CHAPTER 13

The urinary system

ANIMATIONS

The urinary system is the main excretory system and consists of the following structures:

- 2 *kidneys*, which secrete urine
- 2 *ureters* that convey the urine from the kidneys to the urinary bladder
- the *urinary bladder*, which collects and stores urine
- the *urethra* through which urine leaves the body. 13.1

Figure 13.1 shows an overview of the urinary system. The urinary system plays a vital part in maintaining homeostasis of water and electrolytes within the body. The kidneys produce urine that contains metabolic waste products, including the nitrogenous compounds urea and uric acid, excess ions and some drugs. The main functions of the kidneys are:

- formation of urine, maintaining water, electrolyte and acid–base balance
- excretion of waste products
- production and secretion of *erythropoietin*, the hormone that stimulates formation of red blood cells (p. 66)
- production and secretion of *renin*, an important enzyme in the control of blood pressure (p. 225).

Urine is stored in the bladder and excreted by the process of *micturition*.

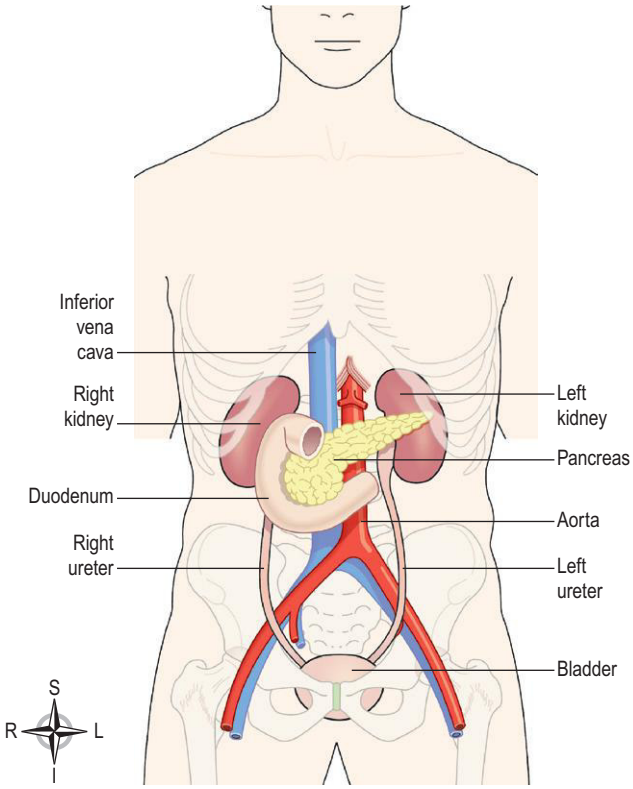


Figure 13.1 The parts of the urinary system (excluding the urethra) and some associated structures.

The first sections of this chapter explore the structures and functions of the organs of the urinary system and the impact of ageing on kidney function. In the final section the consequences of abnormal functioning of the various parts of the urinary system on body function are considered.

Kidneys

Learning outcomes

After studying this section, you should be able to:

- identify the organs associated with the kidneys
- outline the gross structure of the kidneys
- describe the structure of a nephron
- explain the processes involved in the formation of urine
- explain how body water and electrolyte balance is maintained.

The kidneys (Fig. 13.2) lie on the posterior abdominal wall, one on each side of the vertebral column, behind the peritoneum and below the diaphragm. They extend from the level of the 12th thoracic vertebra to the 3rd lumbar vertebra, receiving some protection from the lower rib cage. The right kidney is usually slightly lower than the left, probably because of the considerable space occupied by the liver.

Kidneys are bean-shaped organs, about 11 cm long, 6 cm wide, 3 cm thick and weigh 150 g. They are embedded in, and held in position by, a mass of fat. A sheath of fibrous connective tissue, the *renal fascia*, encloses the kidney and the renal fat.

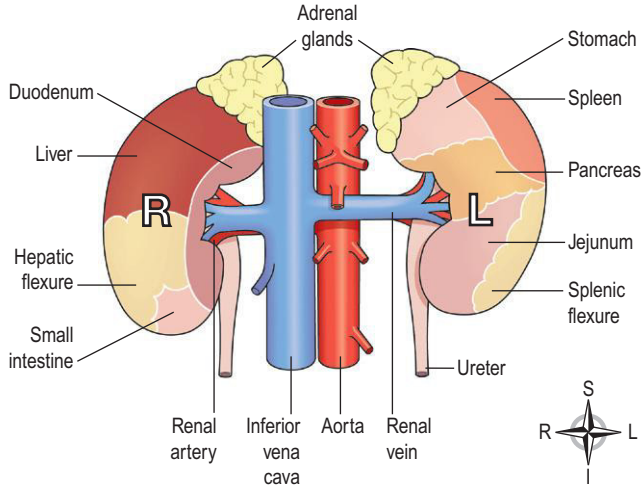


Figure 13.2 Anterior view of the kidneys showing the areas of contact with associated structures.

Organs associated with the kidneys

(Figs 13.1 and 13.2)

As the kidneys lie on either side of the vertebral column, each is associated with different structures.

Right kidney

Superiorly – the right adrenal gland
Anteriorly – the right lobe of the liver, the duodenum and the hepatic flexure of the colon
Posteriorly – the diaphragm, and muscles of the posterior abdominal wall.

Left kidney

Superiorly – the left adrenal gland
Anteriorly – the spleen, stomach, pancreas, jejunum and splenic flexure of the colon
Posteriorly – the diaphragm and muscles of the posterior abdominal wall.

Gross structure of the kidney 13.2

There are three areas of tissue that can be distinguished when a longitudinal section of the kidney is viewed with the naked eye (Fig. 13.3):

- an outer fibrous *capsule*, surrounding the kidney
- the *cortex*, a reddish-brown layer of tissue immediately below the capsule and outside the *renal pyramids*
- the *medulla*, the innermost layer, consisting of pale conical-shaped striations, the renal pyramids.

The *hilum* is the concave medial border of the kidney where the renal blood and lymph vessels, the ureter and nerves enter.

Urine formed within the kidney passes through a *renal papilla* at the apex of a pyramid into a *minor calyx* (Fig. 13.3). Several minor calyces merge into a *major calyx* and two or three major calyces combine forming the *renal pelvis*, a funnel shaped structure that narrows when it leaves the kidney as the ureter. The walls of the calyces and renal pelvis are lined with transitional epithelium and contain smooth muscle. Peristalsis, intrinsic contraction of smooth muscle, propels urine through the calyces, renal pelvis and ureters to the bladder.

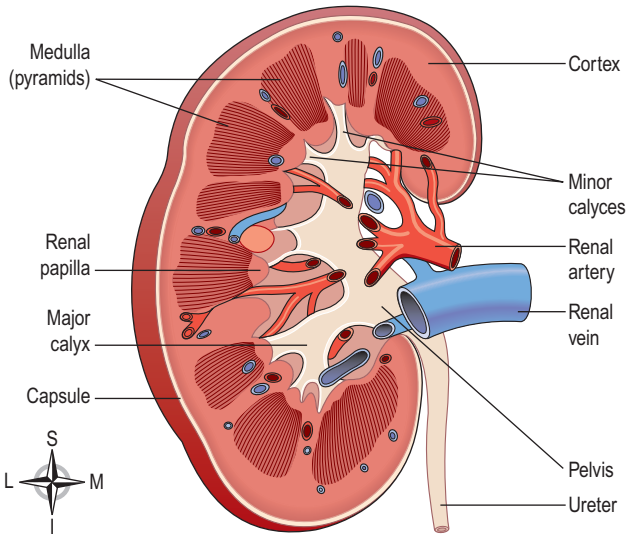


Figure 13.3 A longitudinal section of the right kidney.

Microscopic structure of the kidney

13.3

The kidney contains about 1–2 million functional units, the *nephrons*, and a much smaller number of *collecting ducts*. The collecting ducts transport urine through the pyramids to the calyces, giving the pyramids their striped appearance (Fig. 13.3). The collecting ducts are supported by connective tissue, containing blood vessels, nerves and lymph vessels.

The nephron (Fig. 13.4)

The nephron is essentially a tubule closed at one end that joins a collecting duct at the other end. The closed or blind end is indented to form the cup-shaped *glomerular capsule* (Bowman's capsule), which almost completely encloses a network of tiny arterial capillaries, the *glomerulus*. These resemble a coiled tuft and are shown in Figure 13.5. Continuing from the glomerular capsule, the remainder of the nephron is about 3 cm long and described in three parts:

- the proximal convoluted tubule
- the medullary loop (loop of Henle)
- the distal convoluted tubule, leading into a collecting duct.

The collecting ducts unite, forming larger ducts that empty into the minor calyces.

The kidneys receive about 20% of the cardiac output. After entering the kidney at the hilum, the renal artery divides into smaller arteries and arterioles. In the cortex an arteriole, the *afferent arteriole*, enters each glomerular capsule and then subdivides into a cluster of tiny arterial capillaries, forming the glomerulus. Between these capillary loops are connective tissue phagocytic *mesangial cells*, which are part of the monocyte–macrophage defence system (p. 70). The blood vessel leading away from the glomerulus is the *efferent arteriole*. The afferent arteriole has a larger diameter than the efferent arteriole, which increases pressure inside the glomerulus and drives filtration across the glomerular capillary walls (Fig. 13.6). The efferent arteriole divides into a second peritubular (meaning 'around tubules') capillary network, which wraps around the remainder of the tubule, allowing exchange between the fluid in the tubule and the bloodstream (Figs 13.4 and 13.7). This maintains the local supply of oxygen and nutrients and removes waste products. Venous blood drained from this capillary bed

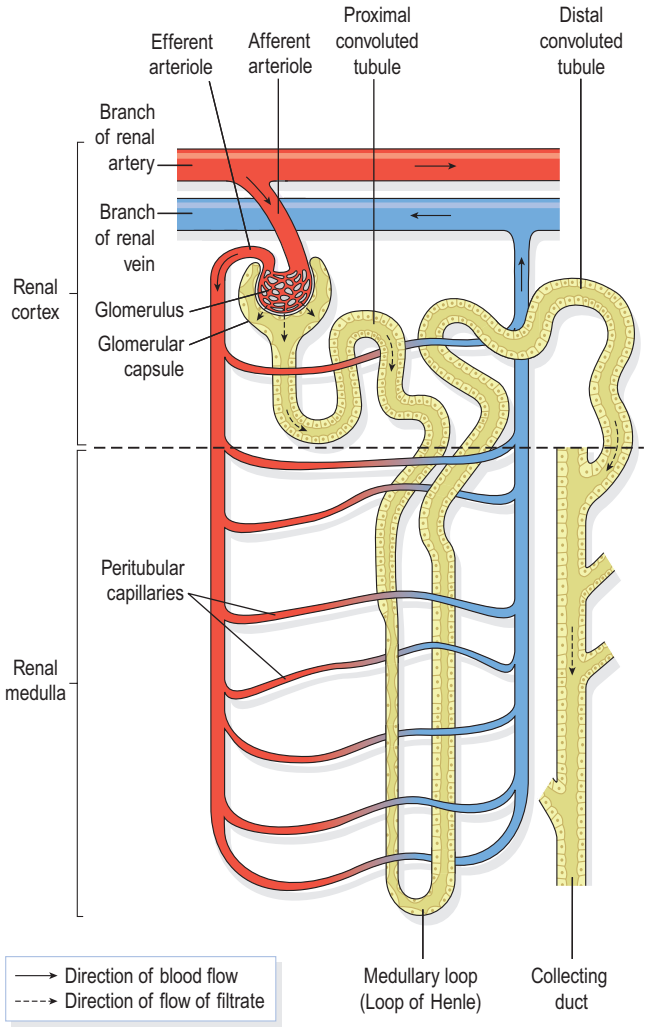


Figure 13.4 A nephron and associated blood vessels.

