Firstly, the glucose in the proximal tubule is co-transported with sodium ions into the proximal convoluted tubule walls via the SGLT2 cotransporter. Some (typically smaller) amino acids are also transported in this way. Once in the tubule wall, the glucose and amino acids diffuse directly into the blood capillaries along a concentration gradient. This blood is flowing, so the gradient is maintained. Lastly, sodium/potassium ion active transport pumps remove sodium from the tubule wall and the sodium is put back into the blood. This maintains a sodium concentration gradient in the proximal tubule lining, so the first step continues to happen.

Gliflozins such as canagliflozin inhibit renal glucose reabsorption,and are used in diabetes mellitus to lower blood glucose.

Glucose handling by the kidney.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.

Electrolytes are particles that carry an electric charge when they are dissolved in blood. The kidneys help to maintain electrolyte concentrations by regulating its concentrations in the body. Any disturbance in this process often leads to an electrolyte imbalance.

The different electrolytes are:

\* Sodium

\* Potassium

\* Phosphorus

\* Calcium

\* Magnesium

Renal Failure is often complicated by elevated potassium, phosphate and magnesium and decreased sodium and calcium.

Low potassium (hypokalemia) may not cause symptoms, but it may affect how your body stores glucogen (your muscles' source of energy) or cause abnormal heart rhythms. A level under three can cause muscle weakness, spasms, cramps, paralysis and respiratory problems. If it continues, kidney problems may occur.

References

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2.) Urination is the release of urine from the urinary bladder through the urethra to the outside of the body. It is the urinary system's form of excretion. It is also known medically as micturition, voiding, uresis, or, rarely, emiction, and known colloquially by various names including peeing, weeing, and pissing.

In healthy humans (and many other animals) the process of urination is under voluntary control. In infants, some elderly individuals, and those with neurological injury, urination may occur as a reflex. It is normal for adult humans to urinate up to seven times during the day.

Physiology of micturition

The physiology of micturition and the physiologic basis of its disorders are subjects about which there is much confusion, especially at the supraspinal level. Micturition is fundamentally a spinobulbospinal reflex facilitated and inhibited by higher brain centers such as the pontine micturition center and, like defecation, subject to voluntary facilitation and inhibition.

In healthy individuals, the lower urinary tract has two discrete phases of activity: the storage (or guarding) phase, when urine is stored in the bladder; and the voiding phase, when urine is released through the urethra. The state of the reflex system is dependent on both a conscious signal from the brain and the firing rate of sensory fibers from the bladder and urethra.At low bladder volumes, afferent firing is low, resulting in excitation of the outlet (the sphincter and urethra), and relaxation of the bladder.At high bladder volumes, afferent firing increases, causing a conscious sensation of urinary urge. When the individual is ready to urinate, he or she consciously initiates voiding, causing the bladder to contract and the outlet to relax. Voiding continues until the bladder empties completely, at which point the bladder relaxes and the outlet contracts to re-initiate storage.

In infants, voiding occurs involuntarily (as a reflex). The ability to voluntarily inhibit micturition develops by the age of 2–3 years, as control at higher levels of the central nervous system develops. In the adult, the volume of urine in the bladder that normally initiates a reflex contraction is about 300–400 millilitres (11–14 imp fl oz; 10–14 US fl oz).

Storage phase

During storage, bladder pressure stays low, because of the bladder's highly compliant nature. A plot of bladder (intravesical) pressure against the depressant of fluid in the bladder (called a cystometrogram), will show a very slight rise as the bladder is filled. This phenomenon is a manifestation of the law of Laplace, which states that the pressure in a spherical viscus is equal to twice the wall tension divided by the radius. In the case of the bladder, the tension increases as the organ fills, but so does the radius. Therefore, the pressure increase is slight until the organ is relatively full. The bladder's smooth muscle has some inherent contractile activity; however, when its nerve supply is intact, stretch receptors in the bladder wall initiate a reflex contraction that has a lower threshold than the inherent contractile response of the muscle.

Diuresis (production of urine by the kidney) occurs constantly, and as the bladder becomes full, afferent firing increases, yet the micturition reflex can be voluntarily inhibited until it is appropriate to begin voiding.

Voiding begins when a voluntary signal is sent from the brain to begin urination, and continues until the bladder is empty.

Bladder afferent signals ascend the spinal cord to the periaqueductal gray, where they project both to the pontine micturition center and to the cerebrum.At a certain level of afferent activity, the conscious urge to void becomes difficult to ignore. Once the voluntary signal to begin voiding has been issued, neurons in pontine micturition center fire maximally, causing excitation of sacral preganglionic neurons. The firing of these neurons causes the wall of the bladder to contract; as a result, a sudden, sharp rise in intravesical pressure occurs. The pontine micturition center also causes inhibition of Onuf's nucleus, resulting in relaxation of the external urinary sphincter.When the external urinary sphincter is relaxed urine is released from the urinary bladder when the pressure there is great enough to force urine to flow out of the urethra. The micturition reflex normally produces a series of contractions of the urinary bladder.

