

Functional Anatomy of the Kidneys and Overview of Kidney Function

7.2 Function

Learning Objectives

- Identify the gross anatomical structures of the renal system including renal artery and vein, ureter, urinary bladder, and urethra
- Identify: renal medulla, renal cortex, renal capsule
- Distinguish between cortical and juxtamedullary nephrons
- Identify: afferent arteriole, efferent arteriole, glomerulus, Bowman's capsule, proximal convoluted tubule, proximal straight tubule, loop of Henle, distal tubule, collecting duct, peritubular capillaries, vasa recta, juxtaglomerular apparatus, macula densa
- Describe the signals that turn on renin secretion
- Describe the action of renin on angiotensinogen
- List the main functions of angiotensin II
- List the functions of the kidney unrelated to the formation of urine

FUNCTION FOLLOWS FORM IN FUNCTIONAL UNITS CALLED NEPHRONS

As discussed in Chapter 7.1, the kidneys regulate the volume and composition of the body fluids and it accomplishes this task by acting solely on the plasma. The effects of kidneys transfer to the interstitial fluid and to the intracellular fluid by altering water flow and material transport across the barriers between these compartments. The remainder of renal physiology seeks to answer the question: how does the renal system alter plasma volume and composition? The full answer is complicated, but a partial answer is that all functions of the kidney occur in miniature in small functional units called **nephrons**. Their mechanisms in turn are entirely dependent on transport properties of nephron parts and the spatial arrangement of these parts within the kidneys. The function of the nephrons follows their form and understanding how the kidneys accomplish their task requires an understanding of the structure of the component nephrons and their spatial arrangement within the kidney.

THE PAIRED KIDNEYS HAVE AN ENORMOUS BLOOD SUPPLY AND DRAIN URINE INTO THE BLADDER THROUGH THE URETERS

The kidneys are bean-shaped organs that lie against the back of the abdominal wall beneath the **peritoneum**, the membranous connective tissue sheet that lines the abdominal cavity and reflects back over all of the intestines. The two kidneys together weigh about 300 g or about 0.4% of the body mass. The blood supply is provided by the **renal artery** that comes directly off the **abdominal aorta** and is drained by the **renal vein** that drains into the **inferior vena cava**. These large vessels deliver about 20% of the cardiac output, or about 1 L min^{-1} , to the kidneys. This is an enormous fraction of the cardiac output for the size of the kidneys and it provides the first clue to their operation: they must do something to the blood other than extract oxygen from it. The kidneys clear the blood of waste products and adjust plasma volume and composition. To do this, the blood has to run through the kidneys at a high rate, far more than is required for the metabolism of the kidneys (see [Figure 7.2.1](#)).

The waste products and excess fluid and electrolytes are disposed of in the urine. The kidneys collect the urine from their many nephrons into the **ureter**. The ureter is a tube that contains smooth muscle. The ureters can actively propel the urine into the bladder by **peristalsis**, a wave of muscular contraction that begins in the kidney and continues to the urinary bladder, which also has a layer of smooth muscle. The urinary bladder stores the urine and voids it to the outside of the body through the **urethra**. Urination requires the active contraction of the bladder and relaxation of the urinary sphincters.

INTERMEDIATE LEVEL OF KIDNEY STRUCTURE REVEALS FUNCTIONAL AREAS

A longitudinal cross-section of the kidney shows two easily distinguished parts (see [Figure 7.2.2](#)). The **cortex** lies beneath the tough **renal capsule** and has a reddish-brown

