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Filtration, Reabsorption, Secretion: The Three Steps of Urine Formation

Cross section of the glomerulus, a structure of the nephron

The kidneys filter unwanted substances from the blood and produce urine to excrete them. There are three main steps of urine formation: glomerular filtration, reabsorption, and secretion. These processes ensure that only waste and excess water are removed from the body.

1. The Glomerulus Filters Water and Other Substances from the Bloodstream

Blood flow through the glomerulus as part of filtration

Each kidney contains over 1 million tiny structures called nephrons. Each nephron has a glomerulus, the site of blood filtration. The glomerulus is a network of capillaries surrounded by a cuplike structure, the glomerular capsule (or Bowman’s capsule). As blood flows through the glomerulus, blood pressure pushes water and solutes from the capillaries into the capsule through a filtration membrane. This glomerular filtration begins the urine formation process.

2. The Filtration Membrane Keeps Blood Cells and Large Proteins in the Bloodstream

Click to play an animation of the filtration membrane filtering water and small solutes, while keeping blood cells and large proteins in the bloodstream

Inside the glomerulus, blood pressure pushes fluid from capillaries into the glomerular capsule through a specialized layer of cells. This layer, the filtration membrane, allows water and small solutes to pass but blocks blood cells and large proteins. Those components remain in the bloodstream. The filtrate (the fluid that has passed through the membrane) flows from the glomerular capsule further into the nephron.

3. Reabsorption Moves Nutrients and Water Back into the Bloodstream

Click to play an animation of the reabsorption of nutrients in the glomerulus

The glomerulus filters water and small solutes out of the bloodstream. The resulting filtrate contains waste, but also other substances the body needs: essential ions, glucose, amino acids, and smaller proteins. When the filtrate exits the glomerulus, it flows into a duct in the nephron called the renal tubule. As it moves, the needed substances and some water are reabsorbed through the tube wall into adjacent capillaries. This reabsorption of vital nutrients from the filtrate is the second step in urine creation.

4. Waste Ions and Hydrogen Ions Secreted from the Blood Complete the Formation of Urine

Click to play an animation of waste ions and hydrogen ions passing from the capillaries into the renal tubule.

The filtrate absorbed in the glomerulus flows through the renal tubule, where nutrients and water are reabsorbed into capillaries. At the same time, waste ions and hydrogen ions pass from the capillaries into the renal tubule. This process is called secretion. The secreted ions combine with the remaining filtrate and become urine. The urine flows out of the nephron tubule into a collecting duct. It passes out of the kidney through the renal pelvis, into the ureter, and down to the bladder.

5. Urine Is 95% Water

The percentage composition of salts, ammonia, urea, water and other components of urine.

The nephrons of the kidneys process blood and create urine through a process of filtration, reabsorption, and secretion. Urine is about 95% water and 5% waste products. Nitrogenous wastes excreted in urine include urea, creatinine, ammonia, and uric acid. Ions such as sodium, potassium, hydrogen, and calcium are also excreted.

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“What’s in Your Pee?” from Popular Science.

A cadaver image of the renal artery and kidney from Stony Brook University School of Medicine. The Body Online

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The concentration of urine

As already indicated, the loop of Henle is critical to the ability of the kidney to concentrate urine. The high concentration of salt in the medullary fluid is believed to be achieved in the loop by a process known as countercurrent exchange multiplication. The principle of this process is analogous to the physical principle applied in the conduction of hot exhaust gases past cold incoming gas so as to warm it and conserve heat. That exchange is a passive one, but in the kidney the countercurrent multiplier system uses energy to “pump” sodium and chloride out of the ascending limb of the loop into the medullary fluid. From there it enters (by diffusion) the filtrate (isotonic with plasma) that is entering the descending limb from the proximal tubule, thus raising its concentration a little above that of plasma. As this luminal fluid in turn reaches the ascending limb, and subsequently the distal tubule, it in turn provides more sodium to be pumped out into the surrounding fluid or blood, if necessary, and transported (by diffusion) back into the descending limb; this concentrating process continues until the osmotic pressure of the fluid is sufficient to balance the resorptive power of the collecting ducts in the medulla, through which all of the final urine must pass. This resorptive capacity in the ducts is regulated by antidiuretic hormone (ADH), which is secreted by the hypothalamus and stored in the posterior pituitary gland at the base of the brain. In the presence of ADH, the medullary collecting ducts become freely permeable to solute and water. As a consequence, the fluid entering the ducts (en route to the renal pelvis and subsequent elimination) acquires the concentration of the interstitial fluid of the medulla; i.e., the urine becomes concentrated. On the other hand, in the absence of ADH, the collecting ducts are impermeable to solute and water, and, thus, the fluid in the lumen, from which some solute has been removed, remains less concentrated than plasma; i.e., the urine is dilute.