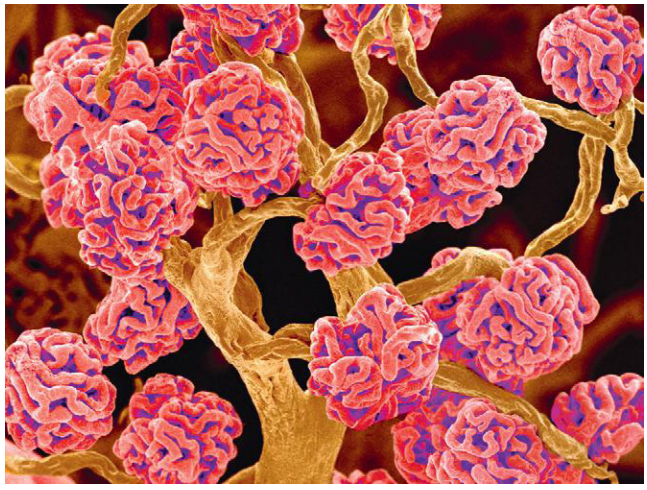


Figure 13.5 Coloured scanning electron micrograph of glomerular capillary tufts.

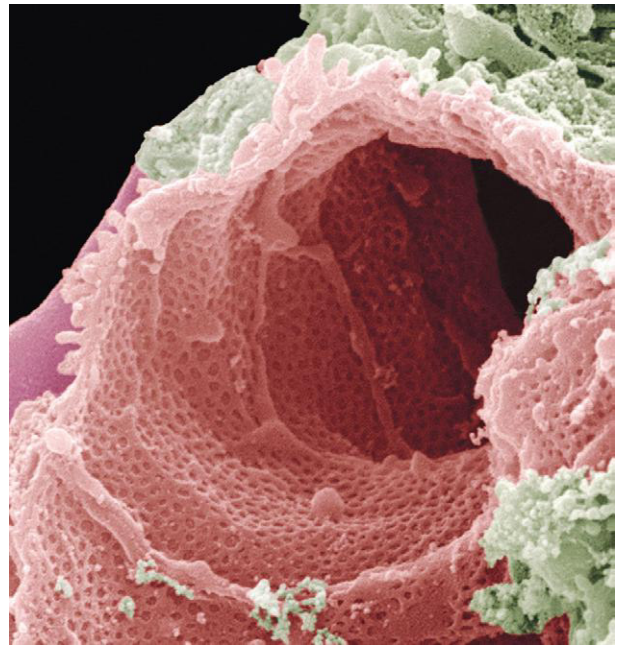


Figure 13.6 Coloured scanning electron micrograph of glomerular capillary.

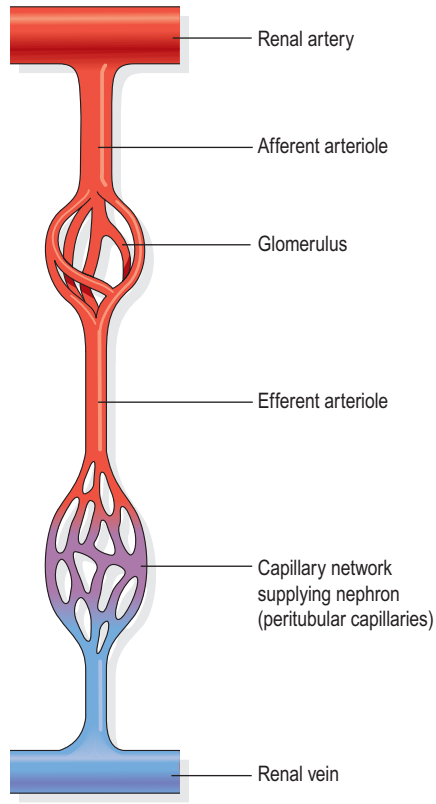


Figure 13.7 The series of blood vessels in the kidney.

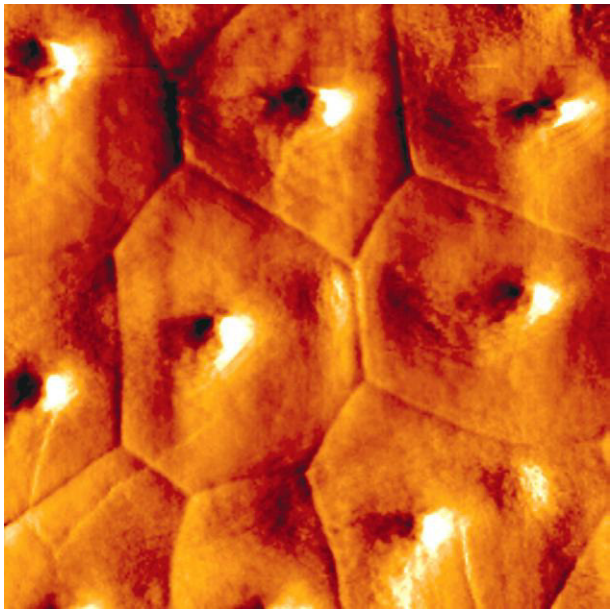


Figure 13.8 Simple squamous epithelium of the collecting ducts. Coloured atomic force micrograph.

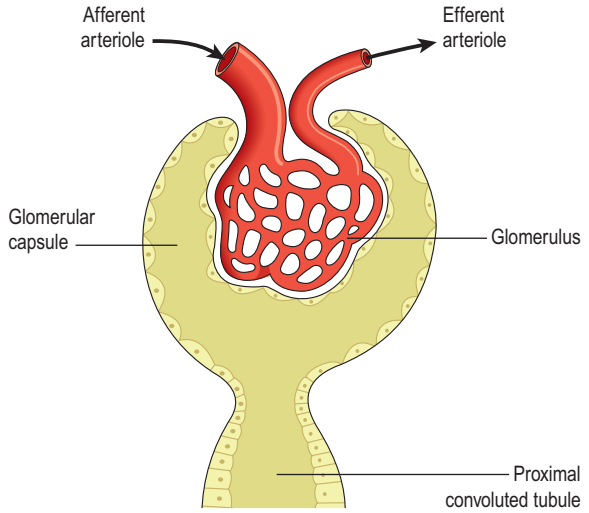


Figure 13.9 The glomerulus and glomerular capsule.

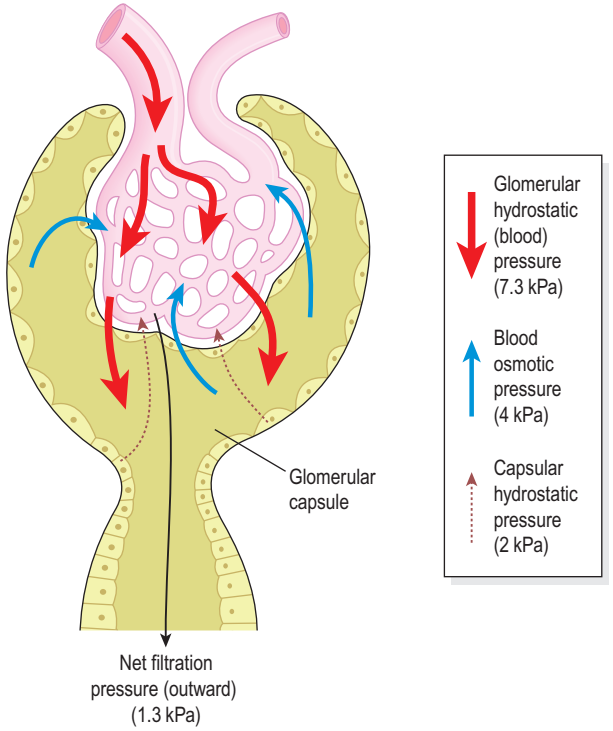


Figure 13.10 Filtration in the glomerulus.

eventually leaves the kidney in the renal vein, which empties into the inferior vena cava.

The walls of the glomerulus and the glomerular capsule consist of a single layer of flattened epithelial cells. The glomerular walls are more permeable than those of other capillaries. The remainder of the nephron and the collecting duct are formed by a single layer of simple squamous epithelium (Fig. 13.8).

Renal blood vessels are supplied by both sympathetic and parasympathetic nerves. The presence of both divisions of the autonomic nervous system controls renal blood vessel diameter and renal blood flow independently of autoregulation (p. 342).

Functions of the kidney

Formation of urine

The kidneys form urine, which passes to the bladder for storage prior to excretion. The composition of urine reflects exchange of substances between the nephron and the blood in the renal capillaries. Waste products of protein metabolism are excreted, water and electrolyte levels are controlled and pH (acid–base balance) is maintained by excretion of hydrogen ions. There are three processes involved in the formation of urine:

- filtration
- selective reabsorption
- secretion.

Filtration (Fig. 13.10) **13.4, 13.5**

This takes place through the semipermeable walls of the glomerulus (Fig. 13.9) and glomerular capsule. Water and other small molecules readily pass through, although some are reabsorbed later. Blood cells, plasma proteins and other large molecules are too large to filter through and therefore remain in the capillaries (Box 13.1). The filtrate in the glomerulus is very similar in composition to plasma with the important exceptions of plasma proteins and blood cells.