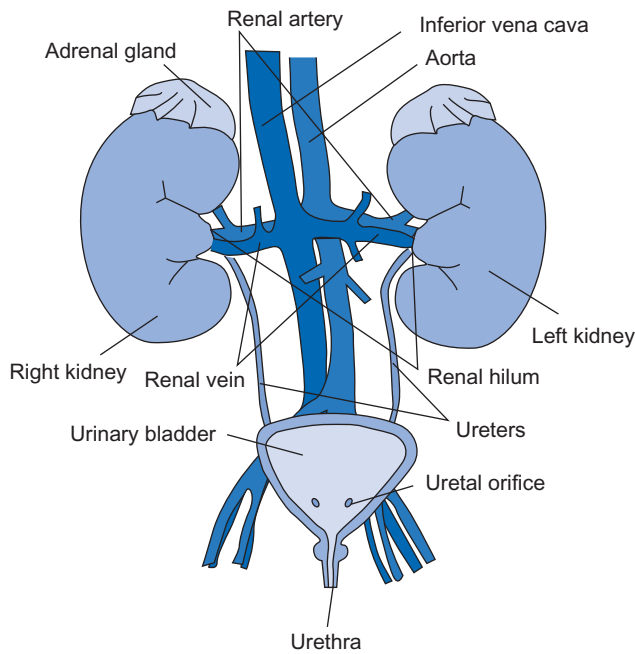


FIGURE 7.2.1 Diagram of the anatomic arrangement of the urinary system. The kidneys are paired structures located behind the peritoneum, a connective tissue sheet that lines the abdominal cavity. The hilum is a longitudinal slit in the medial aspect of the kidney through which the renal artery enters and the renal vein and ureters exit. The kidneys are also supplied by afferent and efferent nerves.

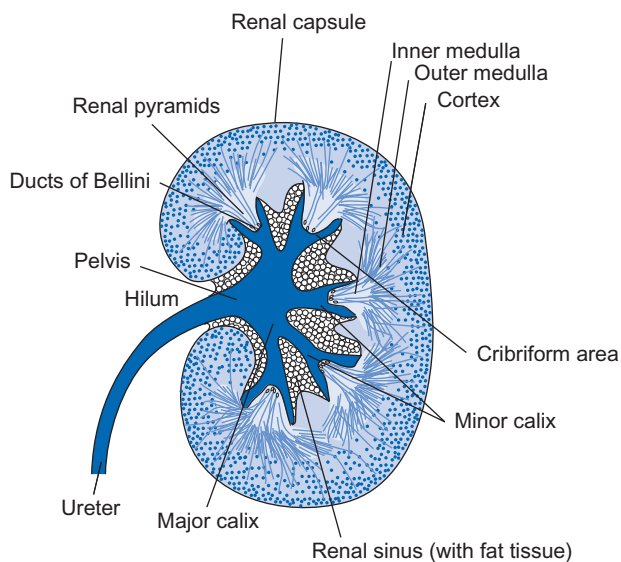


FIGURE 7.2.2 Longitudinal cross-section of the kidney. The reddish-brown, granular cortex lies immediately below the tough connective tissue capsule. The medulla lies more interiorly. Urine is formed inside tiny tubules that eventually coalesce to form ever-larger tubules, eventually forming the ducts of Bellini. These penetrate through the cribriform area of the renal pyramids and drain their fluid, the urine, into the minor calices. The urine eventually drains into the ureters and from there into the urinary bladder from which it is eliminated from the body.

and granular appearance. The cortex contains many **glomeruli** (singular is **glomerulus**), which are little balls of capillaries that are all crammed together and surrounded by a thin epithelial capsule called **Bowman's capsule**. The

granular appearance is due to the many glomeruli in the cortex. The **medulla** comprises the inner part of the kidney. The medulla in turn consists of an outer part and an inner part. The outer part has a striated appearance that is due to the many tubules running from the cortex down into the medulla. These tubules are part of the nephrons, the functional units of the kidneys, and the tubules collect into structures called **renal pyramids**. Each kidney typically has 8–10 of these renal pyramids. The tubules coalesce into progressively larger tubules, the **collecting ducts**. These collecting ducts in turn fuse to form **ducts of Bellini**, which pierce through the apex of the renal pyramids in a series of 18–24 tiny holes in each pyramid. This flattened area of the renal pyramid that is pierced by the ducts of Bellini is called the **cribriform area**. Urine in its final stages passes through the tiny orifices in the cribriform area to reach a minor calix (from the Greek “kalyx,” meaning “cup”). Two or more minor calices fuse to form a major calix. The urine eventually collects into the **ureters**, which drain into the urinary bladder.

THE RENAL ARTERIES ARISE FROM THE ABDOMINAL AORTA

The single renal artery enters the hilum and then branches to form the **interlobar arteries**, so-named because they pass between the lobes of the kidney. At the junction of the cortex and medulla, the interlobar arteries bend over to form incomplete arches. Accordingly, this section of the blood supply is called the **arcuate artery**. Many small **radial arteries** branch off at right angles from the arcuate artery, carrying blood toward the cortex. These radial arteries give rise to short lateral branches called **afferent arterioles** that supply blood to the glomeruli.

The venous drainage of the kidney follows the arterial supply. **Radial veins** arise from stellate (“star shaped”) sinuses in the superficial cortex that drain capillaries from the cortex. These penetrate the cortex and join the **arcuate veins** that arch over the renal pyramids. The arcuate veins join to form the **interlobar veins**, which in turn join to form the single **renal vein**. The anatomy of the kidney's vasculature is shown in [Figure 7.2.3](#).