The secretion of ADH by the hypothalamus and its release from the posterior pituitary is part of a feedback mechanism responsive to the tonicity of plasma. This interrelation between plasma osmotic pressure and ADH output is mediated by specific and sensitive receptors at the base of the brain. These receptors are particularly sensitive to sodium and chloride ions. At normal blood tonicity there is a steady receptor discharge and a steady secretion of ADH. If the plasma becomes hypertonic (i.e., has a greater osmotic pressure than normal), either from the ingestion of crystalloids such as common salt, or from shortage of water, receptor discharge increases, triggering increased ADH output, and more water leaves the collecting ducts to be absorbed into the blood. If the osmotic pressure of plasma becomes low, the reverse is the case. Thus water ingestion dilutes body fluids and reduces or stops ADH secretion; the urine becomes hypotonic, and the extra water is excreted in the urine.

The situation is complex because there are also receptors sensitive to changes in blood volume that reflexively inhibit ADH output if there is any tendency to excessive blood volume. Exercise increases ADH output and reduces urinary flow. The same result may follow emotional disturbance, fainting, pain, and injury, or the use of certain drugs such as morphine or nicotine. Diuresis is an increased flow of urine produced as the result of increased fluid intake, absence of hormonal activity, or the taking of certain drugs that reduce sodium and water reabsorption from the tubules. If ADH secretion is inhibited by the drinking of excess water, or by disease or the presence of a tumour affecting the base of the brain, water diuresis results; and the rate of urine formation will approach the rate of 16 millilitres per minute filtered at the glomeruli. In certain disorders of the pituitary in which ADH secretion is diminished or absent—e.g., diabetes insipidus—there may be a fixed and irreversible output of a large quantity of dilute urine.

Tubular secretion

The only difference between secretory and reabsorptive tubular mechanisms lies in the direction of transport; secretory mechanisms involve the addition of substances to the filtrate from the plasma in the peritubular capillaries. The small amount of secretion that does occur, except for the secretion of potassium and uric acid, takes place in the proximal tubule. Hydrogen ions are also secreted and ammonia is generated, but they are special cases and are discussed below under Regulation of acid-base balance. As in the case of reabsorption, secretion occurs both passively and actively against an electrochemical gradient.

Several drugs are actively secreted, and some of these appear to share a common pathway so that they may compete with each other for a limited amount of energy. This may be turned to therapeutic advantage in the case of penicillin, which is eliminated partly by tubular secretion. The drug probenecid, which can be given simultaneously, competes with penicillin at its secretory site and thus helps to raise the level of penicillin in the blood in the treatment of certain infections. Endogenous (originating within the body) compounds that are secreted also include prostaglandins, bile salts, and hippurate. Uric acid derived from nucleoproteins freely passes the glomerular barrier and is normally largely reabsorbed in the proximal tubule. In some circumstances, however, it is also secreted by other parts of the same convoluted tubule.

The secretion of potassium by the distal tubule is one of the most important events in the kidney as its control is fundamental to the maintenance of overall potassium balance. More than 75 percent of the filtered potassium is reabsorbed in the proximal tubule and in the ascending limb of the loop of Henle, and this percentage remains virtually constant, irrespective of how much is filtered. The amount eliminated in the urine, which is ultimately determined by the dietary intake, is controlled by the distal convoluted tubule. In persons consuming a normal diet, probably about 50 percent of the urinary potassium is secreted into the urine by the distal tubules; this amount can be adjusted according to body need. One of the several factors that influence potassium secretion is a hormone secreted by the cortex of the adrenal gland, aldosterone. In the absence of aldosterone and other mineralocorticoids (adrenocortical steroids affecting electrolyte and fluid balance), potassium secretion is impaired, and potentially dangerous amounts can accumulate in the blood. Excess aldosterone promotes potassium excretion.