Filtration takes place because there is a difference between the blood pressure in the glomerulus and the pressure of the filtrate in the glomerular capsule. Because

Box 13.1 Constituents of glomerular filtrate and glomerular capillaries

Blood constituents in glomerular filtrate	Blood constituents remaining in glomerular capillaries
Water	Leukocytes
Mineral salts	Erythrocytes
Amino acids	Platelets
Ketoacids	Plasma proteins
Glucose	Some drugs (large molecules)
Some hormones	
Creatinine	
Urea	
Uric acid	
Some drugs (small molecules)	

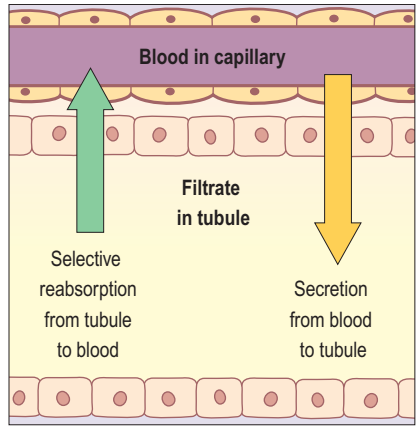


Figure 13.11 Directions of selective reabsorption and secretion in the nephron.

the efferent arteriole is narrower than the afferent arteriole, a *capillary hydrostatic pressure* of about 7.3 kPa (55 mmHg) builds up in the glomerulus. This pressure is opposed by the *osmotic pressure* of the blood, provided mainly by plasma proteins, about 4 kPa (30 mmHg), and by *filtrate hydrostatic pressure* of about 2 kPa (15 mmHg) in the glomerular capsule. The net *filtration pressure* is, therefore:

$$7.3 - (4 + 2) = 1.3 \text{ kPa, or}$$
$$55 - (30 + 15) = 10$$

The volume of filtrate formed by both kidneys each minute is called the *glomerular filtration rate* (GFR). In a healthy adult the GFR is about 125 mL/min, i.e. 180 litres of filtrate are formed each day by the two kidneys. Nearly all of the filtrate is later reabsorbed from the kidney tubules with less than 1%, i.e. 1–1.5 litres, excreted as urine. The differences in volume and concentration are due to selective reabsorption of some filtrate constituents and tubular secretion of others (see below).

Autoregulation. Renal blood flow, and therefore glomerular filtration, is protected by a mechanism called *autoregulation*, whereby renal blood flow is maintained at a constant pressure across a wide range of systolic blood pressures (from around 80–200 mmHg). Autoregulation operates independently of nervous control, i.e. if the nerve supply to the renal blood vessels is interrupted, autoregulation continues to operate. It is therefore a property inherent in renal blood vessels; it may be stimulated by changes in blood pressure in the renal arteries or by fluctuating levels of certain metabolites, e.g. prostaglandins.

In severe shock, when the systolic blood pressure falls below 80 mmHg, autoregulation fails and renal blood flow and the hydrostatic pressure decrease, impairing filtration within the glomeruli.

Selective reabsorption (Fig. 13.11) 13.6

Most reabsorption from the filtrate back into the blood takes place in the proximal convoluted tubule, whose walls are lined with microvilli to increase surface area for absorption. Many substances are reabsorbed here, including some water, electrolytes and organic nutrients such as glucose. Some reabsorption is passive, but some substances, e.g. glucose, are actively transported. Only 60–70% of filtrate reaches the medullary loop. Much of this, especially water, sodium and chloride, is reabsorbed in the loop, so that only 15–20% of the original filtrate reaches the distal convoluted tubule, and the composition of the filtrate is now very different. More electrolytes are reabsorbed here, especially sodium, so the filtrate entering the collecting ducts is actually quite dilute. The main function of the collecting ducts is to reabsorb as much water as the body needs.

Active transport takes place at carrier sites in the epithelial membrane, using chemical energy to transport substances against their concentration gradients (p. 37).

Some ions, e.g. sodium and chloride, can be absorbed by both active and passive mechanisms depending on the site in the nephron.

Some constituents of glomerular filtrate (e.g. glucose, amino acids) do not normally appear in urine because they are completely reabsorbed unless blood levels are excessive.

Reabsorption of nitrogenous waste products, such as urea, uric acid and creatinine is very limited.

The kidneys' maximum capacity for reabsorption of a substance is the *transport maximum*, or renal threshold. For example, the normal blood glucose level is 3.5–8 mmol/L (63 to 144 mg/100 mL) and if this rises above the transport maximum of about 9 mmol/L (160 mg/100 mL), glucose appears in the urine. This occurs because all the carrier sites are occupied and the mechanism for active transport out of the tubules

is overloaded. Other substances reabsorbed by active transport include sodium, calcium, potassium, phosphate and chloride.

The transport maximum, or renal threshold, of some substances varies according to body need at a particular time, and in some cases reabsorption is regulated by hormones.

Hormones that influence selective reabsorption 13.7

Parathyroid hormone. This is secreted by the parathyroid glands and together with *calcitonin* from the thyroid gland regulates the reabsorption of calcium and phosphate from the distal collecting tubules, so that normal blood levels are maintained. Parathyroid hormone increases the blood calcium level and calcitonin lowers it.

Antidiuretic hormone, ADH. This is secreted by the posterior pituitary. It increases the permeability of the distal convoluted tubules and collecting tubules, increasing water reabsorption. Secretion of ADH is controlled by a negative feedback system (Fig. 13.12).

Aldosterone. Secreted by the adrenal cortex, this hormone increases the reabsorption of sodium and water, and the excretion of potassium. Secretion is regulated through a negative feedback system (Fig. 13.13).

Atrial natriuretic peptide, ANP. This hormone is secreted by the atria of the heart in response to stretching of the atrial wall when blood volume is increased. It decreases reabsorption of sodium and water from the proximal convoluted tubules and collecting ducts. Secretion of ANP is also regulated by a negative feedback system (Fig. 13.14).

Tubular secretion (Fig. 13.11) 13.8

Filtration occurs as blood flows through the glomerulus. Substances not required and foreign materials, e.g. drugs including penicillin and aspirin, may not be entirely filtered out of the blood because of the short time it remains in the glomerulus. Such substances are cleared by secretion from the peritubular capillaries into the filtrate within the convoluted tubules. Tubular secretion of hydrogen ions (H^+) is important in maintaining normal blood pH.

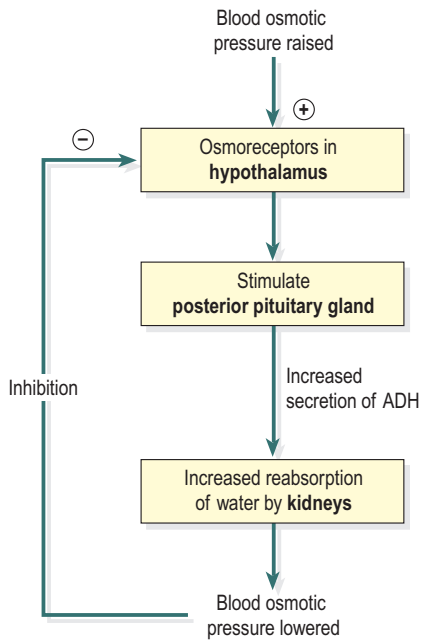


Figure 13.12 Negative feedback regulation of secretion of antidiuretic hormone (ADH).

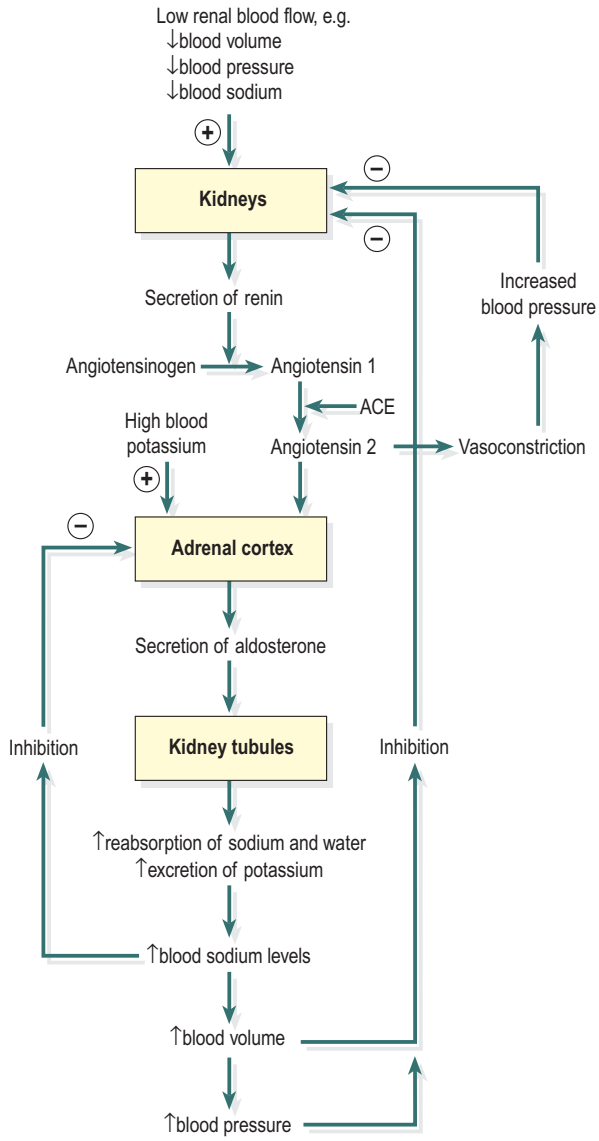


Figure 13.13 Negative feedback regulation of aldosterone secretion. ACE = angiotensin converting enzyme.

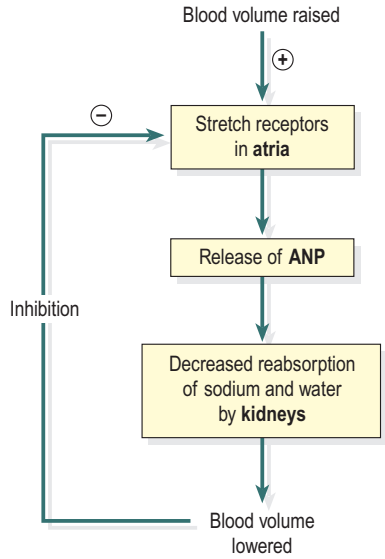


Figure 13.14 Negative feedback regulation of secretion of atrial natriuretic peptide (ANP).

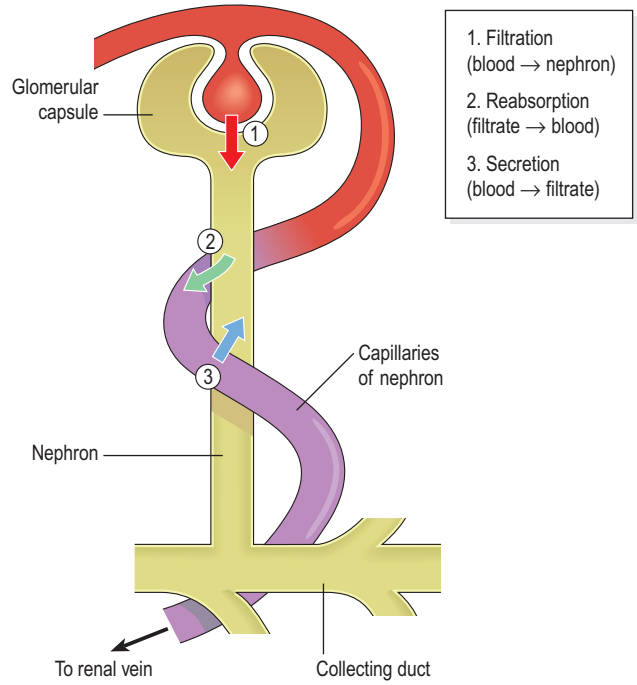


Figure 13.15 Summary of the three processes that form urine.