After urination, the female urethra empties partially by gravity, with assistance from muscles.[clarification needed] Urine remaining in the male urethra is expelled by several contractions of the bulbospongiosus muscle, and, by some men, manual squeezing along the length of the penis to expel the rest of the urine.

For land mammals over 1 kilogram, the duration of urination does not vary with body mass, being dispersed around an average of 21 seconds (standard deviation 13 seconds), despite a 4 order of magnitude (1000×) difference in bladder volume.This is due to increased urethra length of large animals, which amplifies gravitational force (hence flow rate), and increased urethra width, which increases flow rate. For smaller mammals a different phenomenon occurs, where urine is discharged as droplets, and urination in smaller mammals, such as mice and rats, can occur in less than a second.

Voluntary control:

The mechanism by which voluntary urination is initiated remains unsettled.One possibility is that the voluntary relaxation of the muscles of the pelvic floor causes a sufficient downward tug on the detrusor muscle to initiate its contraction.Another possibility is the excitation or disinhibition of neurons in the pontine micturition center, which causes concurrent contraction of the bladder and relaxation of the sphincter.

There is an inhibitory area for micturition in the midbrain. After transection of the brain stem just above the pons, the threshold is lowered and less bladder filling is required to trigger it, whereas after transection at the top of the midbrain, the threshold for the reflex is essentially normal. There is another facilitatory area in the posterior hypothalamus. In humans with lesions in the superior frontal gyrus, the desire to urinate is reduced and there is also difficulty in stopping micturition once it has commenced. However, stimulation experiments in animals indicate that other cortical areas also affect the process.

Experience of urination

The need to urinate is experienced as an uncomfortable, full feeling. It is highly correlated with the fullness of the bladder.In many males the feeling of the need to urinate can be sensed at the base of the penis as well as the bladder, even though the neural activity associated with a full bladder comes from the bladder itself, and can be felt there as well. In females the need to urinate is felt in the lower abdomen region when the bladder is full. When the bladder becomes too full, the sphincter muscles will involuntarily relax, allowing urine to pass from the bladder. Release of urine is experienced as a lessening of the discomfort.

Disorders

Clinical conditions

Many clinical conditions can cause disturbances to normal urination, including:

A.)Urinary incontinence, the inability to hold urine

I.)Stress incontinence, incontinehnce as a result of external mechanical disturbances

ii.)Urge incontinence, incontinence that occurs as a result of the uncontrollable urge to urinate

iii.)Mixed incontinence, a combination of the two types of incontinence

B.)Urinary retention, the inability to initiate urination

C.)Overactive bladder, a strong urge to urinate, usually accompanied by detrusor overactivity

D.)Interstitial cystitis, a condition characterized by urinary frequency, urgency, and pain

E.)Prostatitis, an inflammation of the prostate gland that can cause urinary frequency, urgency, and pain

F.)Benign prostatic hyperplasia, an enlargement of the prostate that can cause urinary frequency, urgency, retention, and the dribbling of urine

G.)Urinary tract infection, which can cause urinary frequency and dysuria

Deafferentation

When the sacral dorsal roots are cut in experimental animals or interrupted by diseases of the dorsal roots such as tabes dorsalis in humans, all reflex contractions of the bladder are abolished. The bladder becomes distended, thin-walled, and hypotonic, but there are some contractions because of the intrinsic response of the smooth muscle to stretch.

Denervation

When the afferent and efferent nerves are both destroyed, as they may be by tumors of the cauda equina or filum terminale, the bladder is flaccid and distended for a while. Gradually, however, the muscle of the "decentralized bladder" becomes active, with many contraction waves that expel dribbles of urine out of the urethra.

Spinal cord injury

During spinal shock, the bladder is flaccid and unresponsive. It becomes overfilled, and urine dribbles through the sphincters (overflow incontinence). After spinal shock has passed, a spinally mediated voiding reflex ensues, although there is no voluntary control and no inhibition or facilitation from higher centers. Some paraplegic patients train themselves to initiate voiding by pinching or stroking their thighs, provoking a mild mass reflex. In some instances, the voiding reflex becomes hyperactive. Bladder capacity is reduced and the wall becomes hypertrophied. This type of bladder is sometimes called the spastic neurogenic bladder. The reflex hyperactivity is made worse, and may be caused, by infection in the bladder wall.

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