THE FUNCTIONAL UNIT OF THE KIDNEY, THE NEPHRON, PARTICIPATES IN ALL ELEMENTARY RENAL PROCESSES

The kidneys use three elementary processes to produce urine and to regulate the volume and composition of the plasma. These are as follows:

1. Ultrafiltration
2. Secretion
3. Reabsorption.

All of these elementary processes occur in each nephron and so these nephrons are functional units of renal function. Each kidney contains some 1–1.3 million of these nephrons.

THE NEPHRON IS A TUBULE WITH FUNCTIONALLY AND MICROSCOPICALLY DISTINCT REGIONS

The nephrons are all long tubes with parts that are oriented approximately parallel to the renal capsule, or

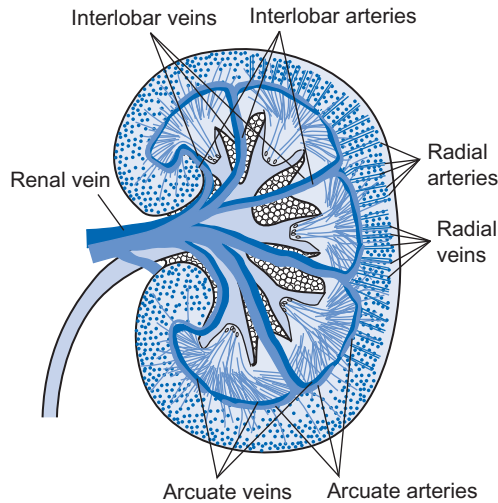


FIGURE 7.2.3 Diagram of the vasculature of the kidney. Blood enters the kidney through the single renal artery that breaks up into several interlobar arteries. These penetrate the renal columns where, at the junction of the cortex and medulla, they bend back over the bases of the renal pyramids, forming the arcuate arteries. The radial arteries come off the arcuate arteries at right angles and these supply blood to the cortex. The afferent arterioles that supply blood to the glomeruli are short lateral branches of the radial arteries. The venous drainage more or less follows the arterial supply except that the arcuate veins form complete arches over the renal pyramids whereas the arcuate arteries form incomplete arches.

perpendicular to it. Each portion of the tube has its own set of transport properties. There are two basic kinds of nephrons: **cortical nephrons** and **juxtamedullary nephrons**. These distinctions have to do with the location of the glomerulus, the tiny ball of capillary network, and the penetration into the medulla by the loops of the nephron tubule. Cortical nephrons have a glomerulus located nearer to the outer parts of the cortex and their **loops of Henle** are short. Juxtamedullary nephrons have a glomerulus near the junction of the cortex and medulla and their loops of Henle penetrate deep into the medulla. The relative number of cortical and juxtamedullary nephrons and the lengths of their loops of Henle determine the ability of the kidney to concentrate the urine. In humans, about 85% of the nephrons are cortical nephrons and about 15% are juxtamedullary nephrons. Kangaroo rats, which live in extremely arid environments, never need to drink water. Their only source of water is from metabolism of food. They achieve this feat by having extraordinarily long loops of Henle (see [Figure 7.2.4](#) for a diagrammatic description of nephron structure).

Although the blood supply is technically not part of the nephron, its anatomical arrangement is crucial to renal function. We have already traced the macroscopic blood flow: blood enters the kidney by the single **renal artery** that divides into the **interlobar arteries**. These curve back over the base of the renal pyramids to form the **arcuate arteries**. **Radial arteries** arise at right angles to the arcuate arteries and rise through the cortex. Short lateral branches of the radial arteries are called the **afferent arterioles**. The afferent arterioles supply blood to the **glomerulus** and regulate its hydrostatic pressure.