Summary of urine formation

The three processes involved – filtration, selective reabsorption and tubular secretion – are described above and summarised in Figure 13.15.

Composition of urine

Urine is clear and amber in colour due to the presence of urobilin, a bile pigment altered in the intestine, reabsorbed then excreted by the kidneys (see Fig. 12.37, p. 311). The specific gravity is between 1020 and 1030, and the pH is around 6 (normal range 4.5–8). A healthy adult passes from 1000 to 1500 mL per day. The volume of urine produced and the specific gravity vary according to fluid intake and the amount of solute excreted. The constituents of urine are:

Water	96%		
Urea	2%		
Uric acid, Creatinine, Ammonia, Sodium, Potassium	2%	Chlorides, Phosphates, Sulphates, Oxalates	2%

Water balance and urine output

The source of most body water is dietary food and fluid although a small amount (called 'metabolic water') is formed by cellular metabolism. Water is excreted as the main constituent of urine, in expired air, faeces and through the skin as sweat. The amount lost in expired air and faeces is fairly constant; the amount of sweat produced is associated with environmental and body temperatures.

The balance between fluid intake and output is controlled by the kidneys. The minimum urinary output, i.e. the smallest volume required to excrete body waste products, is about 500 mL per day. Urinary volume in excess of this is controlled mainly by antidiuretic hormone (ADH) released into the blood by the posterior pituitary gland.

Sensory nerve cells in the hypothalamus (osmoreceptors) detect changes in the osmotic pressure of the blood. Nerve impulses from the osmoreceptors stimulate the posterior pituitary to release ADH. When the osmotic pressure is raised, i.e. the blood is becoming more concentrated, ADH output is increased and as a result, water reabsorption by the distal convoluted tubules and collecting ducts is increased, reducing the blood osmotic pressure and ADH output. This negative feedback mechanism maintains the blood osmotic pressure (and therefore sodium and water concentrations) within normal limits (see Fig. 13.12).

This negative feedback mechanism may be over-ridden even though there may be an excessive amount of a dissolved substance in the blood. For example, in diabetes mellitus when blood glucose levels exceed the transport capacity of the renal tubules the excess glucose remains in the filtrate, drawing water with it. Large volumes of urine are passed (*polyuria*), which may lead to dehydration despite increased ADH secretion. However acute thirst and increased water intake usually compensate for the polyuria, at least to some extent.

When blood volume is increased, stretch receptors in the atria of the heart are stimulated and cardiac muscle cells release atrial natriuretic hormone (ANP). This reduces reabsorption of sodium and water by the proximal convoluted tubules and collecting ducts, meaning

that more sodium and water are excreted. In turn, this lowers blood volume and reduces atrial stretching, and through the negative feedback mechanism ANP secretion is switched off (see Fig. 13.14). Raised ANP levels also inhibit secretion of ADH and aldosterone, further promoting loss of sodium and water.

Electrolyte balance

Changes in the concentration of electrolytes in the body fluids may be due to changes in:

- the body water content, or
- electrolyte levels.

Several mechanisms maintain the balance between water and electrolyte concentration.

Sodium and potassium balance

Sodium is the most common cation (positively charged ion) in extracellular fluid and potassium is the most common intracellular cation.

Sodium is a constituent of almost all foods and, furthermore, salt is often added to food during cooking. This means that intake is usually in excess of the body's needs. It is excreted mainly in urine and sweat.

The amount of sodium excreted in sweat is usually insignificant unless sweating is excessive. This may occur when there is pyrexia (fever), a high environmental temperature or during sustained physical exercise. Normally the renin–angiotensin–aldosterone mechanism (described below) maintains the concentration of sodium and potassium within physiological limits. When excessive sweating is sustained, e.g. living in a hot climate or working in a hot environment, acclimatisation occurs in approximately 7 to 10 days and electrolyte secretion lost in sweat is reduced.

Sodium and potassium occur in high concentrations in digestive juices – sodium in gastric juice and potassium in pancreatic and intestinal juice. Normally these ions are reabsorbed by the colon, but following acute and prolonged diarrhoea they may be excreted in large quantities causing electrolyte imbalance.

Renin–angiotensin–aldosterone system. (Fig. 13.13) Sodium is a normal constituent of urine and its excretion is regulated by the hormone *aldosterone*, secreted by the adrenal cortex. Cells in the afferent arteriole of the nephron release the enzyme *renin* in response to sympathetic stimulation, low blood volume or by low arterial blood pressure. Renin converts the plasma protein *angiotensinogen*, produced by the liver, to angiotensin 1. *Angiotensin converting enzyme* (ACE), formed in small quantities in the lungs, proximal convoluted tubules and other tissues, converts angiotensin 1 into angiotensin 2, which is a very potent vasoconstrictor and increases blood pressure. Renin and raised blood potassium levels also stimulate the adrenal gland to secrete aldosterone. Water is reabsorbed with sodium and together they increase the blood volume, which reduces renin secretion through the negative feedback mechanism. When sodium reabsorption is increased potassium excretion is increased, indirectly reducing intracellular potassium. Profound diuresis may lead to hypokalaemia (low blood potassium levels).

ANP. This hormone is also involved in the regulation of sodium levels (Fig. 13.14).

Calcium balance

Regulation of calcium levels is maintained by secretion of parathyroid hormone and calcitonin (see Ch. 9).

pH balance 13.9

In order to maintain normal blood pH (acid–base balance), the proximal convoluted tubules secrete hydrogen ions into the filtrate where they combine with buffers (p. 25):

- bicarbonate, forming carbonic acid ($H^+ + HCO_3^- \rightarrow H_2CO$)
- ammonia, forming ammonium ions ($H^+ + NH3 \rightarrow NH_4^+$)
- hydrogen phosphate, forming dihydrogen phosphate ($H^+ + HPO_3^{2-} \rightarrow H_2PO_3^-$)

Carbonic acid is converted to carbon dioxide (CO_2) and water (H_2O), and the CO_2 is reabsorbed, maintaining the buffering capacity of the blood. Hydrogen ions are excreted in the urine as ammonium salts and hydrogen phosphate. The normal pH of urine varies from 4.5 to 8 depending on diet, time of day and other factors. Individuals whose diet contains a large amount of animal proteins tend to produce more acidic urine (lower pH) than vegetarians.

Ureters

Learning outcome

After studying this section, you should be able to:

- outline the structure and function of the ureters.

The ureters carry urine from the kidneys to the urinary bladder (Fig. 13.16). They are about 25–30 cm long with a diameter of approximately 3 mm. The ureter is continuous with the funnel-shaped renal pelvis. It passes downwards through the abdominal cavity, behind the peritoneum in front of the psoas muscle into the pelvic cavity, and passes obliquely through the posterior wall of the bladder (Fig. 13.17). Because of this arrangement, as urine accumulates and the pressure in the bladder rises, the ureters are compressed and the openings into the

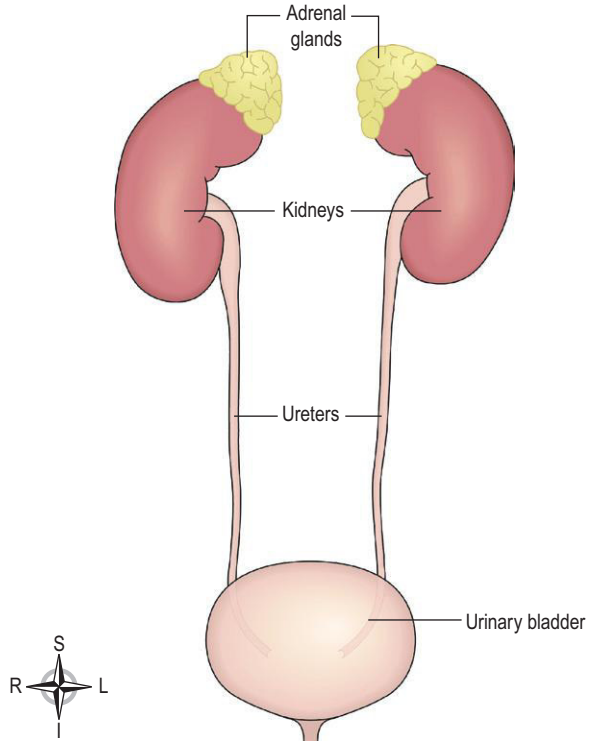


Figure 13.16 The ureters and their relationship to the kidneys and bladder.

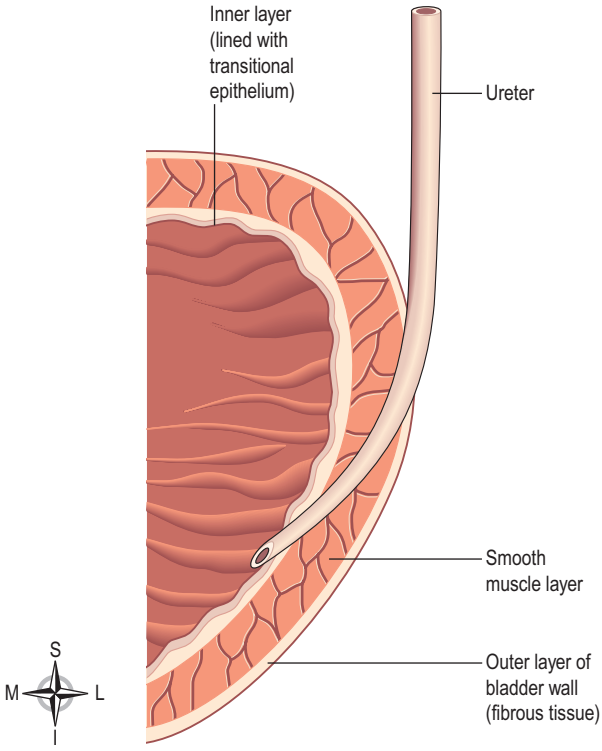


Figure 13.17 The position of the ureter where it passes through the bladder wall.

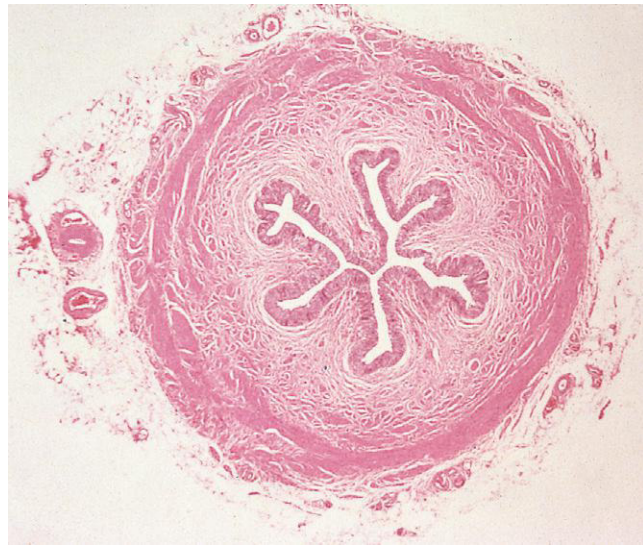


Figure 13.18 Cross-section of the ureter.

bladder are occluded. This prevents backflow (reflux) of urine into the ureters (towards the kidneys) as the bladder fills and also during micturition, when pressure increases as the muscular bladder wall contracts.

