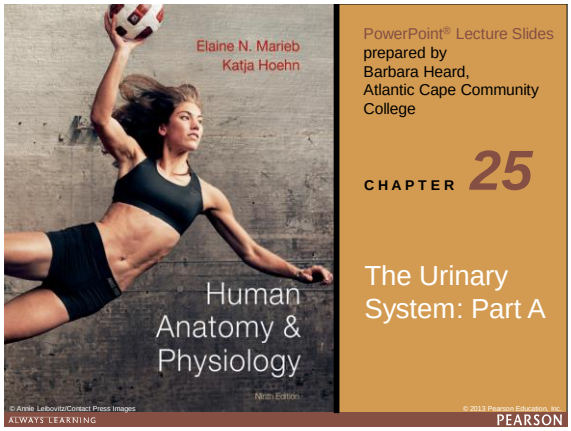

PowerPoint® Lecture Slides
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CHAPTER 25

The Urinary System: Part A

Kidney Functions

- Regulating total water volume and total solute concentration in water
- Regulating ECF ion concentrations
- Ensuring long-term acid-base balance
- Removal of metabolic wastes, toxins, drugs

Kidney Functions

- Endocrine functions
  - **Renin** - regulation of blood pressure
  - **Erythropoietin** - regulation of RBC production
- Activation of vitamin D
- Gluconeogenesis during prolonged fasting

Urinary System Organs

- **Kidneys** - major excretory organs
- **Ureters** - transport urine from kidneys to urinary bladder
- **Urinary bladder** - temporary storage reservoir for urine
- **Urethra** transports urine out of body

Figure 25.1 The urinary system.

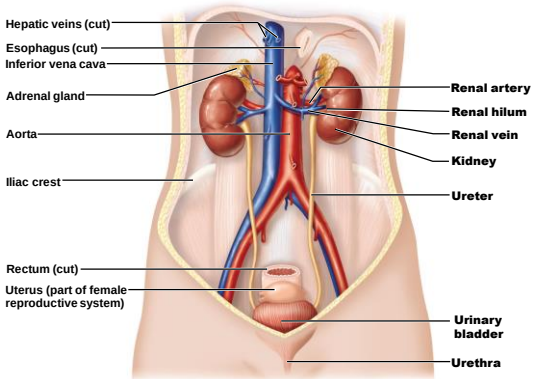
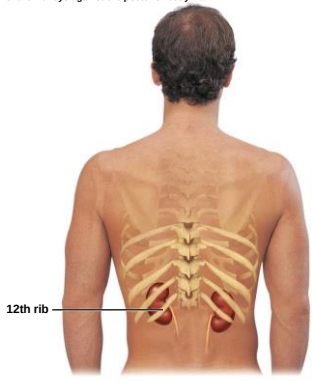


Figure 25.2b Position of the kidneys against the posterior body wall.



Internal Anatomy

- **Renal cortex**
  - Granular-appearing superficial region
- **Renal medulla**
  - Composed of cone-shaped **medullary** (**renal**) **pyramids**
  - Pyramids separated by **renal columns**
    - Inward extensions of cortical tissue

Homeostatic Imbalance

- **Pyelitis**
  - Infection of renal pelvis and calyces
- **Pyelonephritis**
  - Infection/inflammation of entire kidney
- Normally - successfully treated with antibiotics

Figure 25.2a Position of the kidneys against the posterior body wall.

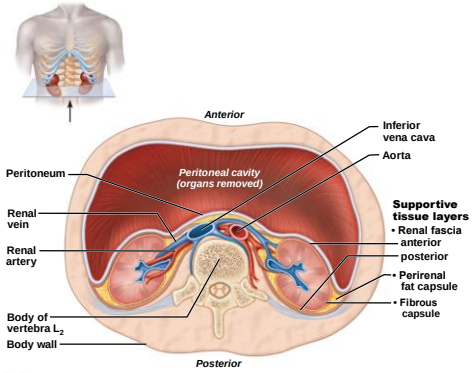
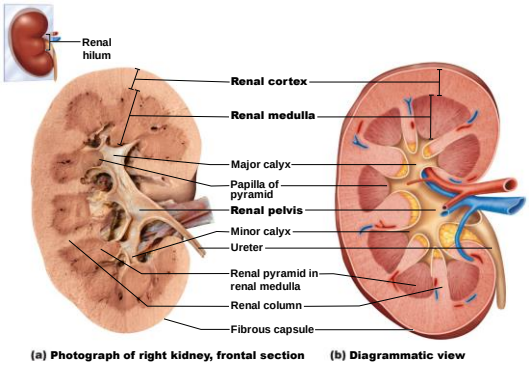


Figure 25.3 Internal anatomy of the kidney.



Blood and Nerve Supply

- Kidneys cleanse blood; adjust its composition → rich blood supply
- Renal arteries deliver ~ ¼ (1200 ml) of cardiac output to kidneys each minute
- Arterial flow into and venous flow out of kidneys follow similar paths
- Nerve supply via sympathetic fibers from **renal plexus**

Figure 25.4a Blood vessels of the kidney.

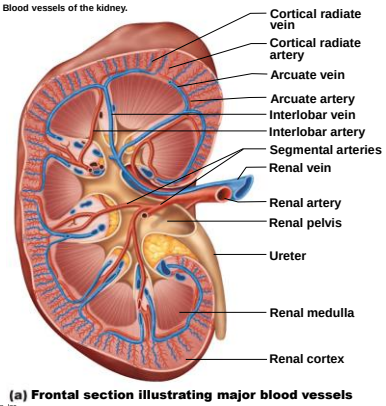
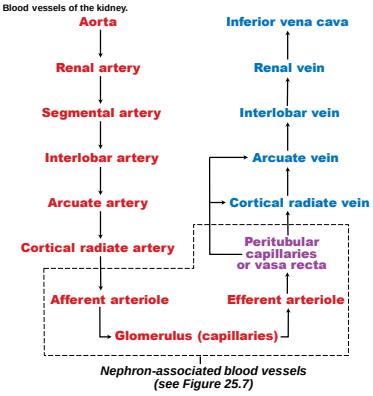


Figure 25.4b Blood vessels of the kidney.