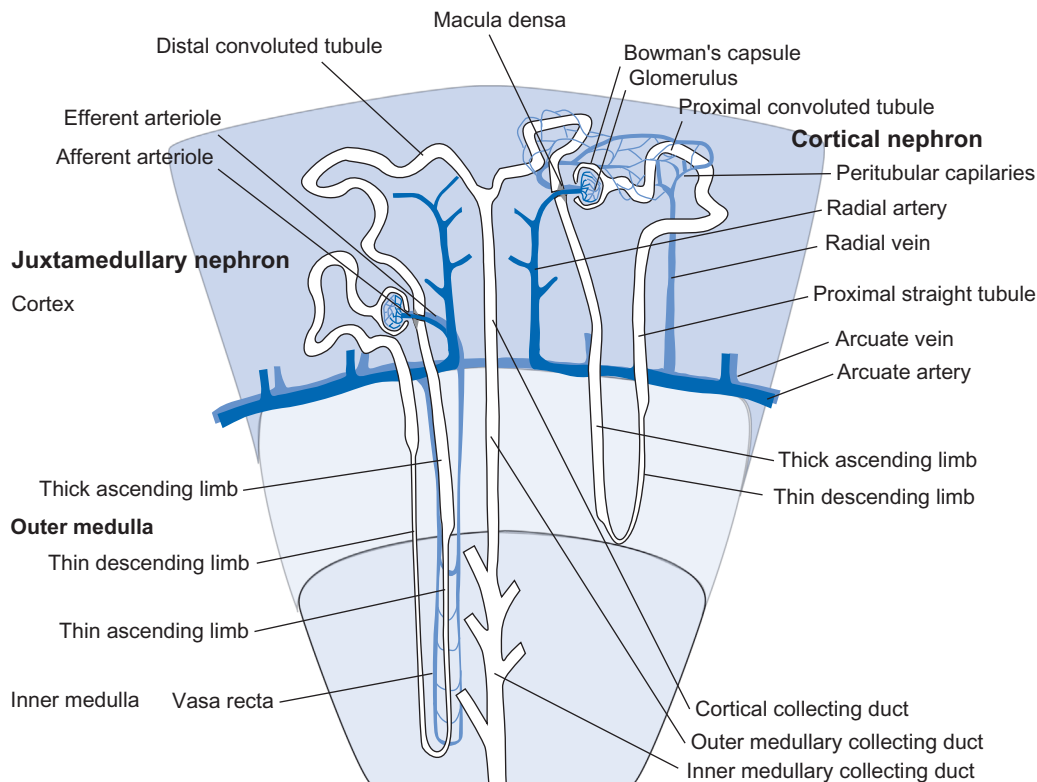


FIGURE 7.2.4 Diagram of nephron structure. Structures are not drawn to scale. Two general kinds of nephrons are shown: a cortical nephron and a juxtamedullary nephron. Blood supply is partially shown with most capillaries omitted. See text for explanation.

The glomerulus is a group of anastomosing capillaries that, together with its closely apposed **Bowman's capsule**, forms an ultrafiltrate of plasma. This ultrafiltration is the first step in the formation of urine.

After leaving the glomerulus, blood enters the **efferent arteriole**. Thus the capillary network of the glomerulus is highly unusual in that it sits between two arterioles rather than between an arteriole and a venule. It is a high-pressure capillary bed. The efferent arteriole helps regulate the fraction of plasma that is filtered by the glomerulus. The fate of blood in the efferent arterioles depends on the location of the glomerulus. For cortical nephrons, the efferent arterioles break up into an anastomosing network of capillaries called the **peritubular capillaries**. These supply oxygen to cells in the nephron to energize transport processes and they provide materials for secretion and a conduit to remove materials that are reabsorbed from the ultrafiltrate. These capillaries eventually drain into sinuses that drain into the **radial veins**, which drain into the **arcuate veins**, into the **interlobar veins** and, finally, into the single **renal vein**.

In juxtamedullary nephrons, the efferent arterioles give rise to the **vasa recta**, which plunges down into the renal papilla to supply blood to that tissue. The **descending vasa recta** and **ascending vasa recta** make a loop. The vasa recta forms a capillary network within the renal medulla. It acts as a **countercurrent exchanger** which exchanges material with the medullary interstitium without destroying the osmotic gradient that is established there. These ideas will be discussed in depth in Chapter 7.5.

Now let us trace the path of urine formation. Urine formation begins in **Bowman's space**. As mentioned earlier, the glomerulus together with the closely apposed **Bowman's capsule** forms an ultrafiltrate of plasma, and this is the first step in the formation of urine. This ultrafiltrate flows through the tube of the nephron, traveling first through the **proximal convoluted tubule** where many of the filtered substances are reabsorbed from the ultrafiltrate including water, salt, amino acids, glucose, and bicarbonate. Secretion of some materials into the incipient urine also occurs here. Fluid then enters the **loop of Henle**. The loop of Henle can be considered to be a loop of nephron tubule oriented perpendicular to the surface of the capsule of the kidney: it descends through the cortex into the medulla, makes a hairpin turn, and returns again to the cortex. This arrangement is crucial to its function as a **countercurrent multiplier** to establish an osmotic gradient that runs from 300 mOsm in the cortex to as high as 1200 mOsm in the inner medullary interstitium. This osmotic gradient is essential for the kidney to concentrate urine. How this is accomplished will be discussed in Chapter 7.5. For now, you should learn to associate functional descriptors with the various parts of the nephron: the glomerulus is an ultrafilter; the proximal convoluted tubule reabsorbs and secretes; the vasa recta is a countercurrent exchanger; the loop of Henle is a countercurrent multiplier.