Structure 13.10

The walls of the ureters consist of three layers of tissue which are shown in cross-section in Figure 13.18:

- an outer covering of *fibrous tissue*, continuous with the fibrous capsule of the kidney
- a middle *muscular layer* consisting of interlacing smooth muscle fibres that form a functional unit round the ureter and an additional outer longitudinal layer in the lower third
- an inner layer, the *mucosa*, composed of transitional epithelium (see Fig. 3.15, p. 39).

Function

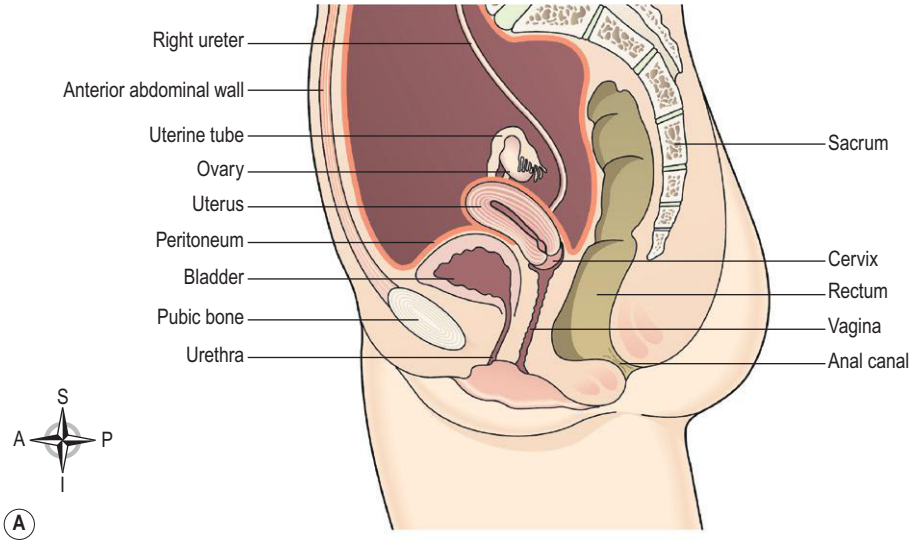
Peristalsis is an intrinsic property of the smooth muscle layer that propels urine along the ureter. Peristaltic waves occur several times per minute, increasing in frequency with the volume of urine produced, sending little spurts of urine along the ureter towards the bladder.

Urinary bladder

Learning outcome

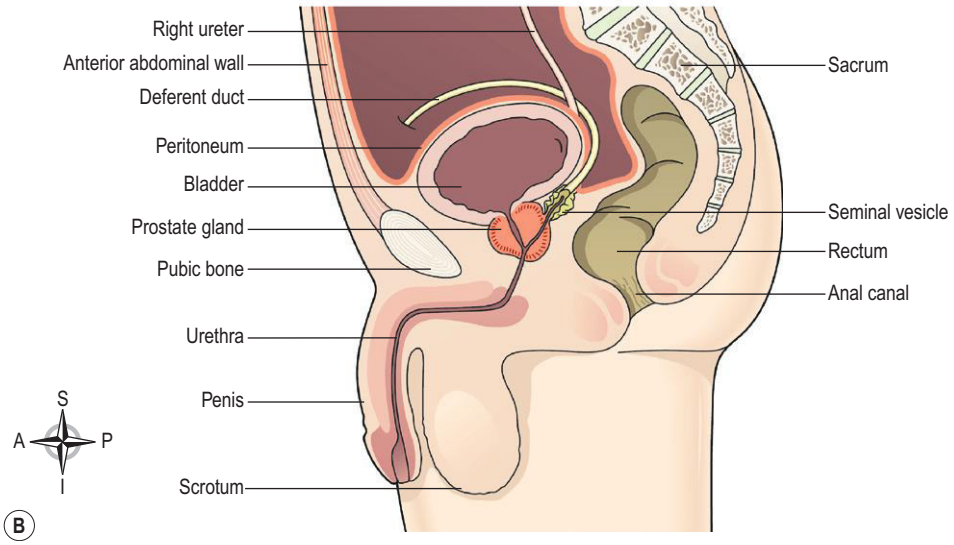
After studying this section, you should be able to:

- describe the structure of the bladder.



Organs associated with the bladder in the female

Anteriorly — the symphysis pubis
Posteriorly — the uterus and upper part of the vagina
Superiorly — the small intestine
Inferiorly — the urethra and the muscles forming the pelvic floor



Organs associated with the bladder in the male

Anteriorly — the symphysis pubis
Posteriorly — the rectum and seminal vesicles
Superiorly — the small intestine
Inferiorly — the urethra and prostate gland

Figure 13.19 The pelvic organs associated with the bladder and the urethra. A. The female. **B.** The male.

The urinary bladder is a reservoir for urine. It lies in the pelvic cavity and its size and position vary, depending on the volume of urine it contains. When distended, the bladder rises into the abdominal cavity.

Organs associated with the bladder

See Figure 13.19.

Structure (Fig. 13.20) 13.11

The bladder is roughly pear shaped, but becomes more balloon shaped as it fills with urine. The posterior surface is the *base*. The bladder opens into the urethra at its lowest point, the *neck*.

The peritoneum covers only the superior surface before it turns upwards as the parietal peritoneum, lining the anterior abdominal wall. Posteriorly it surrounds the uterus in the female and the rectum in the male. The bladder wall is composed of three layers:

- the outer layer of loose connective tissue, containing blood and lymphatic vessels and nerves, covered on the upper surface by the peritoneum
- the middle layer, consisting of interlacing smooth muscle fibres and elastic tissue loosely arranged in three layers. This is called the *detrusor muscle* and when it contracts, it empties the bladder
- the inner mucosa, composed of transitional epithelium (Fig. 3.15, p. 39) that readily permits distension of the bladder as it fills.

When the bladder is empty the inner lining is arranged in folds, or rugae, which gradually disappear as it fills. The bladder is distensible but as it fills, awareness of the

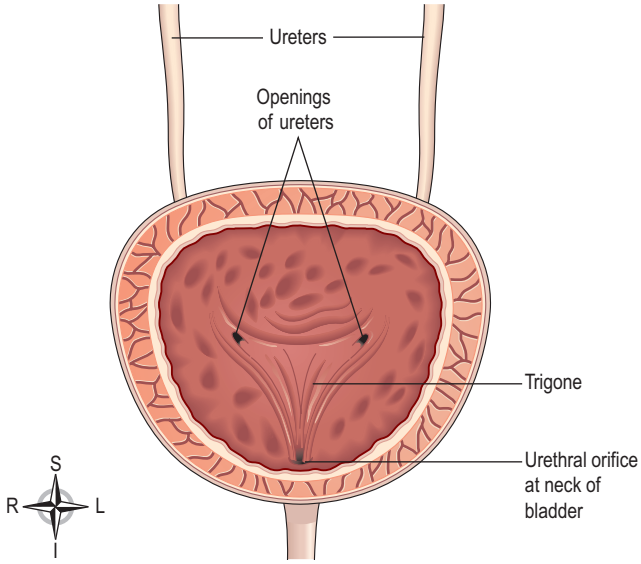


Figure 13.20 Section of the bladder showing the trigone.

need to pass urine is felt. The total capacity is rarely more than about 600 mL.

The three orifices in the bladder wall form a triangle or *trigone* (Fig. 13.20). The upper two orifices on the posterior wall are the openings of the ureters; the lower orifice is the opening into the urethra. The *internal urethral sphincter*, a thickening of the urethral smooth muscle layer in the upper part of the urethra, controls outflow of urine from the bladder. This sphincter is not under voluntary control.

Urethra

Learning outcome

After studying this section, you should be able to:

- outline the structure and function of the urethra in males and females.

The urethra is a canal extending from the neck of the bladder to the exterior, at the external urethral orifice. It is longer in the male than in the female.

The male urethra is associated with both the urinary and reproductive systems, and is described in detail in Chapter 18. **13.12**

The female urethra is approximately 4 cm long and 6 mm in diameter. It runs downwards and forwards behind the symphysis pubis and opens at the *external urethral orifice* just in front of the vagina. The external urethral orifice is guarded by the *external urethral sphincter*, which is under voluntary control.

The wall of the female urethra has two main layers: an outer muscle layer and an inner lining of mucosa, which is continuous with that of the bladder. The muscle layer has two parts, an inner layer of smooth muscle that is under autonomic nerve control, and an outer layer of striated (voluntary) muscle surrounding it. The striated muscle forms the external urethral sphincter and is under voluntary control. The mucosa is supported by loose fibroelastic connective tissue containing blood vessels and nerves. Proximally it consists of transitional epithelium while distally it is composed of stratified epithelium.

Micturition

Learning outcome

After studying this section, you should be able to:

- compare and contrast the process of micturition in infants and adults.

In infants, accumulation of urine in the bladder activates stretch receptors in the bladder wall generating sensory (afferent) impulses that are transmitted to the spinal cord, where a *spinal reflex* (p. 164) is initiated. This stimulates involuntary contraction of the detrusor muscle and relaxation of the internal urethral sphincter (Fig. 13.21), and expels urine from the bladder – this is *micturition* or voiding of urine.

When bladder control is established, the micturition reflex is still stimulated but sensory impulses also pass upwards to the brain and there is awareness of the need to pass urine as the bladder fills (around 300–400 mL in adults). By learned and conscious effort, contraction of the external urethral sphincter and muscles of the pelvic floor can inhibit micturition until it is convenient to pass urine (Fig. 13.22).

Urination can be assisted by increasing the pressure within the pelvic cavity, achieved by lowering the diaphragm and contracting the abdominal muscles. Overdistension of the bladder is extremely painful, and when this occurs there is a tendency for involuntary relaxation of the external sphincter to occur allowing a small amount of urine to escape, provided there is no mechanical obstruction. *Incontinence* is the involuntary loss of urine after bladder control has been established.

The effects of ageing of the urinary system

Learning outcome

After studying this section, you should be able to:

- describe the effects of ageing on the urinary system.