(b) Path of blood flow through renal blood vessels

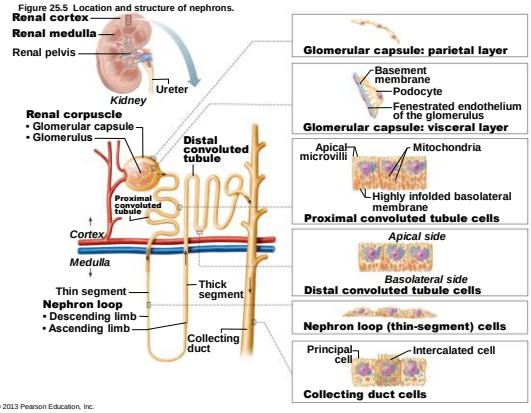
Nephrons

- Structural and functional units that form urine
- > 1 million per kidney
- Two main parts
  - **Renal corpuscle**
  - **Renal tubule**

Renal Corpuscle

- Two parts of renal corpuscle
  - **Glomerulus**
    - Tuft of capillaries; fenestrated endothelium → highly porous → allows **filtrate** formation
  - **Glomerular capsule** (**Bowman's capsule**)
    - Cup-shaped, hollow structure surrounding glomerulus

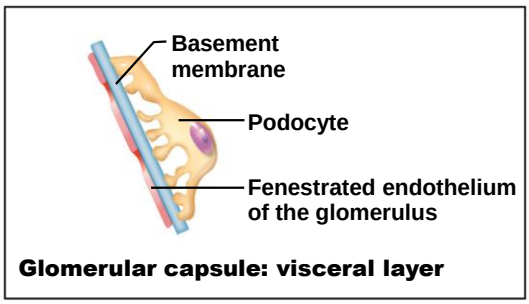
Figure 25.5 Location and structure of nephrons.



Renal Corpuscle

- Glomerular capsule
  - *Parietal layer* - simple squamous epithelium
  - *Visceral layer* - branching epithelial **podocytes**
    - Extensions terminate in **foot processes** that cling to basement membrane
    - **Filtration slits** between foot processes allow filtrate to pass into **capsular space**

Figure 25.5 Location and structure of nephrons. (3 of 7)



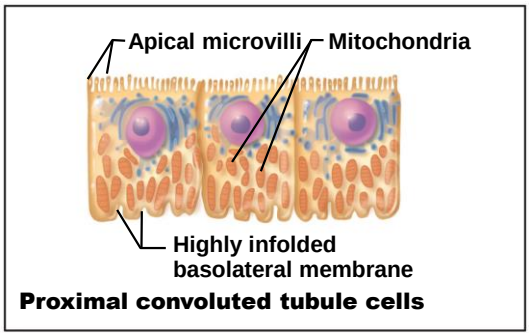
Renal Tubule

- Three parts
  - **Proximal convoluted tubule**
    - Proximal → closest to renal corpuscle
  - **Nephron loop**
  - **Distal convoluted tubule**
    - Distal → farthest from renal corpuscle

Renal Tubule

- Proximal convoluted tubule (PCT)
  - Cuboidal cells with dense microvilli (brush border → ↑surface area); large mitochondria
  - Functions in reabsorption and secretion
  - Confined to cortex

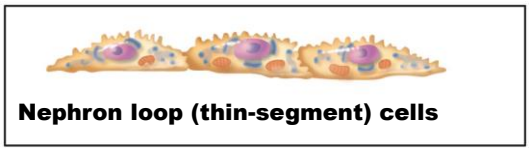
Figure 25.5 Location and structure of nephrons. (4 of 7)



Renal Tubule

- Nephron loop
  - Descending and ascending limbs
  - Proximal descending limb continuous with proximal tubule
  - Distal descending limb = *descending thin limb*; simple squamous epithelium
  - Thick ascending limb
    - Cuboidal to columnar cells; thin in some nephrons

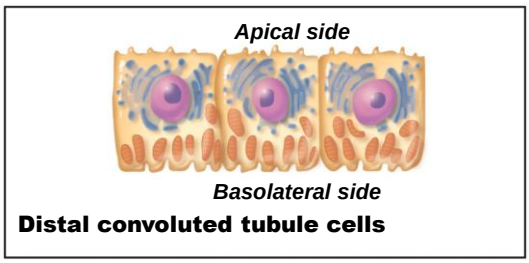
Figure 25.5 Location and structure of nephrons. (6 of 7)



Renal Tubule

- Distal convoluted tubule (DCT)
  - Cuboidal cells with very few microvilli
  - Function more in secretion than reabsorption
  - Confined to cortex

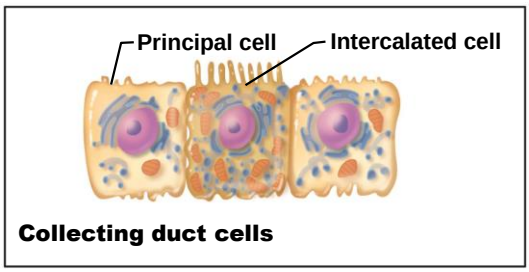
Figure 25.5 Location and structure of nephrons. (5 of 7)



Collecting Ducts

- Two cell types
  - **Principal cells**
    - Sparse, short microvilli
    - Maintain water and Na^+ balance
  - **Intercalated cells**
    - Cuboidal cells; abundant microvilli; two types
      - A and B; both help maintain acid-base balance of blood

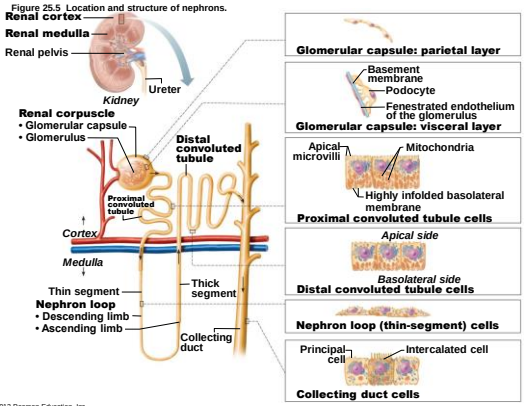
Figure 25.5 Location and structure of nephrons. (7 of 7)



Collecting Ducts

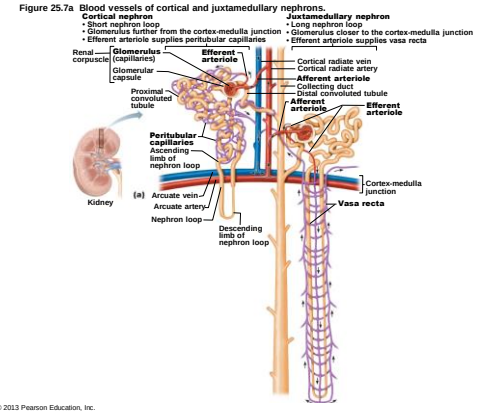
- Receive filtrate from many nephrons
- Run through medullary pyramids → striped appearance
- Fuse together to deliver urine through papillae into minor calyces

Figure 25.5 Location and structure of nephrons.



