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Course Assignment Title: Renal physiology for pharmacology students

**Question**

Q1. Discuss renal handling of glucose and electrolytes?

Q2. Discuss the physiology of micturition?

Answer

Q1) Renal handling of glucose and electrolytes

The effects of hyperglycemia and hyperinsulinemia on renal handling of sodium, calcium, and phosphate were studied in dogs employing the recollection micropuncture technique. Subthreshold sustained hyperglycemia resulted in an isonatric inhibition of proximal tubular sodium, fluid, calcium, and phosphate reabsorption by 8-14%. Fractional excretion of sodium and phosphate, however, fell (P is less than 0.01) indicating that the increased delivery of these ions was reabsorbed in portions of the nephron distal to the site of puncture and in addition net sodium and phosphate transport was enhanced resulting in a significant antinatriuresis and antiphosphaturia. The creation of a steady state plateau of hyperinsulinemia while maintaining the blood glucose concentration of euglycemic levels mimicked the effects of hyperglycemia on proximal tubular transport and fractional excretion of sodium and calcium. Tubular fluid to plasma insulin ratio fell, similar to the hyperglycemic studies. These results suggest that the effects of hyperglycemia on renal handling of sodium and calcium may be mediated through changes in plasma insulin concentration. In contrast to hyperglycemia, however, hyperinsulinemia cuased a significant fall in tubular fluid to plasma phosphate ratio with enhanced proximal tubular phosphate reabsorption (P is less than 0.02). This occurred concomitantly with a significant inhibition of proximal tubular sodium transport. These data indicate that insulin has a direct effect on proximal tubular phosphate reabsorption, and this effect of insulin is masked by the presence of increased amounts of unreabsorbed glucose in the tubule that ensues when hyperinsulinemia occurs secondary to hyperglycemia. Fractional excretion of phosphate fell significantly during insulin infusion but unlike the hyperglycemic studies, the fall in phosphate excretion could be entirely accounted for by enhanced proximal reabsorption.

Q2) Physiology of 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.

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. 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 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 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.