The loop of Henle consists of all of the tubule parts between the proximal convoluted tubule and the distal convoluted tubule. In cortical nephrons, the loop of Henle is not so long, penetrating down through the outer medulla to the edge of the inner medulla. It consists of a proximal straight tubule, a thin descending limb, and a thick ascending limb. Each thick ascending limb returns to its own glomerulus where it lies closely apposed to the afferent and efferent arterioles of its own glomerulus. The complex of the thick ascending limb, afferent arteriole, and efferent arteriole is the **juxtaglomerular apparatus**. The thick ascending limb ends at this point and the **distal convoluted tubule** begins. Here additional reabsorption of water or salt occurs and the distal tubule connects to a **collecting duct**. The collecting duct may be considered as part of the nephron, but many distal convoluted tubules drain into a single collecting duct. Thus the collecting ducts individually belong to several nephrons. The final concentration or dilution of the urine occurs in the collecting ducts as the fluid flows successively through the **cortical collecting duct**, **outer medullary collecting duct**, and **inner medullary collecting duct**.

The structure of **juxtamedullary nephrons** differs from that of cortical nephrons. In juxtamedullary nephrons, the loop of Henle penetrates further into the inner medulla. It consists of the proximal straight tubule, thin descending limb, thin ascending limb, and thick ascending limb. Here the proximal straight tubule becomes the thin descending limb at about the same depth as that of the cortical nephrons. In juxtamedullary nephrons, the thin descending limb descends all the way to the renal papilla. It returns as a thin ascending limb to the junction of the inner and outer medulla, becoming the thick ascending limb. This thick limb returns to its own glomerulus, forming its own juxtaglomerular apparatus with it.

THE JUXTAGLOMERULAR APPARATUS PRODUCES RENIN

RENIN IS AN ENZYME RELEASED BY GRANULE CELLS IN THE AFFERENT ARTERIOLE

As mentioned above, the thick ascending limb of the loop of Henle rises out of the outer medulla and travels toward the glomerulus from which it arose. It makes close contact with the afferent arteriole and efferent arteriole at the point at which they enter and exit the glomerulus. At this point, the cortical thick ascending limb ends and the distal convoluted tubule begins. The region of close apposition of the afferent arteriole, efferent arteriole, and cortical thick ascending limb is called the **juxtaglomerular apparatus** because it is next to the glomerulus. This structure contains a number of specialized cells. The cells in the thick ascending limb adjacent to the arterioles form the **macula densa**. The volume of these cells responds to the [NaCl] in the tubular fluid. These macula densa cells send signals through **mesangial cells** to affect cells in the afferent arteriole. The afferent arteriole contains vascular smooth muscle cells and specialized **granule cells** that contain granules of