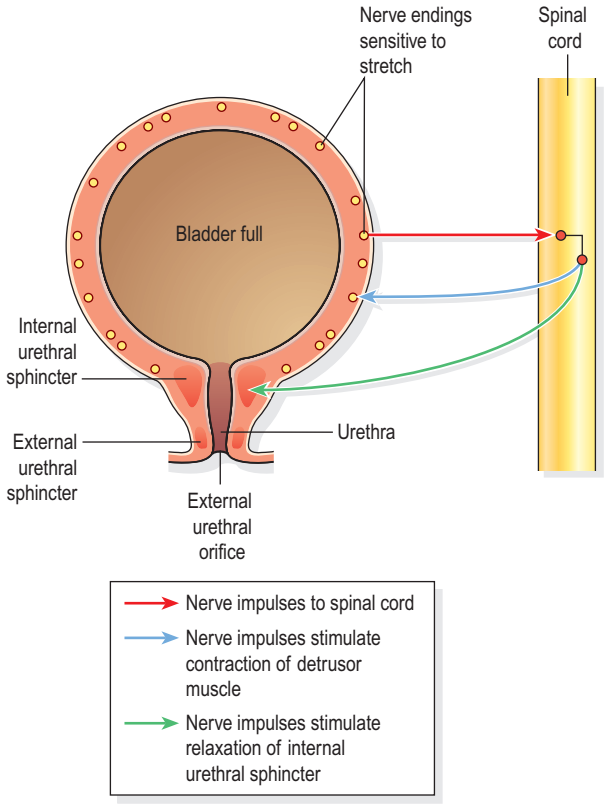


Figure 13.21 Reflex control of micturition when conscious effort cannot override the reflex action.

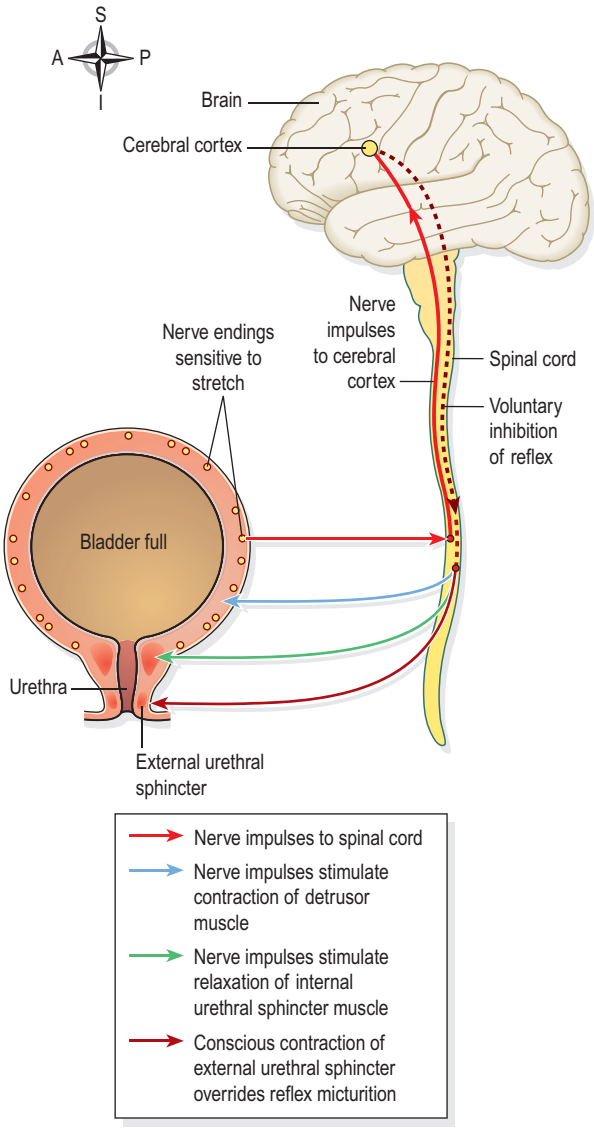


Figure 13.22 Control of micturition after bladder control is established.

The kidneys have a substantial functional reserve; the loss of one kidney does not cause problems in an otherwise healthy individual. The number of nephrons declines with age, glomerular filtration rate falls and the renal tubules function less efficiently; the kidneys become less able to concentrate urine.

These changes mean that older adults become more sensitive to alterations in fluid balance, and problems associated with fluid overload or dehydration are more prevalent. Elimination of drugs also becomes less efficient with declining kidney function which may lead to accumulation and toxicity.

The ability to inhibit contraction of the detrusor muscle declines and may result in the urgent need to pass urine and urinary frequency. Nocturia becomes increasingly common in older adults. Incontinence (p. 356) is more prevalent in older adults affecting 15% of women and 10% of men over 65, figures which double by 85 years of age. Enlargement of the prostate gland is common in older men and may cause retention of urine and problems with micturition (see Ch. 18).

Diseases of the kidneys

Learning outcomes

After studying this section, you should be able to:

- outline the principal effects of glomerulonephritis
- describe the effects of diabetes mellitus and hypertension on kidney function
- discuss the sources and consequences of kidney infections
- explain the causes and implications of acute and chronic renal failure
- describe the pathogenesis of kidney stones
- list common congenital abnormalities of the kidneys
- outline the development and spread of common kidney tumours.

As the kidneys have considerable functional reserve impairment of renal function does not become evident until the equivalent of more than one kidney is lost. This is why it is possible for a person with healthy kidneys to donate one for transplantation. Table 13.1 lists common signs and symptoms of renal disorders.

Table 13.1 Common signs and symptoms of disorders of the urinary system

Sign/symptom	Definition and description
Oliguria	Urine output less than 400 mL per day
Haematuria	Presence of blood in the urine. Leaky glomeruli allow red blood cells to escape from the glomerular capillaries and they cannot be reabsorbed from the filtrate as they are too large. Bleeding in the urinary tract also causes haematuria
Proteinuria	Presence of protein in the urine. This is abnormal and occurs when leaky glomeruli allow plasma proteins to escape into the filtrate but they are too large to be reabsorbed
Anuria	Absence of urine
Dysuria	Pain on passing urine, often described as a burning sensation
Glycosuria	Presence of sugar in the urine. This is abnormal and occurs in diabetes mellitus (see p. 236)
Ketonuria	Presence of ketones in the urine. This is abnormal and occurs in, e.g., starvation, diabetes mellitus
Nocturia	Passing urine during the night
Polyuria	Passing unusually large amounts of urine
Frequency of micturition	Requiring to pass, often small amounts of, urine frequently
Incontinence	Involuntary loss of urine (p. 356)

Glomerulonephritis (GN)

This term suggests inflammatory conditions of the glomerulus, but there are several types of GN and inflammatory changes are not always present. In many cases GN has an autoimmune component which leads to production of immune complexes that may lodge in the glomerular capillaries causing inflammation and impairment of glomerular filtration. Other immune mechanisms are also implicated in GN.

Classification of GN is based on a number of features: the cause, immunological characteristics and findings on microscopy. Microscopic distinction is based on:

- the extent of damage:
  - *diffuse*: affecting all glomeruli
  - *focal*: affecting some glomeruli
- appearance:
  - *proliferative*: increased number of cells in the glomeruli
  - *membranous*: thickening of the glomerular basement membrane.

Examples of different types of GN, their causes, features and prognoses are shown in Table 13.2.

Effects of glomerulonephritis

These depend on the type and are listed below.

Haematuria. This is usually painless and not accompanied by other symptoms. When microscopic, it may be found on routine urinalysis when red blood cells have passed through damaged glomeruli into the filtrate.

Overt haematuria occurs when there is considerable escape of red blood cells into the renal tubules while smaller amounts give urine a smoky appearance.

Asymptomatic proteinuria. Damaged glomeruli may allow protein to escape from the blood into the filtrate, which may be asymptomatic and only found during routine urinalysis, however there are also other causes of asymptomatic proteinuria e.g. urinary tract infection. Significant proteinuria is associated with nephrotic syndrome (p. 351).

Acute nephritis. This is characterised by the presence of:

- oliguria (<400 mL urine/day in adults)
- hypertension
- haematuria
- uraemia (p. 353).

Loin pain, headache and malaise are also common.

Table 13.2 Glomerulonephritis: features and prognosis of different types

Type	Cause	Presenting features	Prognosis
Diffuse proliferative GN	Usually follows a transient infection, especially *β-haemolytic Streptococcus* but also other microbes	Acute nephritis Haematuria Proteinuria	Good in children; less good in adults, up to 40% develop hypertension or chronic renal failure
Focal proliferative GN	Associated with other systemic conditions, e.g. systemic lupus erythematosus (p. 434), infective endocarditis (p. 128)	Acute nephritis Haematuria Proteinuria	Variable
Membranous GN	Only sometimes has an identified cause such as infection, e.g. syphilis, malaria, hepatitis B; some drugs, e.g. penicillamine, gold, diamorphine; tumours	Nephrotic syndrome Haematuria Proteinuria	Variable, but most cases progress to chronic renal failure as sclerosis of glomeruli progresses
Minimal-change GN	Unknown	Nephrotic syndrome Haematuria Proteinuria	Good in children, but recurrences are common in adults

Nephrotic syndrome. (See below.)

Chronic renal failure. (p. 353) This occurs when nephrons are progressively and irreversibly damaged after the renal reserve is lost.

Nephrotic syndrome

This is not a disease in itself but is an important feature of several kidney diseases. The main characteristics are:

- marked proteinuria
- hypoalbuminaemia
- generalised oedema
- hyperlipidaemia.

When glomeruli are damaged, the permeability of the glomerular membrane increases and plasma proteins pass through into the filtrate. Albumin is the main protein lost because it is the most common and is the smallest of the plasma proteins. When the daily loss exceeds the rate of production by the liver there is a significant fall in the total plasma protein level. The consequent low plasma osmotic pressure leads to widespread oedema and reduced plasma volume (see Fig. 5.57, p. 125). This reduces the renal blood flow and stimulates the renin–angiotensin–aldosterone system (Fig. 13.13), causing increased reabsorption of water and sodium from the renal tubules. The reabsorbed water further reduces the blood osmotic pressure and increases the oedema. The key factor is the loss of albumin across the glomerular membrane and as long as this continues, the vicious circle is perpetuated (Fig. 13.23). Levels of nitrogenous waste products, i.e. uric acid, urea and creatinine, usually remain normal. Hyperlipidaemia, especially hypercholesterolaemia, also occurs but the cause is unknown.

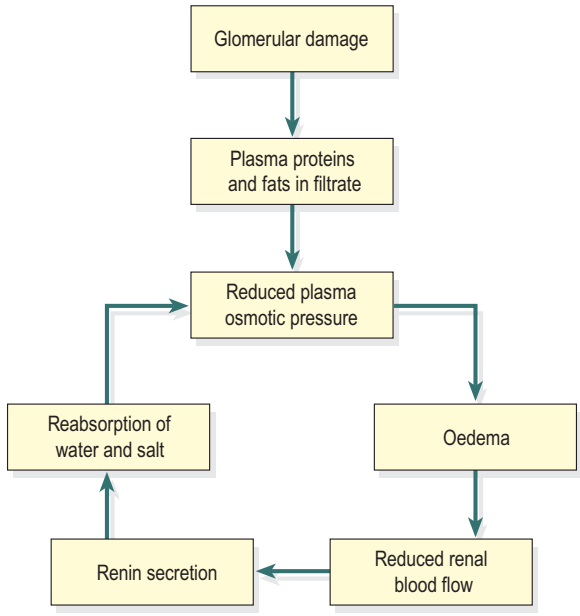


Figure 13.23 Stages of development of nephrotic syndrome.