Classes of Nephrons

- **Cortical nephrons**—85% of nephrons; almost entirely in cortex
- **Juxtamedullary nephrons**
  - Long nephron loops deeply invade medulla
  - Ascending limbs have thick and thin segments
  - Important in production of concentrated urine



Nephron Capillary Beds

- Renal tubules associated with two capillary beds
  - **Glomerulus**
  - **Peritubular capillaries**
- Juxtamedullary nephrons also associated with
  - **Vasa recta**

Nephron Capillary Beds

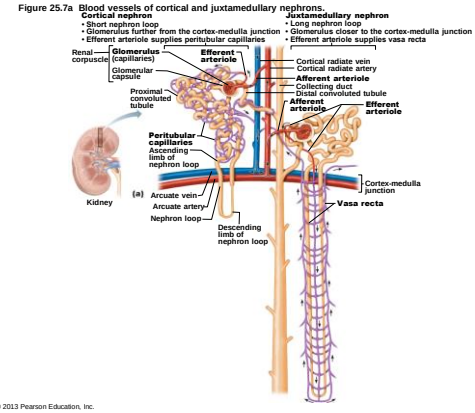
- Glomerulus - specialized for filtration
- Different from other capillary beds – fed and drained by arteriole
  - **Afferent arteriole** → glomerulus → **efferent arteriole**
- Blood pressure in glomerulus high because
  - Afferent arterioles larger in diameter than efferent arterioles
  - Arterioles are high-resistance vessels

Nephron Capillary Beds

- Peritubular capillaries
  - Low-pressure, porous capillaries adapted for absorption of water and solutes
  - Arise from efferent arterioles
  - Cling to adjacent renal tubules in cortex
  - Empty into venules

Nephron Capillary Beds

- Vasa recta
  - Long, thin-walled vessels parallel to long nephron loops of juxtamedullary nephrons
  - Arise from efferent arterioles serving juxtamedullary nephrons
    - Instead of peritubular capillaries
  - Function in formation of concentrated urine



Juxtaglomerular Complex (JGC)

- One per nephron
- Involves modified portions of
  - Distal portion of ascending limb of nephron loop
  - Afferent (sometimes efferent) arteriole
- Important in regulation of rate of filtrate formation and blood pressure

Juxtaglomerular Complex (JGC)

- Three cell populations
  - Macula densa, granular cells, extraglomerular mesangial cells
- **Macula densa**
  - Tall, closely packed cells of ascending limb
  - Chemoreceptors; sense NaCl content of filtrate

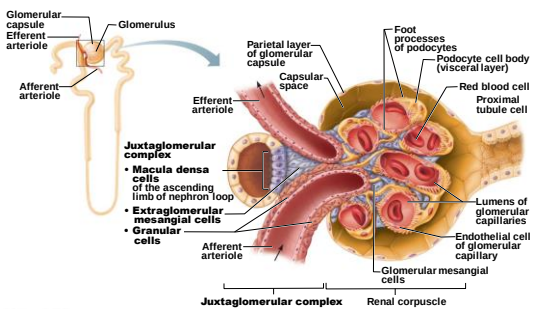
Juxtaglomerular Complex (JGC)

- **Granular cells** (**juxtaglomerular**, or **JG** cells)
  - Enlarged, smooth muscle cells of arteriole
  - Secretory granules contain enzyme **renin**
  - Mechanoreceptors; sense blood pressure in afferent arteriole

Juxtaglomerular Complex (JGC)

- Extraglomerular mesangial cells
  - Between arteriole and tubule cells
  - Interconnected with gap junctions
  - May pass signals between macula densa and granular cells

Figure 25.8 Juxtaglomerular complex (JGC) of a nephron.



Kidney Physiology: Mechanisms of Urine Formation

- 180 L fluid processed daily; only 1.5 L → urine
- Three processes in urine formation and adjustment of blood composition
  - **Glomerular filtration**
  - **Tubular reabsorption**
  - **Tubular secretion**

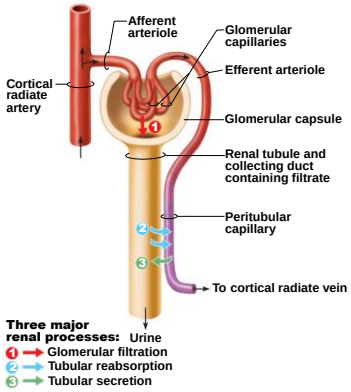
Kidney Physiology: Mechanisms of Urine Formation

- **Glomerular filtration** – produces cell- and protein-free filtrate
- **Tubular reabsorption**
  - Selectively returns 99% of substances from filtrate to blood in renal tubules and collecting ducts
- **Tubular secretion**
  - Selectively moves substances from blood to filtrate in renal tubules and collecting ducts

Kidney Physiology: Mechanisms of Urine Formation

- Kidneys filter body's entire plasma volume 60 times each day; consume 20-25% oxygen used by body at rest; produce urine from filtrate
- Filtrate (produced by glomerular filtration)
  - Blood plasma minus proteins
- Urine
  - <1% of original filtrate
  - Contains metabolic wastes and unneeded substances

Figure 25.9 A schematic, uncoiled nephron showing the three major renal processes that adjust plasma composition.



Glomerular Filtration

- Passive process
- No metabolic energy required
- Hydrostatic pressure forces fluids and solutes through filtration membrane
- No reabsorption into capillaries of glomerulus

The Filtration Membrane

- Porous membrane between blood and interior of glomerular capsule
  - Water, solutes smaller than plasma proteins pass; normally no cells pass
- Three layers
  - **Fenestrated endothelium** of glomerular capillaries
  - **Basement membrane** (fused basal laminae of two other layers)
  - **Foot processes of podocytes** with filtration slits; slit diaphragms repel macromolecules

Figure 25.10a The filtration membrane.