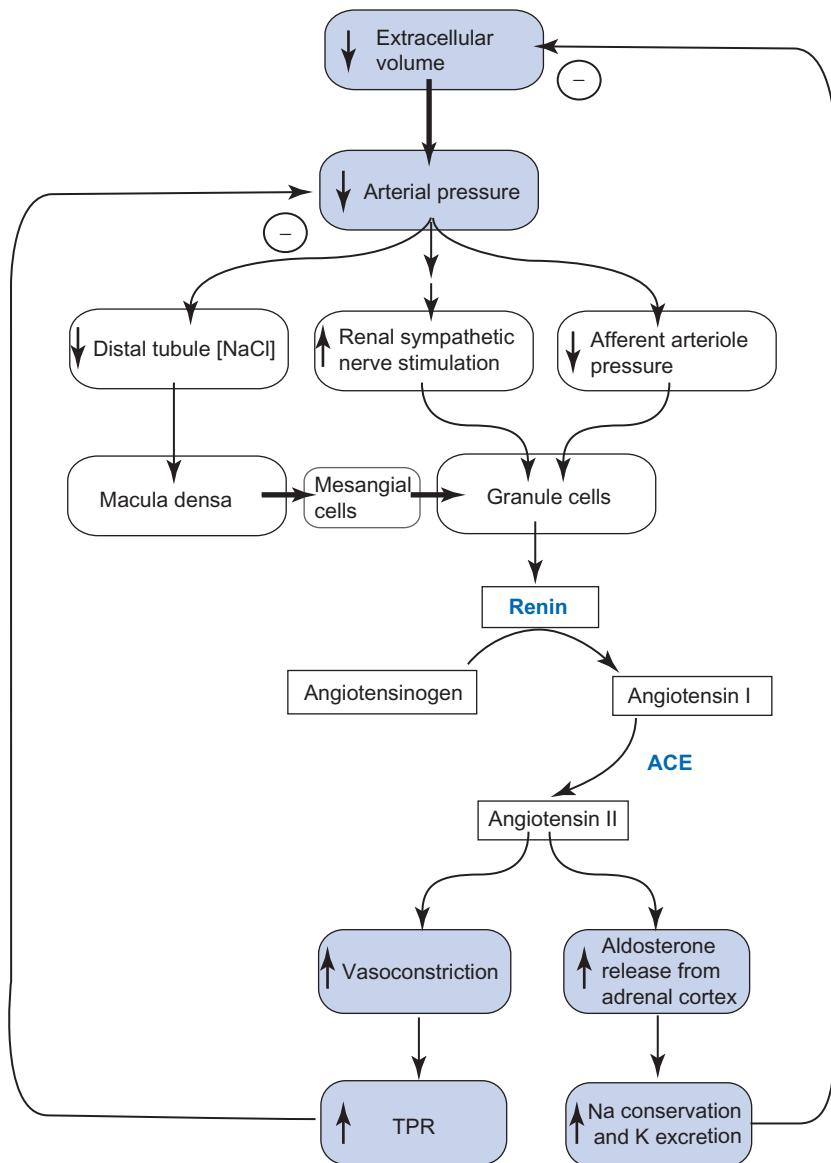


FIGURE 7.2.5 The renin–angiotensin–aldosterone system. Renin is an enzyme secreted by granule cells in the afferent arteriole in response to lower afferent arteriolar pressure, renal sympathetic nervous stimulation, or signals arriving from the macula densa and probably relayed through mesangial cells. Renin converts a precursor plasma protein, made in the liver, to angiotensin I, a decapeptide. Angiotensin I is further converted to angiotensin II by ACE, predominantly located in the lung. Angiotensin II has two major effects: it is a potent vasoconstrictor and it releases aldosterone from the adrenal cortex. The vasoconstrictor effects of angiotensin II increase total peripheral resistance, thereby tending to return the blood pressure toward normal. Thus the vasoconstrictor effects of angiotensin II complete a negative feedback loop in which the original perturbation, a decrease in blood pressure, is corrected. The increased circulating aldosterone has the effect of lessening Na^+ excretion and increasing K^+ excretion. The result is a tendency to preserve the extracellular volume, thereby defending against decrease in blood pressure. Thus the system forms a second negative feedback loop.

renin. These granule cells are also called juxtaglomerular or JG cells. Activation of the macula densa cells leads to afferent arteriolar constriction and release of renin into the circulation. The events leading to renin secretion and the consequences of renin secretion are shown in Figure 7.2.5.

RENIN CLIPS ANGIOTENSIN I OFF A PLASMA PRECURSOR, WHICH IS THEN CONVERTED TO ANGIOTENSIN II

Renin is an enzyme that converts an inactive plasma protein, **angiotensinogen**, to **angiotensin I**. This is the first step in the renin–angiotensin–aldosterone system of blood pressure and volume regulation that we will discuss in detail in Chapter 7.6. Angiotensin I is a polypeptide 10 amino acids long. It is converted to **angiotensin II** when **angiotensin converting enzyme (ACE)** clips off two amino acids. ACE is

mainly located in the lungs, but the kidney also has some ACE activity.

ANGIOTENSIN II VASOCONSTRICTS AND RELEASES ALDOSTERONE

Angiotensin II binds to AT_1 or AT_2 receptors, exerting two major effects: it is a potent vasoconstrictor of the resistance vessels of the body, thereby producing an increase in total peripheral resistance and a rise in blood pressure, and it releases **aldosterone** from the **adrenal cortex**. Aldosterone is a steroid hormone that conserves salt by decreasing salt excretion from the kidney. Although aldosterone cannot add NaCl to the extracellular fluid of its own accord, it can retain the NaCl that enters the body through the food. Since NaCl is largely extracellular, retaining salt increases the extracellular fluid volume, which tends to expand the blood volume. Thus the retention of salt caused by aldosterone indirectly tends to elevate blood pressure and cardiac output.