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Regulation of acid-base balance

The cells of the body derive energy from oxidative processes that produce acidic waste products. Acids are substances that ionize to yield free protons, or hydrogen ions. Those hydrogen ions that derive from nonvolatile acids—such as lactic, pyruvic, sulfuric, and phosphoric acids—are eliminated in the urine. The kidney contains transport mechanisms that are capable of raising the concentration of hydrogen ions in the urine to 2,500 times that in the plasma or, when appropriate, lowering it to one-quarter that of the plasma.

Theoretically, acidification of urine could be brought about either by the secretion of hydrogen ions into the tubular fluid or by the selective absorption of a buffer base (a substance capable of accepting hydrogen ions; e.g., filtered bicarbonate). Current evidence indicates that both filtration and secretion are essential to hydrogen ion excretion and that both proximal and distal convoluted tubules are involved.

The bulk of the bicarbonate filtered at the glomerulus is reabsorbed in the proximal tubule, from which it passes back into the peritubular capillaries. This mechanism is designed to keep the normal plasma bicarbonate concentration constant at about 25 millimoles per litre. When the plasma concentration falls below this level, no bicarbonate is excreted and all filtered bicarbonate is reabsorbed into the blood. This level is often referred to as the bicarbonate threshold. When the plasma bicarbonate rises above 27 millimoles per litre, bicarbonate appears in the urine in increasing amounts.

The brush borders of the cells of the proximal tubules are rich in the enzyme carbonic anhydrase. This enzyme facilitates the formation of carbonic acid (H2CO3) from CO2 and H2O, which then ionizes to hydrogen ions (H+) and bicarbonate ions (HCO3-). The starting point for bicarbonate reabsorption is probably the active secretion of hydrogen ions into the tubular fluid. These ions may be formed under the influence of carbonic anhydrase from CO2 liberated from oxidation of cell nutrients and H2O already in the cells. The filtered base, bicarbonate, accepts the hydrogen ions to form carbonic acid, which is unstable and dissociates to form CO2 and H2O. The partial pressure of CO2 in the filtrate rises, and, as CO2 is highly diffusible, it passes readily from the tubular fluid into the tubular cells and the blood, and the water is either dealt with in the same way or is excreted. In the meantime the proximal tubular cells are actively reabsorbing filtered sodium, which is balanced by the HCO3- formed within the cells from the CO2 generated by the hydrogen ions in the luminal fluid. Thus the bicarbonate actually reabsorbed is not that which was originally the filtrate, but the net effect is the same as if this were the case.

Other bases besides HCO3- may buffer the hydrogen ions secreted into the distal tubules; in addition, the ions may combine with ammonia also secreted by the tubules. The most important non-bicarbonate base present in the filtrate is dibasic phosphate (Na2HPO4), which accepts hydrogen ions to form monobasic phosphate (NaH2PO4). A measure of the amount of hydrogen ion in the urine that is buffered by bases such as bicarbonate and phosphate is made by the titration of urine with strong base until the pH of the plasma from which the filtrate is derived (7.4) is achieved. This is called the titratable acidity of urine and usually amounts to between 20 and 40 millimoles of H+ per day.

In normal circumstances about two-thirds of the hydrogen ions to be secreted in the urine is in the form of ammonium salts (e.g., ammonium chloride). Ammonia (NH3) is not present in plasma or filtrate but is generated in the distal tubular cells and passes into the lumen probably by passive diffusion down a concentration gradient. In the lumen the NH3 combines with hydrogen ions secreted into the tubule to form ammonium ions (NH4+), which are then trapped in the lumen because the lipid walls of the tubular cells are much less permeable to the charged than to the uncharged molecules.

It is now known that ammonia is formed from the hydrolysis of glutamine (an amino acid) to form glutamic acid and ammonia by the enzyme glutaminase. A further molecule of ammonia is obtained by the deamination of glutamic acid to form glutaric acid, which is then metabolized. The more acidic the urine is, the greater is its content of ammonium ions; the introduction of hydrogen ions (e.g., from the diet) stimulates production of ammonium by the tubular cells. The ammonium is excreted in the urine as ammonium salts of surplus anions (negative ions) such as chloride, sulfate, and phosphate, thus sparing for retention other cations (positive ions) such as sodium or potassium.