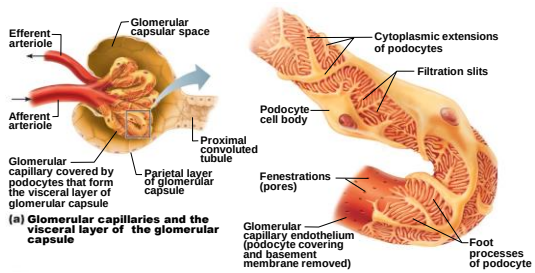
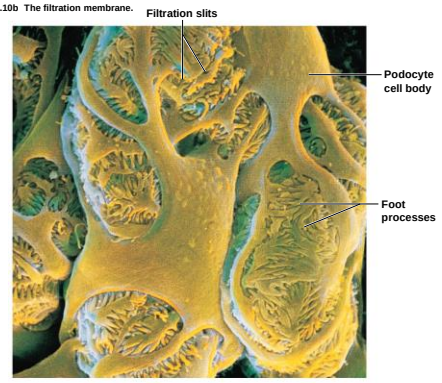
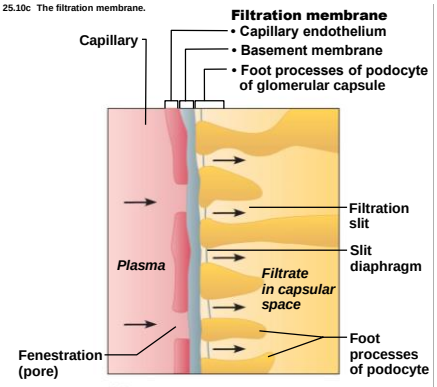


Figure 25.10b The filtration membrane.



(b) Filtration slits between the podocyte foot processes

Figure 25.10c The filtration membrane.



(c) Three layers of the filtration membrane

The Filtration Membrane

- Macromolecules "stuck" in filtration membrane engulfed by glomerular mesangial cells
- Allows molecules smaller than 3 nm to pass
  - Water, glucose, amino acids, nitrogenous wastes
- Plasma proteins remain in blood → maintains colloid osmotic pressure → prevents loss of all water to capsular space
  - Proteins in filtrate indicate membrane problem

Pressures That Affect Filtration

- Outward pressures promote filtrate formation
  - **Hydrostatic pressure in glomerular capillaries** = Glomerular blood pressure
    - Chief force pushing water, solutes out of blood
    - Quite high – 55 mm Hg (most capillary beds ~ 26 mm Hg)
      - Because efferent arteriole is high resistance vessel with diameter smaller than afferent arteriole

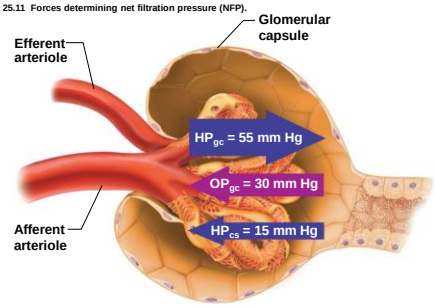
Pressures That Affect Filtration

- Inward forces inhibiting filtrate formation
  - Hydrostatic pressure in capsular space (HP_{cs})
    - Pressure of filtrate in capsule – 15 mm Hg
  - Colloid osmotic pressure in capillaries (OP_{gc})
    - "Pull" of proteins in blood – 30 mm Hg
- Sum of forces → **Net filtration pressure (NFP)**
  - 55 mm Hg forcing out; 45 mm Hg opposing = net outward force of 10 mm Hg

Net Filtration Pressure (NFP)

- Pressure responsible for filtrate formation (10 mm Hg)
- Main controllable factor determining **glomerular filtration rate (GFR)**

Figure 25.11 Forces determining net filtration pressure (NFP).



NFP = Net filtration pressure
= outward pressures – inward pressures
= $(HP_{gc}) – (HP_{cs} + OP_{gc})$
= (55) – (15 + 30)
= 10 mm Hg

Glomerular Filtration Rate (GFR)

- Volume of filtrate formed per minute by both kidneys (normal = 120–125 ml/min)
- GFR directly proportional to
  - **NFP** – primary pressure is hydrostatic pressure in glomerulus
  - **Total surface area available for filtration** – glomerular mesangial cells control by contracting
  - **Filtration membrane permeability** – much more permeable than other capillaries

Regulation of Glomerular Filtration

- Constant GFR allows kidneys to make filtrate and maintain extracellular homeostasis
  - Goal of *intrinsic controls* - maintain GFR in kidney
- GFR affects systemic blood pressure
  - ↑ GFR → ↑urine output → ↓ blood pressure, and vice versa
  - Goal of *extrinsic controls* - maintain systemic blood pressure

Regulation of Glomerular Filtration

- Intrinsic controls (renal autoregulation)
  - Act locally within kidney to maintain GFR
- Extrinsic controls
  - Nervous and endocrine mechanisms that maintain blood pressure; can negatively affect kidney function
  - Take precedence over intrinsic controls if systemic BP < 80 or > 180 mm Hg

Regulation of Glomerular Filtration

- Controlled via glomerular hydrostatic pressure
  - If rises → NFP rises → GFR rises
  - If falls only 18% GFR = 0

Intrinsic Controls

- Maintains nearly constant GFR when MAP in range of 80–180 mm Hg
  - Autoregulation ceases if out of that range
- Two types of renal autoregulation
  - **Myogenic mechanism**
  - **Tubuloglomerular feedback mechanism**

Intrinsic Controls: Myogenic Mechanism

- Smooth muscle contracts when stretched
- ↑ BP → muscle stretch → constriction of afferent arterioles → restricts blood flow into glomerulus
  - Protects glomeruli from damaging high BP
- ↓ BP → dilation of afferent arterioles
- Both help maintain normal GFR despite normal fluctuations in blood pressure

Intrinsic Controls: Tubuloglomerular Feedback Mechanism

- Flow-dependent mechanism directed by macula densa cells; respond to filtrate NaCl concentration
- If GFR ↑→ filtrate flow rate ↑→ ↓ reabsorption time → high filtrate NaCl levels → constriction of afferent arteriole → ↓ NFP & GFR → more time for NaCl reabsorption
- Opposite for ↓ GFR

Extrinsic Controls: Sympathetic Nervous System

- Under normal conditions at rest
  - Renal blood vessels dilated
  - Renal autoregulation mechanisms prevail

Extrinsic Controls: Sympathetic Nervous System

- If extracellular fluid volume extremely low (blood pressure low)
  - Norepinephrine released by sympathetic nervous system; epinephrine released by adrenal medulla →
    - Systemic vasoconstriction → increased blood pressure
    - Constriction of afferent arterioles → ↓ GFR → increased blood volume and pressure

Extrinsic Controls: Renin-Angiotensin-Aldosterone Mechanism

- Main mechanism for increasing blood pressure – see Chapters 16 and 19
- Three pathways to renin release by granular cells
  - Direct stimulation of granular cells by sympathetic nervous system
  - Stimulation by activated macula densa cells when filtrate NaCl concentration low
  - Reduced stretch of granular cells