Nephrotic syndrome occurs in a number of diseases. In children the most common cause is minimal-change glomerulonephritis. In adults it may complicate:

- most forms of glomerulonephritis
- diabetic nephropathy (see below)
- systemic lupus erythematosus (p. 434)
- infections, e.g. malaria, syphilis, hepatitis B
- drugs, e.g. penicillamine, gold, captopril, phenytoin.

Diabetic nephropathy

Renal failure is the commonest cause of death in young people with diabetes mellitus (p. 236), especially if hypertension and severe, long-standing hyperglycaemia are also present. Diabetes causes damage to large and small blood vessels throughout the body, although the effects vary considerably between individuals. In the kidney, these are known collectively as *diabetic nephropathy* or *diabetic kidney* and include:

- progressive damage of glomeruli, proteinuria and nephrotic syndrome
- ascending infection leading to acute pyelonephritis
- atheroma (see Ch. 5) of the renal arteries and their branches leading to renal ischaemia and hypertension
- chronic renal failure (p. 353).

Hypertension and the kidneys

Hypertension can be the cause or the result of renal disease. Essential and secondary hypertension (p. 131) both affect the kidneys when renal blood vessel damage causes ischaemia. The reduced blood flow stimulates the renin–angiotensin–aldosterone system (see Fig. 13.13), raising the blood pressure still further.

Rising blood pressure is common in older adult (p. 117) and can cause gradual and progressive damage to the glomeruli, which may lead to renal failure after the renal reserve has been lost or to malignant hypertension.

Secondary hypertension

This can be caused by long-standing kidney diseases (described in this chapter) and may lead to chronic renal ischaemia, worsening hypertension and renal failure.

Malignant hypertension

Damage to arterioles spreads to the glomeruli with subsequent destruction of nephrons and leads to a further rise in blood pressure and a variable degree of renal impairment. Sometimes there are more serious effects; increased permeability of the glomeruli allows escape of plasma proteins and red blood cells into the filtrate causing proteinuria and haematuria, which may progress to renal failure.

Acute pyelonephritis

This is acute bacterial infection of the renal pelvis and calyces, which spreads to the kidney substance causing small abscesses. Bacteria usually reach the kidney by travelling up the urinary tract from the perineum but are sometimes blood-borne. This condition is accompanied by fever, malaise and loin pain.

Ascending infection. Upward spread of bacteria from the bladder (see cystitis, p. 356) is the most common cause of this condition. Reflux of infected urine into the ureters when the bladder contracts during micturition predisposes to upward spread of infection to the renal pelves and kidney substance. Normally the relative positions of the ureters and bladder (Fig. 13.16) prevents reflux that permits ascending access of bacteria to the kidneys.

Blood-borne infection. The kidneys are susceptible to blood-borne infection due to their large blood supply (20% of cardiac output). Bacteria may reach the kidney directly in septicaemia or travel there from distant sites e.g. a respiratory tract infection, infected wound or abscess.

Pathophysiology

Bacterial infection of the kidney tissues causes suppuration and destruction of nephrons. The prognosis depends on the amount of healthy kidney tissue remaining after the infection subsides. Necrotic tissue is eventually replaced by fibrous tissue but there may be some hypertrophy of healthy nephrons. The outcomes are healing; recurrence, especially if there is a structural abnormality of the urinary tract; and reflux nephropathy. Papillary necrosis is a rare complication, usually occurring if the condition is untreated.

Reflux nephropathy

Previously known as chronic pyelonephritis, this is almost always associated with reflux of urine from the bladder to the ureter allowing spread of infection upwards towards the kidneys. A congenital abnormality of the angle of insertion of the ureter into the bladder predisposes to this, but it is sometimes caused by an obstruction that develops later in life. Progressive damage to the renal papillae and collecting ducts may lead to chronic renal failure and concurrent hypertension is common.

Renal failure

Acute renal failure

The causes of acute renal failure are classified as:

- *prerenal*: the result of reduced renal blood flow, especially as a consequence of, e.g., severe and prolonged shock
- *renal*: due to damage to the kidney itself, e.g. acute tubular necrosis, glomerulonephritis
- *postrenal*: arises from obstruction to the outflow of urine, e.g. disease of the prostate gland, tumour of the bladder, uterus or cervix, large calculus (stone) in the renal pelvis.

There is a sudden and severe reduction in the glomerular filtration rate and kidney function that is often reversible over days or weeks if treated. Oliguria or anuria is accompanied by metabolic acidosis due to retention of H^+, electrolyte imbalance and accumulation of mainly nitrogenous

Box 13.2 Some causes of ATN

Ischaemia – severe shock, dehydration, haemorrhage, trauma; extensive burns; myocardial infarction; prolonged and complex surgery, especially in older people

Drugs – e.g. aminoglycoside antibiotics, non-steroidal anti-inflammatory drugs (NSAIDs), ACE inhibitors, lithium compounds, paracetamol overdose

Haemoglobinaemia – accumulation of haemoglobin, released by haemolysis of red blood cells, e.g. incompatible blood transfusion, malaria

Myoglobinaemia – accumulation of myoglobin released from damaged muscle raises blood levels, e.g. following crush injury.

waste products. This occurs as a complication of conditions not necessarily associated with the kidneys.

Acute tubular necrosis (ATN)

This is the most common cause of acute renal failure. There is severe damage to the tubular epithelial cells caused by ischaemia or, less often, by nephrotoxic substances (Box 13.2).

Oliguria, *severe oliguria* (less than 100 mL of urine per day in adults) or *anuria* (see Table 13.1) may last for a few weeks, followed by profound diuresis. There is a reduction in glomerular filtration, selective reabsorption and secretion by the tubules, leading to:

- heart failure due to fluid overload
- generalised and pulmonary oedema
- accumulation of urea and other metabolic wastes
- electrolyte imbalance which may be exacerbated by the retention of potassium (hyperkalaemia) released from damaged cells anywhere in the body
- acidosis due to retention of hydrogen ions.

Profound diuresis (the diuretic phase) occurs during the healing process when the epithelial cells of the tubules have regenerated but are still incapable of selective reabsorption and secretion. Diuresis may lead to acute dehydration, complicating the existing high plasma urea, acidosis and electrolyte imbalance. If the patient survives the initial acute phase, a considerable degree of renal function is usually restored over several weeks (the recovery phase).

Chronic renal failure

Also known as chronic kidney disease (CKD), this is present when GFR has fallen to around 20% of normal. Onset is usually slow and asymptomatic, progressing over several years. The main causes are diabetes mellitus, glomerulonephritis and hypertension.

The effects on glomerular filtration rate (GFR), selective reabsorption and tubular secretion are significant.

Table 13.3 Polyuria in chronic renal failure

	Normal kidney	End-stage kidney
GFR	125 mL/min or 180 L/day	10 mL/min or 14 L/day
Reabsorption of water	>99%	Approx. 30%
Urine output	<1 mL/min or 1.5 L/day	Approx. 7 mL/min or 10 L/day

GFR and filtrate volumes are greatly reduced, and reabsorption of water is seriously impaired. This results in production of up to 10 litres of urine per day (Table 13.3). Reduced glomerular filtration leads to accumulation of waste substances in the blood, notably urea and creatinine. When renal failure becomes evident, blood urea levels are raised and this is referred to as *uraemia*. Some of the signs and symptoms that may accompany this condition include nausea, vomiting, gastrointestinal bleeding, anaemia and pruritus (itching). Others are explained below.

Polyuria. Large volumes of dilute urine (with a low specific gravity) are passed, because water reabsorption is impaired. *Nocturia* is a common presenting symptom.

Acidosis. As the kidney buffer system that normally controls the pH of body fluids fails, hydrogen ions accumulate.

Electrolyte imbalance. This is also the result of impaired tubular reabsorption and secretion.

Anaemia. Deficiency of erythropoietin (p. 66) occurs after a few months, causing anaemia that is exacerbated by haemodialysis which damages red blood cells. If untreated, anaemia results in fatigue, and may also lead to dyspnoea and cardiac failure (p. 126). Tiredness and breathlessness are sometimes the initial symptoms of chronic renal failure.

Hypertension. This is often a consequence, if not a cause, of renal failure.

End-stage renal failure

When death is likely without renal replacement therapy, such as haemodialysis, peritoneal dialysis or a kidney transplant, the condition is referred to as *end-stage renal failure*. The excretory functions of the kidneys are lost, acid–base balance cannot be maintained and endocrine functions of the kidney are disrupted.

Towards the end of life anorexia, nausea and very deep (Kussmaul's) respirations occur as uraemia progresses. In the final stages there may be hiccoughs, itching, vomiting, muscle twitching, seizures, drowsiness and coma.

Renal calculi 13.13

Calculi (stones) form in the kidneys and bladder when urinary constituents normally in solution, usually oxalate and phosphate salts, are precipitated. They are more common in males and after 30 years of age and often recur. Most originate in the collecting tubules or renal papillae. They then pass into the renal pelvis where they may increase in size. Some become too large to pass through the ureter and may obstruct the outflow of urine, causing kidney damage. Others pass to the bladder and are either excreted or increase in size and obstruct the urethra (Fig. 13.24). In developing countries and often in children, stones sometimes originate in the bladder. Predisposing factors include:

- *dehydration*: this leads to increased reabsorption of water from the tubules but does not change solute reabsorption, resulting in a low volume of highly concentrated filtrate in the collecting tubules
- *pH of urine*: when the normally acid filtrate becomes alkaline, some substances may be precipitated, e.g. phosphates. This occurs when the kidney buffering system is impaired and in some infections
- *infection*: necrotic material and pus provide foci upon which solutes in the filtrate may be deposited and the products of infection may alter the pH of the urine. Infection sometimes leads to alkaline urine (see above)
- *metabolic conditions*: these include hyperparathyroidism (p. 233) and gout (p. 434).