In summary, hydrogen ion secretion can be considered in three phases. The first occurs in the proximal tubule, where the net result is tubular reabsorption of filtered bicarbonate. The second and third phases take place in the distal tubule, where monobasic phosphate and ammonium salts are formed. The total tubular cell secretion of hydrogen ion is therefore the sum of titratable acidity, the amount of ammonium ion excreted, and the amount of bicarbonate ion reabsorbed. The last may be assessed by calculating the amount of bicarbonate filtered (i.e., plasma concentration of bicarbonate × glomerular filtration rate and subtracting any bicarbonate excreted in the urine). Total hydrogen ion secretion normally amounts to 50–100 millimoles per day but may rise considerably above this in disorders associated with excess acid production, such as diabetes.

Volume and composition

The volume and composition of normal urine vary widely from day to day, even in healthy individuals, as a result of food and fluid intake and of fluid loss through other channels as affected by environmental conditions and exercise. The daily volume averages 1.5 litres (about 1.6 quarts) with a range of 1–2.5 litres, but after copious sweating it may fall as low as 500 millilitres, and after excess fluid intake it may reach three litres or more. There is also variation within a 24-hour period. Excretion is reduced in the early hours, maximal during the first few hours after rising, with peaks after meals and during the early stages of exertion. The urine produced between morning and evening is two to four times the night volume. The excessive secretion of urine (polyuria) of chronic renal disease is typically nocturnal.

The volume of urine is regulated to keep plasma osmotic concentration constant, to control the total water content of the tissues, and to provide a vehicle for the daily excretion to the exterior of some 50 grams of solids, mostly urea and sodium chloride. In a man who ingests 100 grams of protein and 10 grams of salt daily, the urine will contain 30 grams of urea and 10 grams of salt; there are many other possible constituents, but they amount to less than 10 grams overall.

Some urinary constituents (Table 3)—the products of metabolism of nitrogenous substances obtained from food—vary widely in relation to the composition of the diet; thus the excretion of urea and sulfate is dependent on the diet-protein content. A high-protein diet may yield a 24-hour output of 17 grams of nitrogen, a low-protein diet of the same calorific value only three to four grams.

Some urine constituents

(g/24 hours)

urea 25–30

uric acid 0.6–0.7

creatinine 1.0–1.2

hippuric acid 0.7

ammonia 0.7

amino acids 3

sodium 1–5 (NaCl 15.0)

potassium 2–4

calcium 0.2–0.3

magnesium 0.1

chloride 7

phosphate 1.7–2.5

sulfate 1.8–2.5

The urine is normally clear. It may be turbid from calcium phosphate, which clears if acetic acid is added. Microscopic deposits include occasional casts, vaguely resembling in form the renal tubules from whose lining they have been shed. An ammoniacal smell is the result of decomposition of urea to ammonia by bacteria and is commonly present on babies’ diapers. Certain foods and drugs may cause distinctive odours. The colour of urine depends on its concentration but is normally a bright clear yellow from the pigment urochrome, an end product of protein metabolism. There are also traces of other pigments: urobilin and uroerythrin. The colour may be influenced as well by vitamins, food dyes, beetroot, and certain drugs.

The specific gravity of urine may vary between 1.001 and 1.04 but is usually 1.01–1.025. Such variation is normal, and a fixed low specific gravity is an indication of chronic renal disease. If fluid intake is stopped for 24 hours, a normal kidney will secrete urine with a specific gravity of at least 1.025. There is a limit to the concentrating powers of the kidney, so that the urine is rarely more than four times as concentrated as plasma. In order to excrete their normal solute load, the kidneys need a minimum water output of 850 millilitres as a vehicle; this volume is often called the minimum obligatory volume of urine. If this is not available from intake it has to be withdrawn from the tissues, causing dehydration; but the usual intake is well above the minimum and the urine is rarely at its maximum possible concentration. The reaction of the urine is usually acid, with an overall range of pH 4 to 8 (lemon pie has a pH of 2.3; the value 8 is slightly alkaline, about equal to the pH of a 1 percent solution of sodium bicarbonate).