Extrinsic Controls: Other Factors Affecting GFR

- Kidneys release chemicals; some act as paracrines that affect renal arterioles
  - Adenosine
  - Prostaglandin E_2
  - Intrinsic angiotensin II – reinforces effects of hormonal angiotensin II

Tubular Reabsorption

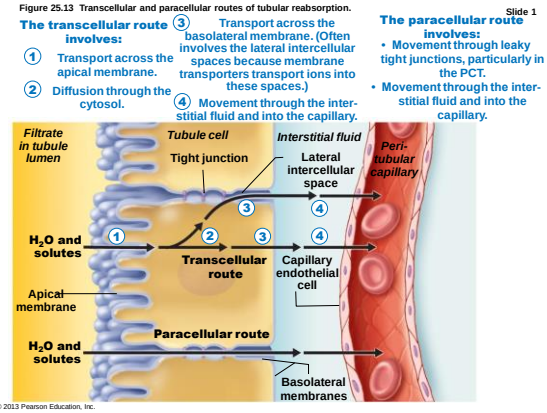
- Most of tubular contents reabsorbed to blood
- Selective transepithelial process
  - ~ All organic nutrients reabsorbed
  - Water and ion reabsorption hormonally regulated and adjusted
- Includes **active** and **passive tubular reabsorption**
- Two routes
  - Transcellular or paracellular

Tubular Reabsorption

- **Paracellular route**
  - Between tubule cells
    - Limited by tight junctions, but leaky in proximal nephron
      - Water, Ca^{2+}, Mg^{2+}, K^+, and some Na^+ in the PCT

Figure 25.13 Transcellular and paracellular routes of tubular reabsorption.

Slide 1



Tubular Reabsorption of Sodium

- Na^+ - most abundant cation in filtrate
  - Transport across basolateral membrane
    - Primary active transport out of tubule cell by Na^+-K^+ ATPase pump → peritubular capillaries
  - Transport across apical membrane
    - Na^+ passes through apical membrane by secondary active transport or facilitated diffusion mechanisms

Reabsorption of Nutrients, Water, and Ions

- Na^+ reabsorption by primary active transport provides energy and means for reabsorbing most other substances
- Creates electrical gradient → passive reabsorption of anions
- Organic nutrients reabsorbed by secondary active transport; cotransported with Na^+
  - Glucose, amino acids, some ions, vitamins

Passive Tubular Reabsorption of Water

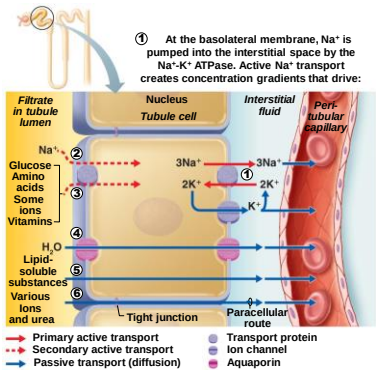
- Movement of Na^+ and other solutes creates osmotic gradient for water
- Water reabsorbed by osmosis, aided by water-filled pores called **aquaporins**
  - Aquaporins always present in PCT → **obligatory water reabsorption**
  - Aquaporins inserted in collecting ducts only if ADH present → **facultative water reabsorption**

Passive Tubular Reabsorption of Solutes

- Solute concentration in filtrate increases as water reabsorbed → concentration gradients for solutes →
- Fat-soluble substances, some ions and urea, follow water into peritubular capillaries down concentration gradients
  - → Lipid-soluble drugs, environmental pollutants difficult to excrete

Figure 25.14 Reabsorption by PCT cells.

Slide 1



② "Downhill" Na^+ entry at the apical membrane.

③ Reabsorption of organic nutrients and certain ions by cotransport at the apical membrane.

④ Reabsorption of water by osmosis through aquaporins. Water reabsorption increases the concentration of the solutes that are left behind. These solutes can then be reabsorbed as they move down their gradients:

⑤ Lipid-soluble substances diffuse by the transcellular route.

⑥ Various ions (e.g., Cl^-, Ca^{2+}, K^+) and urea diffuse by the paracellular route.

Transport Maximum

- Transcellular transport systems specific and limited
  - Transport maximum (T_m) for ~ every reabsorbed substance; reflects number of carriers in renal tubules available
  - When carriers saturated, excess excreted in urine
    - E.g., hyperglycemia → high blood glucose levels exceed T_m → glucose in urine

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- PCT
  - Site of most reabsorption
    - All nutrients, e.g., glucose and amino acids
    - 65% of Na^+ and water
    - Many ions
    - ~ All uric acid; ½ urea (later secreted back into filtrate)

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- Nephron loop
  - Descending limb - H_2O can leave; solutes cannot
  - Ascending limb – H_2O cannot leave; solutes can
    - Thin segment – passive Na^+ movement
    - Thick segment – Na^+-K^+-$2Cl^-$ symporter and Na^+-H^+ antiporter; some passes by paracellular route

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- DCT and collecting duct
  - Reabsorption hormonally regulated
    - **Antidiuretic hormone** (**ADH**) – Water
    - **Aldosterone** – Na^+ (therefore water)
    - **Atrial natriuretic peptide** (**ANP**) – Na^+
    - **PTH** – Ca^{2+}

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- Antidiuretic hormone (ADH)
  - Released by posterior pituitary gland
  - Causes principal cells of collecting ducts to insert aquaporins in apical membranes → water reabsorption
    - As ADH levels increase → increased water reabsorption

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- Aldosterone
  - Targets collecting ducts (principal cells) and distal DCT
  - Promotes synthesis of apical Na^+ and K^+ channels, and basolateral Na^+-K^+ ATPases for Na^+ reabsorption; water follows
  - → little Na^+ leaves body; aldosterone absence → loss of 2% filtered Na^+ daily - incompatible with life
  - Functions – increase blood pressure; decrease K^+ levels

Reabsorptive Capabilities of Renal Tubules and Collecting Ducts

- Atrial natriuretic peptide
  - Reduces blood Na^+ → decreased blood volume and blood pressure
  - Released by cardiac atrial cells if blood volume or pressure elevated
- Parathyroid hormone acts on DCT to increase Ca^{2+} reabsorption

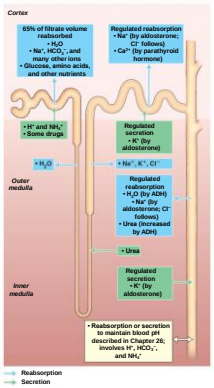
Tubular Secretion

- Reabsorption in reverse; almost all in PCT
  - Selected substances
  - K^+, H^+, NH_4^+, creatinine, organic acids and bases move from peritubular capillaries through tubule cells into filtrate
  - Substances synthesized in tubule cells also secreted – e.g., HCO_3^-

Tubular Secretion

- Disposes of substances (e.g., drugs) bound to plasma proteins
- Eliminates undesirable substances passively reabsorbed (e.g., urea and uric acid)
- Rids body of excess K^+ (aldosterone effect)
- Controls blood pH by altering amounts of H^+ or HCO_3^- in urine

Figure 25.15 Summary of tubular reabsorption and secretion.