OUR EVOLVING KNOWLEDGE OF THE RAAS HAS ADDITIONAL COMPONENTS

The above description applies to the classical RAAS (renin–angiotensin–aldosterone system). Newer results show that another enzyme, ACE2, can cleave angiotensin II to produce angiotensin (1–7) that interacts with a different receptor, the Mas receptor, and that aminopeptidase A and aminopeptidase N cleave angiotensin II to angiotensin IV that interacts with the AT₄ receptor. In addition, granule cells release prorenin, a precursor form of renin, that binds to a prorenin receptor. The physiological importance of these accessory pathways remains to be clarified.

NONEXCRETORY FUNCTIONS OF THE KIDNEY

THE KIDNEYS PRODUCE THE HORMONE ERYTHROPOIETIN

In addition to its primary function of regulating the volume and composition of the plasma, and hence contributing to homeostasis of all body fluids, the kidney performs other essential functions. These include the secretion of **erythropoietin** in response to hypoxia. Erythropoietin is a glycoprotein hormone that stimulates the rate of formation of red blood cells or erythropoiesis. This forms another negative feedback loop: poor supply of oxygen to the kidneys stimulates the secretion of erythropoietin, which stimulates the formation of red blood cells that in turn increase the supply of oxygen to the kidney (see Chapter 5.2).

THE KIDNEYS ACTIVATE VITAMIN D

Vitamin D is synthesized in the skin and then subjected to two hydroxylation reactions. The first occurs in the liver and produces 25-OH-cholecalciferol. The second hydroxylation reaction occurs in mitochondria of renal proximal tubule cells and produces 1,25-(OH)₂ cholecalciferol, the active circulating form of vitamin D. The hydroxylation reaction in the kidney is stimulated by **parathyroid hormone**, PTH, and by low plasma [Pi]. 1,25-(OH)₂ cholecalciferol stimulates Ca²⁺ and Pi absorption from the intestine and helps maintain plasma [Ca²⁺] and [Pi] by actions on bone and kidney. The kidney hydroxylation reaction is crucial to the activity of vitamin D and forms part of the negative feedback loop that controls homeostasis of both plasma Ca²⁺ and phosphate. See Chapter 9.8 for detailed descriptions of these negative feedback loops.

KIDNEYS PRODUCE GLUCOSE BY GLUCONEOGENESIS

The kidneys help maintain blood glucose during fasting by converting amino acids into glucose. This metabolic process is called **gluconeogenesis**, which literally means “new glucose origination.”

THE KIDNEYS DEGRADE MANY HORMONES

Hormones generally are secreted in response to some stimulus in order to affect some system that then

counteracts the original stimulus. This is the essence of negative feedback loops and their role in maintaining homeostasis. To have effective control, the hormone signals must be shut off. Theoretically, this could be accomplished several ways. One way is to regulate negative feedback by a dual system in which hormone release and degradation are both regulated. A second, simpler solution is to regulate release of hormone and degrade the hormones according to first-order kinetics. In this case, the rate of degradation is proportional to hormone concentration. This is the typical manner of regulation of circulating hormone levels. The rate of release depends on the stimulus, whereas degradation is accomplished by mass action. The kidney participates in the degradation of a number of polypeptide hormones including angiotensin II, parathyroid hormone, insulin, and glucagon.

Clinical Applications: Kidney Failure

The best illustration of the importance of the kidney is what happens when it fails. Complete kidney failure, also called renal failure, universally leads to death unless the blood is cleansed by dialysis.

Such death is caused by a buildup of waste products and water and electrolyte imbalances.

Kidney failure can result from acute kidney injury, which is a rapid and progressive loss of renal function characterized by oliguria (low urine output, less than 400 mL per day for an adult) and fluid and electrolyte imbalance. There are a variety of causes, usually having to do with reduced blood supply to the kidney or when the kidneys are overwhelmed by toxins. Drug overdoses and crush syndrome are two examples. In the crush syndrome, breakdown of muscle tissue (rhabdomyolysis) releases myoglobin, potassium, and phosphate into the blood stream. The myoglobin clogs up the kidney and reduces its filtration, the first step in the formation of urine.