Foreign proteins of molecular weight less than 68,000 are excreted in the urine, while those of the plasma are retained in the body. If, however, the kidneys are damaged by disease or toxins, the glomeruli will transmit some of the normal serum albumin and globulin and the urine will coagulate on warming. Normally, the urine contains only very small amounts of protein (less than 50 milligrams per 24 hours); however, protein content in the urine is increased after exercise, in pregnancy, and in some persons when standing (orthostatic albuminuria). The protein loss may be greatly increased in certain chronic renal diseases; in the nephrotic syndrome it may even reach 50 grams in a 24-hour period. Certain specific and easily identifiable proteins appear in the urine in diseases associated with the overgrowth of cells that make immunoglobulins.

Glucose is found in the urine in diabetes mellitus. In some healthy persons, however, there may also be an abnormal amount of glucose in the urine because of a low threshold for tubular reabsorption, without any disturbance of glucose metabolism. Lactosuria (abnormal amount of lactose in the urine) may occur in nursing mothers. Ketone bodies (acetone, acetoacetic acid) are present in traces in normal urine but in quantity in severe untreated diabetes and in relative or actual carbohydrate starvation—e.g., in a person on a high-fat diet.

The urine may contain hemoglobin or its derivatives after hemolysis (liberation of hemoglobin from red blood cells), after incompatible blood transfusion, and in malignant malaria (blackwater fever). Fresh blood may derive from bleeding in the urinary tract. Bile salts and pigments are increased in jaundice, particularly the obstructive variety; urobilin is greatly increased in certain diseases such as cirrhosis of the liver.

Porphyrins are normally present only in minute amounts but may be increased in congenital porphyria, a disease characterized by sensitivity to sunlight or by insanity. The presence of porphyrins also may increase after ingestion of sulfonamides and some other drugs.

The normally small quantities of amino acids in the urine may be much increased in advanced liver disease, in failure of tubular reabsorption, and in certain diseases due to inborn errors of protein metabolism. Phenylketonuria, a disease identified by the presence of phenylpyruvic acid in the urine, is due to lack of the enzyme phenylalanine hydroxylase, so that phenylalanine is converted not to tyrosine but to phenylpyruvic acid. The presence of this acid in blood and tissues causes mental retardation; it may be readily detected if the urine of every newborn infant is tested. Restriction of phenylalanine in the diet in such cases may be beneficial. Alkaptonuria, a disease identified by the presence of homogentisic acid in the urine, is due to lack of the enzyme that catalyzes the oxidation of homogentisic acid; deposits of the acid in the tissues may cause chronic arthritis or spinal disease. Other such disorders are cystinuria, the presence of the amino acid cystine in the urine, when the bladder may contain cystine stones; and maple syrup disease, another disorder involving abnormal levels of amino acid in the urine and blood plasma.

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Urine collection and emission

From the nephrons the urine enters the final 15 or 20 collecting tubules that open on to each papilla of the renal medulla, projecting into a minor calyx. These open into two or three major calyxes, and these in turn open into the renal pelvis, which connects with the upper expanded portion of the ureter.

Urine is passed down the channel of the renal pelvis and ureter by a succession of peristaltic waves of contraction that begin in the muscle fibres of the minor calyxes, travel out to the major calyxes and then along the ureter every 10–15 seconds. Each wave sends urine through the ureteric orifice into the bladder in discontinuous spurts; these can be seen through a cystoscope if a dye is injected into the bloodstream. Gravity aids this downward flow, which is faster when one is standing erect. Though the overall picture suggests that there is a pacemaker (a set of specialized cells capable of rhythmic contractions) near the pelviureteric junction, this has never been satisfactorily demonstrated in the tissue. The pressure in the renal pelvis is normally low, but the smooth muscle coat of the ureter is a powerful one and the pressure above an obstructed ureter may rise as high as 50 millimetres of mercury. The ureters are doubly innervated from the splanchnic nerves above and the hypogastric network below.

The bladder

The bladder is a hollow organ of variable capacity, with a powerful intermediate muscle coat that empties the organ when it contracts, and two muscular sphincters that keep the exit closed at all other times. This smooth muscle coat constitutes the powerful detrusor muscle. At the base of the bladder the region of the bladder neck, or trigone, is demarcated by the two ureteric orifices and the internal opening of the urethra. Muscle fibres loop around the urethral opening to form the internal sphincter, which is under involuntary control. The external sphincter consists of two layers of striated muscles under voluntary control.