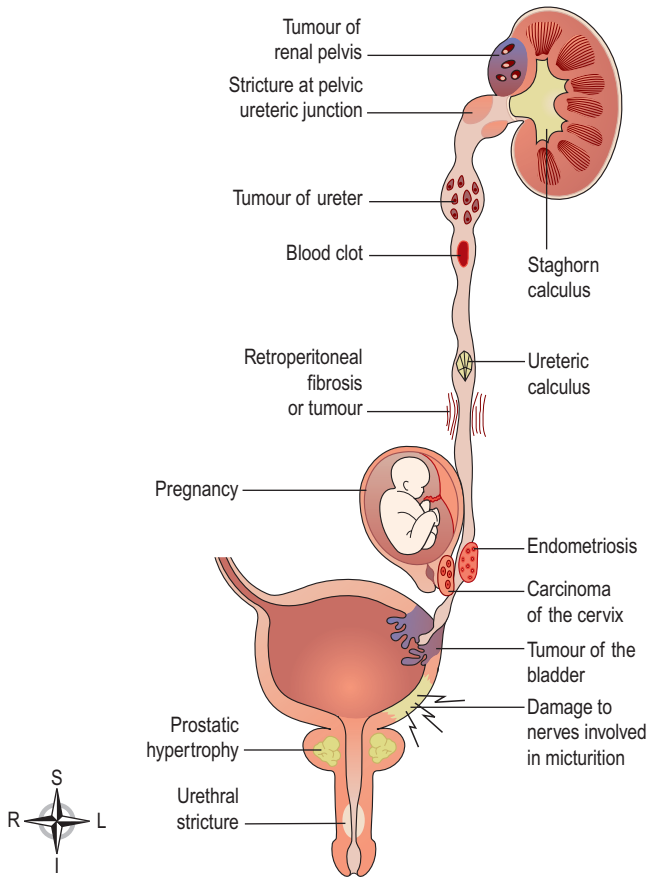


Figure 13.24 Summary of obstructions of the urinary tract.

Small calculi 13.14

These may pass through or become impacted in a ureter and damage the epithelium, leading to haematuria and after healing, fibrosis and stricture. In ureteric obstruction, which is usually unilateral, there is spasmodic contraction of the ureter, causing acute intermittent ischaemic pain (*renal colic*) as the smooth muscle of the ureter contracts over the stone in an attempt to move it. Stones reaching the bladder may be passed in urine or increase in size and eventually obstruct the urethra. Consequences include retention of urine and bilateral hydronephrosis (p. 355), infection proximal to the blockage, pyelonephritis and severe kidney damage.

Large calculi (staghorn calculus)

One large stone may form, usually over many years, filling the renal pelvis and the calyces (see Fig. 13.24). It causes stagnation of urine, predisposing to infection, hydronephrosis and occasionally kidney tumours. It may cause chronic renal failure.

Congenital abnormalities of the kidneys

Misplaced (ectopic) kidney

One or both kidneys may develop in abnormally low positions. Misplaced kidneys function normally if the blood vessels are long enough to provide an adequate blood supply but a kidney in the pelvic cavity may cause problems during pregnancy as the expanding uterus compresses renal blood vessels or the ureters. If the ureters become kinked there is increased risk of infection as there is a tendency for reflux and backflow to the kidney. There may also be difficulties during childbirth.

Polycystic disease

The *infantile form* is very rare and is usually fatal in early childhood.

Autosomal dominant polycystic kidney disease (ADPKD). This is inherited as an autosomal dominant condition (Ch. 17) that may become apparent any time between childhood and late adult life. Both kidneys are affected. Dilations (cysts) form at the junction of the distal convoluted tubules and collecting ducts. The cysts slowly enlarge and pressure causes ischaemia and destruction of nephrons. The disease is progressive and secondary hypertension is common; chronic renal failure affects

about 50% of patients. Death may be due to chronic renal failure, cardiac failure or subarachnoid haemorrhage due to increased incidence of berry aneurysms of the circulus arteriosus (p. 105). Cysts may also develop in the liver, spleen and pancreas but are not associated with dysfunction of these organs.

Tumours of the kidney

Benign tumours are relatively uncommon.

Malignant tumours

These are most common in the bladder or kidney.

Renal adenocarcinoma

This tumour of tubular epithelium is more common after 50 years of age, and in males. Clinical features include haematuria, back or loin pain, anaemia, weight loss and fever. Local spread involves the renal vein and leads to early blood spread of tumour fragments, most commonly to the lungs and bones. The causes are unknown although there is an increased incidence in cigarette smokers.

Nephroblastoma (Wilms' tumour)

This is one of the most common malignant tumours in children under 10 years, usually occurring in the first 4 years. Clinical features include haematuria, hypertension, abdominal pain and, sometimes, intestinal obstruction. It is usually unilateral but rapidly becomes very large and invades the renal blood vessels, causing early blood spread to the lungs.

Diseases of the renal pelvis, ureters, bladder and urethra

Learning outcomes

After studying this section, you should be able to:

- describe the causes and implications of urinary obstruction
- explain the pathological features of urinary tract infections
- outline the characteristics of the main bladder tumours
- discuss the principal causes of urinary incontinence.

These structures are considered together because their combined functions are to collect and store urine prior to excretion. Obstruction and infection are the main problems (Fig. 13.24).

Obstruction to the outflow of urine

Hydronephrosis 13.15

This is dilation of the renal pelvis and calyces caused by accumulation of urine above an obstruction in the urinary tract (Fig. 13.24). It leads to destruction of the nephrons, fibrosis and atrophy of the kidney. One or both kidneys may be involved, depending on the cause and site. When there is an abnormality of the bladder or urethra, both kidneys are affected whereas an obstruction above the bladder is more common and affects only one kidney. The effects depend on the site and extent of the obstruction. Stasis of urine within the urinary tract predisposes to infection.

Complete sustained obstruction

In this condition hydronephrosis develops quickly, pressure in the nephrons rises and urine production stops. The most common causes are a large calculus or tumour. The outcome depends on whether one or both kidneys are involved (adequate renal function can be maintained by one kidney).

Partial or intermittent obstruction

This may progress undetected for many years. It leads to progressive hydronephrosis and is caused by, e.g.:

- a succession of renal calculi in a ureter, eventually moved onwards by peristalsis
- constriction of a ureter or the urethra by fibrous tissue, following epithelial inflammation caused by the passage of a stone or by infection
- a tumour in the urinary tract or in the abdominal or pelvic cavity
- enlarged prostate gland in the male.

Spinal lesions

When the nerve supply to the bladder is interrupted, e.g. transverse spinal cord lesions, micturition does not occur. When the bladder fills, the rise in pressure causes overflow incontinence (p. 356), back pressure into the ureters and hydronephrosis. Reflex micturition is usually re-established after a time, but loss of voluntary control may be irreversible. Pressure on the spinal cord and other abnormalities, e.g. spina bifida, can also impair micturition.

Urinary tract infections (UTIs)

Infection of any part of the urinary tract may spread upwards causing acute pyelonephritis (p. 352) and kidney damage.

Ureteritis

Inflammation of a ureter is usually due to the upward spread of infection in cystitis.

Cystitis

This is inflammation of the bladder and may be due to:

- upward spread of commensal bacteria of the bowel (*Escherichia coli* and *Streptococcus faecalis*) from the perineum via the urethra, especially in women
- trauma, with or without infection, following health-care interventions, e.g. radiotherapy, insertion of a urinary catheter or instrument into the bladder.

The effects of inflammation include oedema and small haemorrhages of the mucosa, which may be accompanied by *haematuria*. The sensory nerve endings in the bladder wall become hypersensitive and are stimulated when the bladder contains only small volumes of urine, leading to *frequency of micturition* and *dysuria*. The urine may appear cloudy and have an unpleasant smell. Lower abdominal pain often accompanies cystitis. If untreated, upward spread may cause acute pyelonephritis (see p. 352) or septicaemia.

Cystitis is *uncomplicated* when it occurs in otherwise healthy individuals with a normal urinary tract. When it affects people with structural or functional abnormalities of the urinary tract or those with pre-existing conditions, e.g. diabetes mellitus or urinary outflow obstruction, it is described as *complicated*. Complicated UTIs sometimes cause permanent renal damage, whereas this is very rare in uncomplicated infections. Recurrence is fairly common, especially in women, either when the original infection is not eradicated or reinfection occurs.

Predisposing factors. These include stasis of urine in the bladder and the shorter female urethra, which is close to the anus (Fig. 13.19A), and the moist perineal conditions there that may harbour commensal microbes. Sexual intercourse may cause trauma to the urethra and transfer of microbes from the perineum, especially in the female. Hormones associated with pregnancy relax perineal muscle, and cause relaxation and kinking of the ureters. Towards the end of pregnancy, pressure caused by the fetus may obstruct the outflow of urine. In the male, prostatitis provides a focus of local infection or an enlarged prostate gland may cause progressive urethral obstruction.

Urethritis

This is inflammation of the urethra and is described in Chapter 18.

Tumours of the bladder

It is not always clear whether bladder tumours are benign or malignant. Tumours are often multiple and recurrence is common. Predisposing factors include cigarette smoking, prolonged use of certain analgesics and occupational exposure to some chemicals, e.g. aniline dyes used in the textile and printing industries.

Transitional cell carcinomas

These tumours, also known as papillomas, arise from transitional epithelium and are often benign. They consist of a stalk with fine-branching fronds, which tend to break off causing painless bleeding and haemturia. Papillomas commonly recur, even when benign. Sometimes the tumour cells are well differentiated and non-invasive but in other cases they behave as carcinomas and invade surrounding blood and lymph vessels.

Solid tumours

These are all malignant to some degree. At an early stage the more malignant and solid tumours rapidly invade the bladder wall and spread in lymph and blood to other parts of the body. If the surface ulcerates there may be haemorrhage and necrosis.

Urinary incontinence

In this condition normal micturition (p. 348) is affected and there is involuntary loss of urine. Several types are recognised and described below. In addition to the mechanisms below, neurological abnormalities can also impair voluntary control of micturition, e.g. spinal cord injury, multiple sclerosis (p. 185).

Stress incontinence

This is leakage of urine when intra-abdominal pressure is raised, e.g. on coughing, laughing, sneezing or lifting. It usually affects women when there is weakness of the pelvic floor muscles or pelvic ligaments, e.g. after childbirth or as part of the ageing process. It occurs physiologically in young children before bladder control is achieved.

Urge incontinence

Leakage of urine follows a sudden and intense urge to void and there is inability to delay passing urine. This may be due to a urinary tract infection, calculus, tumour or hypersensitivity of the detrusor muscle.

Overflow incontinence

This occurs when there is chronic overfilling of the bladder and may be due to retention of urine due to incomplete voiding when there is obstruction of urinary outflow, e.g. enlarged prostate gland or urethral stricture and/or impaired contraction of the detrusor muscle during micturition. It may also arise as a complication of pelvic nerve damage caused by e.g. surgery, trauma, or when the cauda equina is compressed by a tumor or prolapsed intervertebral disc.

The bladder becomes distended and when the pressure inside overcomes the resistance of the external urethral sphincter, urine dribbles from the urethra. There may be difficulty initiating and/or maintaining micturition. Larger than normal residual volumes of urine in the bladder (>50–100 mL) predispose to infection.

For a range of self-assessment exercises on the topics in this chapter, visit Evolve online resources: https://evolve.elsevier.com/Waugh/anatomy/