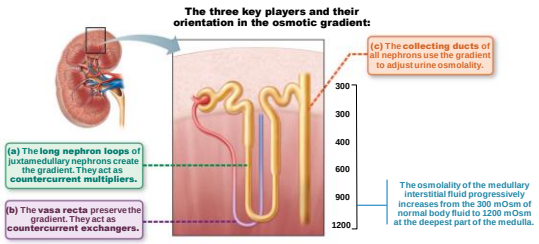
Regulation of Urine Concentration and Volume

- Osmolality of body fluids
  - Expressed in milliosmols (mOsm)
  - Kidneys maintain osmolality of plasma at ~300 mOsm by regulating urine concentration and volume
  - Kidneys regulate with **countercurrent mechanism**

Countercurrent Mechanism

- Occurs when fluid flows in opposite directions in two adjacent segments of same tube with hair pin turn
  - **Countercurrent multiplier** – interaction of filtrate flow in ascending/descending limbs of nephron loops of juxtamedullary nephrons
  - **Countercurrent exchanger** - Blood flow in ascending/descending limbs of vasa recta

Figure 25.16a Juxtamedullary nephrons create an osmotic gradient within the renal medulla that allows the kidney to produce urine of varying concentration. (1 of 4)



The Countercurrent Multiplier

- Constant 200 mOsm difference between two limbs of nephron loop and between ascending limb and interstitial fluid
- Difference "multiplied" along length of loop to ~ 900 mOsm

The Countercurrent Exchanger

- Vasa recta
- Preserve medullary gradient
  - Prevent rapid removal of salt from interstitial space
  - Remove reabsorbed water
- Water entering ascending vasa recta either from descending vasa recta or reabsorbed from nephron loop and collecting duct →
  - Volume of blood at end of vasa recta greater than at beginning

Figure 25.16a Juxtamedullary nephrons create an osmotic gradient within the renal medulla that allows the kidney to produce urine of varying concentration. (4 of 4)

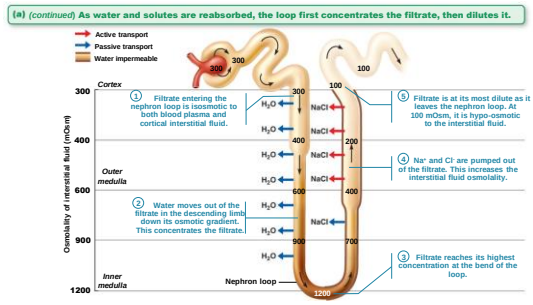


Figure 25.16b Juxtamedullary nephrons create an osmotic gradient within the renal medulla that allows the kidney to produce urine of varying concentration.

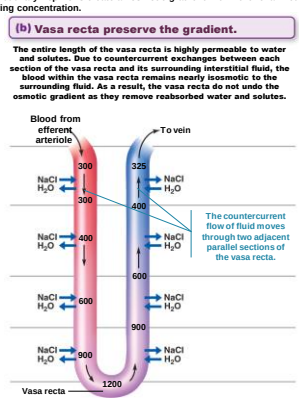
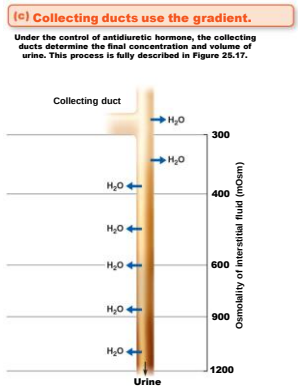


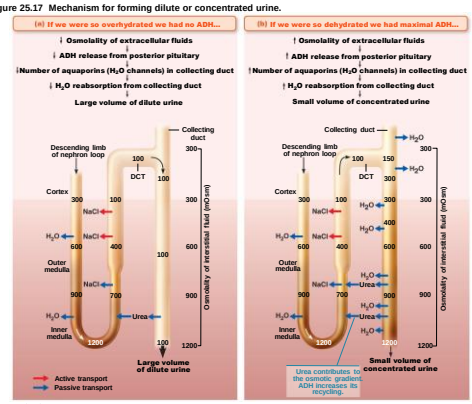
Figure 25.16c Juxtamedullary nephrons create an osmotic gradient within the renal medulla that allows the kidney to produce urine of varying concentration.



Formation of Dilute or Concentrated Urine

- Osmotic gradient used to raise urine concentration > 300 mOsm to conserve water
  - Overhydration → large volume dilute urine
    - ADH production ↓; urine ~ 100 mOsm
    - If aldosterone present, additional ions removed → ~ 50 mOsm
  - Dehydration → small volume concentrated urine
    - Maximal ADH released; urine ~ 1200 mOsm
    - Severe dehydration – 99% water reabsorbed

Figure 25.17 Mechanism for forming dilute or concentrated urine.



Urea Recycling and the Medullary Osmotic Gradient

- Urea helps form medullary gradient
  - Enters filtrate in ascending thin limb of nephron loop by facilitated diffusion
  - Cortical collecting duct reabsorbs water; leaves urea
  - In deep medullary region now highly concentrated urea → interstitial fluid of medulla → back to ascending thin limb → high osmolality in medulla

Diuretics

- Chemicals that enhance urinary output
  - ADH inhibitors, e.g., alcohol
  - Na^+ reabsorption inhibitors (and resultant H_2O reabsorption), e.g., caffeine, drugs for hypertension or edema
  - Loop diuretics inhibit medullary gradient formation
  - *Osmotic diuretics* - substance not reabsorbed so water remains in urine, e.g., high glucose of diabetic patient

Renal Clearance

- Volume of plasma kidneys clear of particular substance in given time
- Renal clearance tests used to determine GFR
  - To detect glomerular damage
  - To follow progress of renal disease

Renal Clearance

- C = UV/P
  - C = renal clearance rate (ml/min)
  - U = concentration (mg/ml) of substance in urine
  - V = flow rate of urine formation (ml/min)
  - P = concentration of same substance in plasma