Kidney failure can also result from chronic conditions that progressively reduce kidney function. The most common cause is diabetes mellitus, which produces a nephropathy having to do with dysfunction of renal capillaries. Hypertension also slowly and progressively damages the kidney. Polycystic kidney disease is a third well-known cause of chronic kidney disease. Polycystic kidney disease has a known genetic component.

Clinicians use the glomerular filtration rate (see Chapter 7.3) as an indicator of the stages of renal failure. Without filtration, the kidney cannot rid the body of specific wastes such as creatinine and urea, and these substances build up in the blood. Accumulation of nitrogenous wastes in the blood is called **azotemia**. An older term for this is uremia, referring, literally, to urine in the blood. The term “uremia” is now used to describe the illness accompanying kidney failure. In the early stages of kidney failure, excretion of urea and creatinine may be near normal, but the normal excretion requires higher than normal plasma levels. At progressive stages of failure, urea and creatinine levels soar and the patient needs dialysis or kidney transplantation to remove these wastes.

(Continued)

Clinical Applications: Kidney Failure (Continued)

Symptoms of renal failure include the following:

- high levels of urea in the blood that can lead to nausea and weight loss;
- high levels of phosphate in the blood, leading to bone damage or failure to heal fractures and muscle cramping due to hypocalcemia;
- increased plasma $[K^+]$, leading to cardiac arrhythmias;
- swelling of the legs and ankles and shortness of breath;
- anemia due to reduced erythropoietin secretion;
- protein in the urine.

SUMMARY

The kidney has a well-defined microscopic anatomy that is integral to its function of regulating the volume and composition of body fluids by performing three fundamental operations on the plasma: ultrafiltration, secretion, and reabsorption. All three of these fundamental operations are performed by the functional unit of the kidney, the nephron.

Blood enters the kidney by the renal artery, which subdivides into the interlobar arteries, arcuate arteries, radial arteries, and finally the afferent arterioles. The afferent arterioles supply two major categories of nephrons: cortical nephrons and juxtamedullary nephrons, named for the location of the glomerulus within the kidney. The glomerulus is a group of capillaries located between the afferent arteriole and the efferent arteriole. Together with its closely apposed Bowman's capsule, the glomerulus produces an ultrafiltrate of plasma in the first step of forming the urine. The efferent arteriole in cortical nephrons forms the peritubular capillaries that carry off reabsorbed materials and supply blood for secretion by the first tubular part of the nephron, the proximal tubule. The efferent arterioles of the juxtamedullary nephrons make a hairpin loop to the inner medulla of the kidney, forming vessels called the vasa recta. Venous blood collects into radial veins, leading to arcuate veins and interlobar veins and eventually into the renal veins.

The nephron consists of the glomerulus, proximal tubule, loop of Henle, distal tubule, and collecting

ducts. The collecting ducts are formed from several nephrons, so they actually belong to a group of nephrons. Specific modification of the tubular fluid occurs along each segment of the nephron. The loop of Henle differs between the cortical nephrons, which have short loops, and the juxtamedullary nephrons, which have long loops. These loops form an osmotic gradient within the kidney interstitium that is crucial to the ability to concentrate urine.

The juxtaglomerular apparatus refers to the junction of the afferent arteriole, efferent arterial, and the thick ascending limb of the loop of Henle from the same nephron. Specialized granule cells in the afferent arteriole secrete renin, an enzyme that cleaves a plasma protein, angiotensinogen, to form angiotensin I. Angiotensin I is further cleaved by ACE to form angiotensin II. Angiotensin II has several effects, including vasoconstriction and release of a steroid hormone, aldosterone, from the adrenal cortex. The effects of angiotensin II are to restore blood pressure and increase the ECF volume. The major stimuli for renin release are as follows: (1) decreased afferent arteriolar pressure; (2) increased activity of renal sympathetic nerves; and (3) decrease in distal tubule $[NaCl]$.

REVIEW QUESTIONS

1. Trace the blood flow in the kidneys from the abdominal aorta through the kidneys and back through the veins to the inferior vena cava. How does the blood flow differ in cortical and juxtamedullary nephrons?
2. Trace the flow of fluid in the nephron from Bowman's capsule to the collecting duct. How does this differ in cortical and juxtamedullary nephrons?
3. What is the main function of the glomerulus together with its Bowman's capsule (the renal corpuscle)? Loop of Henle? Proximal convoluted tubule? Vasa recta?
4. The kidney is involved in the formation/activation of several circulating substances. Name three of these and describe their function.
5. What is renin? Where does it originate? What does it do? What is the juxtaglomerular apparatus?