The mucous membrane lining the bladder is distensible; it is ridged in the empty organ and smoothed out in distension. In micturition the longitudinal muscle of the bladder shortens to widen the bladder neck and allow urine to enter the urethra. The urethra normally contains no urine except during the act of micturition, its walls remaining apposed by muscle tone. The external sphincter can maintain continence even if the internal sphincter is not functioning.

The innervation of the bladder and urethra is complex and important. Essentially, there are three groups of nerves: (1) The parasympathetic nerves constitute the main motor supply to the detrusor; they make it contract, raise pressure within the bladder, relax the internal sphincter, and cause emptying. Afferent parasympathetic channels convey impulses from stretch receptors in the bladder wall to higher centres, permitting cognizance of the state of distension of the organ and stimulating the desire to micturate. (2) The sympathetic nerves stimulate closure of the ureteric and internal urethral orifices and contraction of the internal sphincter, and their action on the detrusor is inhibitory; i.e., the effect is to prevent bladder outflow. Thus the sympathetic nerves act to control the situation in the distending bladder up to the point when evacuation can be deferred no longer. Afferent paths in the sympathetic system convey sensations of pain, overdistension, and temperature from the mucosa of the bladder and the urethra. (3) The somatic nerves cause contraction of the external sphincter; their sensory fibres relay information as to the state of distension of the posterior urethra.

Both the parasympathetic nerves and the somatic nerves (pudendal nerve) to the external sphincter relay impulses to the second through fourth sacral segments of the spinal cord, which constitute a reflex centre for the control of bladder function. This centre connects with higher centres in the brain by ascending and descending fibres in the spinal cord.

Bladder function in micturition

Certain reflexes combine to ensure both maintenance of a steady holding state for urine and normal progressive micturition with complete emptying. When the internal pressure of the bladder rises, it contracts; and it also contracts when urine enters the urethra.

Both bladder sphincters are normally closed. As the organ fills with urine, the contractile response of the muscle wall causes a rise in internal pressure. Relaxation then occurs as an active process of adjustment so that the organ may hold its contents at a lower pressure. As urine continues to enter the bladder, this rise and fall of pressure continues in steplike fashion, with the final pressure always gradually rising.

The repeated transient contraction waves at first are small and are not consciously felt; later, stimuli reach the brain and cause pain and a sharp rise of pressure. These later major contractions can be inhibited voluntarily. The desire to micturate begins at around a content of 400 millilitres, but it can be voluntarily overridden until the content reaches 600–800 millilitres, with a resulting pressure within the bladder of up to 100 millimetres of water. Until this point the sphincters remain contracted to keep the urethral exit closed, but eventually the desire to micturate becomes urgent and irrepressible. Until that time, voluntary inhibition of the detrusor and contraction of the perineal muscles have kept the internal pressure as low as possible and have prevented urine leakage. The threshold is dependent to some extent on the rate of filling and is higher when filling is slow; and training affects the amount the bladder can retain. In young children the situation is less controllable, and even small amounts of urine may excite reflex evacuation. Emotional influences are important. Anxiety inhibits the capacity of the bladder to relax on filling, so that under conditions of stress there may be some involuntary passage of small quantities of urine.

Micturition

Micturition is a complex activity, partly reflex and unconscious and mediated by the lower spinal cord centres, and partly under conscious control by the higher centres of the brain. Voluntary micturition begins with willed messages from the brain that reach the bladder via the motor fibres of the pelvic nerves to stimulate the detrusor, at the same time actively relaxing both urethral sphincters. But the reflexes already mentioned ensure that, once the process has begun and urine has entered the urethra, the contraction of the detrusor will continue and the sphincters will remain relaxed until evacuation is complete and the bladder empty. Evacuation is aided by voluntary contraction of a wide range of accessory muscles. The muscles of the abdominal wall contract to increase pressure on the bladder from without; the diaphragm descends and the breath is held; at the same time there is relaxation of the muscles of the perineal floor. Thus voluntary initiation and control of micturition is effected partly by an active process of stimulating parasympathetic sacral nerve outflow, partly by removing the normal inhibition exerted by the higher centres on the reflex centres in the spinal cord. Once begun, micturition is carried through to completion by lower and higher centres acting in concert; sensory messages from the urine-distended urethra also play a part. It follows that even if a bladder is not particularly distended and if reflex emptying is not urgent, the bladder can nevertheless be evacuated by voluntary contraction of the abdominal wall, so initiating the reflex process that, once begun, takes over.