Renal Clearance

- Inulin (plant polysaccharide) is standard used
  - Freely filtered; neither reabsorbed nor secreted by kidneys; its renal clearance = GFR = 125 ml/min
- If C < 125 ml/min, substance reabsorbed
- If C = 0, substance completely reabsorbed, or not filtered
- If C = 125 ml/min, no net reabsorption or secretion
- If C > 125 ml/min, substance secreted (most drug metabolites)

Homeostatic Imbalance

- **Chronic renal disease** - GFR < 60 ml/min for 3 months
  - E.g., in diabetes mellitus; hypertension
- **Renal failure** – GFR < 15 ml/min
  - Causes **uremia** – ionic and hormonal imbalances; metabolic abnormalities; toxic molecule accumulation
  - Treated with **hemodialysis** or transplant

Physical Characteristics of Urine

- Color and transparency
  - Clear
    - Cloudy may indicate urinary tract infection
  - Pale to deep yellow from **urochrome**
    - Pigment from hemoglobin breakdown; more concentrated urine → deeper color
  - Abnormal color (pink, brown, smoky)
    - Food ingestion, bile pigments, blood, drugs

Physical Characteristics of Urine

- Odor
  - Slightly aromatic when fresh
  - Develops ammonia odor upon standing
    - As bacteria metabolize solutes
  - May be altered by some drugs and vegetables

Physical Characteristics of Urine

- pH
  - Slightly acidic (~pH 6, with range of 4.5 to 8.0)
    - Acidic diet (protein, whole wheat) → ↓ pH
    - Alkaline diet (vegetarian), prolonged vomiting, or urinary tract infections → ↑pH
- Specific gravity
  - 1.001 to 1.035; dependent on solute concentration

Chemical Composition of Urine

- 95% water and 5% solutes
- **Nitrogenous wastes**
  - **Urea** (from amino acid breakdown) – largest solute component
  - **Uric acid** (from nucleic acid metabolism)
  - **Creatinine** (metabolite of creatine phosphate)

Chemical Composition of Urine

- Other normal solutes
  - Na^+, K^+, PO_4^{3-}, and SO_4^{2-}, Ca^{2+}, Mg^{2+} and HCO_3^-
- Abnormally high concentrations of any constituent, or abnormal components, e.g., blood proteins, WBCs, bile pigments, may indicate pathology

Urine transport, Storage, and Elimination: Ureters

- Convey urine from kidneys to bladder
  - Begin at L_2 as continuation of renal pelvis
- Retroperitoneal
- Enter base of bladder through posterior wall
  - As bladder pressure increases, distal ends of ureters close, preventing backflow of urine

Homeostatic Imbalance

- **Renal calculi** - kidney stones in renal pelvis
  - Crystallized calcium, magnesium, or uric acid salts
- Large stones block ureter → pressure & pain
- May be due to chronic bacterial infection, urine retention, ↑Ca^{2+} in blood, ↑pH of urine
- Treatment - *shock wave lithotripsy* – noninvasive; shock waves shatter calculi

Urinary Bladder

- Muscular sac for temporary storage of urine
- Retroperitoneal, on pelvic floor posterior to pubic symphysis
  - Males—prostate inferior to bladder neck
  - Females—anterior to vagina and uterus

Urinary Bladder

- Openings for ureters and urethra
- **Trigone**
  - Smooth triangular area outlined by openings for ureters and urethra
  - Infections tend to persist in this region

Urinary Bladder

- Layers of bladder wall
  - Mucosa - transitional epithelial mucosa
  - Thick **detrusor** - three layers of smooth muscle
  - Fibrous adventitia (peritoneum on superior surface only)

Urinary Bladder

- Collapses when empty; *rugae* appear
- Expands and rises superiorly during filling without significant rise in internal pressure
- ~ Full bladder 12 cm long; holds ~ 500 ml
  - Can hold ~ twice that if necessary
  - Can burst if overdistended

Figure 25.18 Pyelogram.

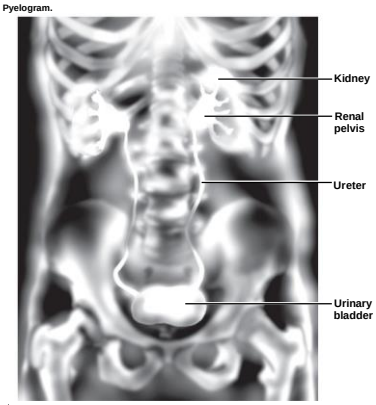
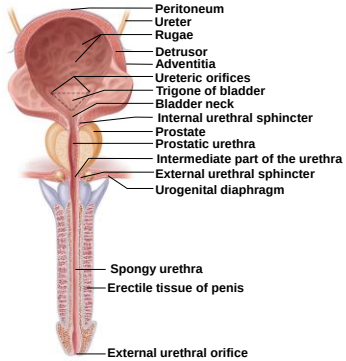
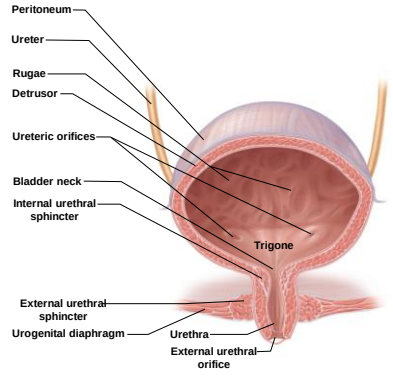


Figure 25.20a Structure of the urinary bladder and urethra.



(a) Male. The long male urethra has three regions: prostatic, intermediate, and spongy.

Figure 25.20b Structure of the urinary bladder and urethra.



(b) Female.

Urethra

- Sphincters
  - **Internal urethral sphincter**
    - Involuntary (smooth muscle) at bladder-urethra junction
    - Contracts to open
  - **External urethral sphincter**
    - Voluntary (skeletal) muscle surrounding urethra as it passes through pelvic floor

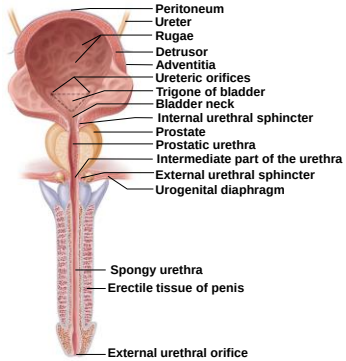
Urethra

- Female urethra (3–4 cm)
  - Tightly bound to anterior vaginal wall
  - **External urethral orifice**
    - Anterior to vaginal opening; posterior to clitoris

Urethra

- Male urethra carries semen and urine
  - Three named regions
    - **Prostatic urethra** (2.5 cm)—within prostate
    - **Intermediate part of the urethra** (*membranous urethra*) (2 cm)—passes through urogenital diaphragm from prostate to beginning of penis
    - **Spongy urethra** (15 cm)—passes through penis; opens via **external urethral orifice**

Figure 25.20a Structure of the urinary bladder and urethra.



(a) Male. The long male urethra has three regions: prostatic, intermediate, and spongy.

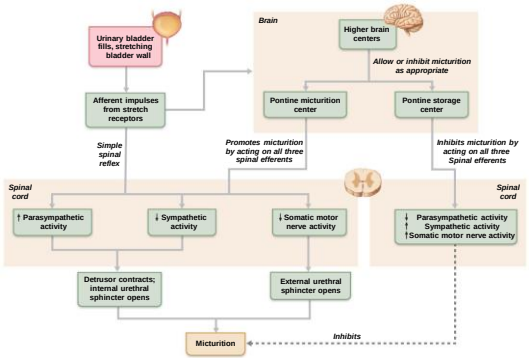
Micturition

- **Urination** or voiding
- Three simultaneous events must occur
  - Contraction of detrusor by ANS
  - Opening of internal urethral sphincter by ANS
  - Opening of external urethral sphincter by somatic nervous system

Micturition

- Reflexive urination (urination in infants)
  - Distension of bladder activates stretch receptors
  - Excitation of parasympathetic neurons in reflex center in sacral region of spinal cord
  - Contraction of detrusor
  - Contraction (opening) of internal sphincter
  - Inhibition of somatic pathways to external sphincter, allowing its relaxation (opening)

Figure 25.21 Control of micturition.



Homeostatic Imbalance

- **Incontinence** usually from weakened pelvic muscles
  - *Stress incontinence*
    - Increased intra-abdominal pressure forces urine through external sphincter
  - *Overflow incontinence*
    - Urine dribbles when bladder overfills

Homeostatic Imbalance

- Urinary retention
  - Bladder unable to expel urine
  - Common after general anesthesia
  - Hypertrophy of prostate
  - Treatment - catheterization

Developmental Aspects

- Three sets of embryonic kidneys form in succession
  - **Pronephros** degenerates but **pronephric duct** persists
  - **Mesonephros** claims this duct; becomes **mesonephric duct**
  - **Metanephros** develop by fifth week, develops into adult kidneys and ascends

Figure 25.22a Development of the urinary system in the embryo.

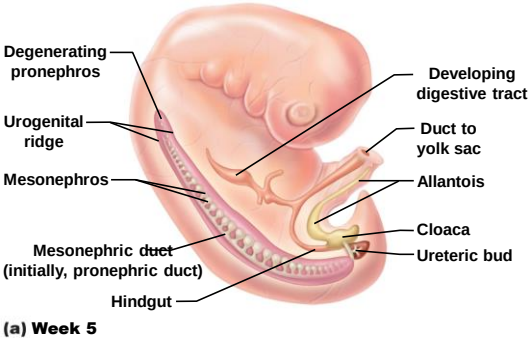
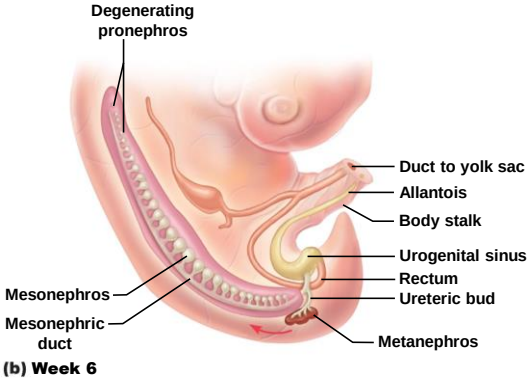


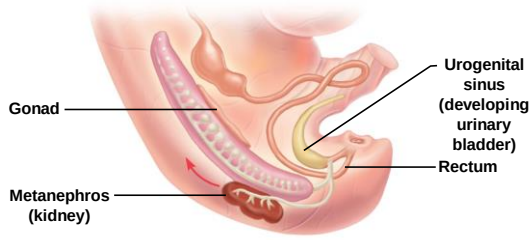
Figure 25.22b Development of the urinary system in the embryo.



Developmental Aspects

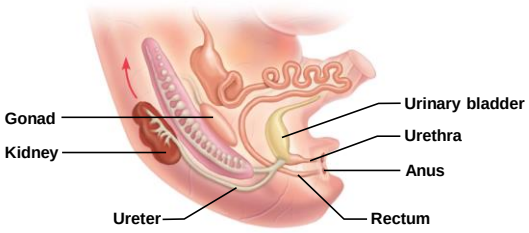
- Metanephros develops as ureteric buds that induce mesoderm of urogenital ridge to form nephrons
  - Distal ends of ureteric buds form renal pelves, calyces, and collecting ducts
  - Proximal ends become ureters
- Kidneys excrete urine into amniotic fluid by third month
- Cloaca subdivides into rectum, anal canal, and **urogenital sinus**

Figure 25.22c Development of the urinary system in the embryo.



(c) Week 7

Figure 25.22d Development of the urinary system in the embryo.



(d) Week 8

Homeostatic Imbalance

- Three common congenital abnormalities
- *Horseshoe kidney*
  - Two kidneys fuse across midline → single U-shaped kidney; usually asymptomatic
- *Hypospadias*
  - Urethral orifice on ventral surface of penis
  - Corrected surgically at ~ 12 months

Homeostatic Imbalance

- *Polycystic kidney disease*
  - Many fluid-filled cysts interfere with function
    - Autosomal dominant form – less severe but more common
    - Autosomal recessive – more severe
  - Cause unknown but involves defect in signaling proteins

Developmental Aspects

- Frequent micturition in infants due to small bladders and less-concentrated urine
- Incontinence normal in infants: control of voluntary urethral sphincter develops with nervous system
- *E. coli* bacteria account for 80% of all urinary tract infections
- Untreated childhood streptococcal infections may cause long-term renal damage
- Sexually transmitted diseases can also inflame urinary tract

Developmental Aspects

- Most elderly people have abnormal kidneys histologically
  - Kidneys shrink; nephrons decrease in size and number; tubule cells less efficient
  - GFR ½ that of young adult by age 80
    - Possibly from atherosclerosis of renal arteries
- Bladder shrinks; loss of bladder tone → *nocturia* and *incontinence*