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Know the flow

Did you know that 26 million—about 1 in 9—Americans have kidney disease? We discuss the continuum of kidney failure from the healthy kidney to complete renal support via dialysis or transplant.

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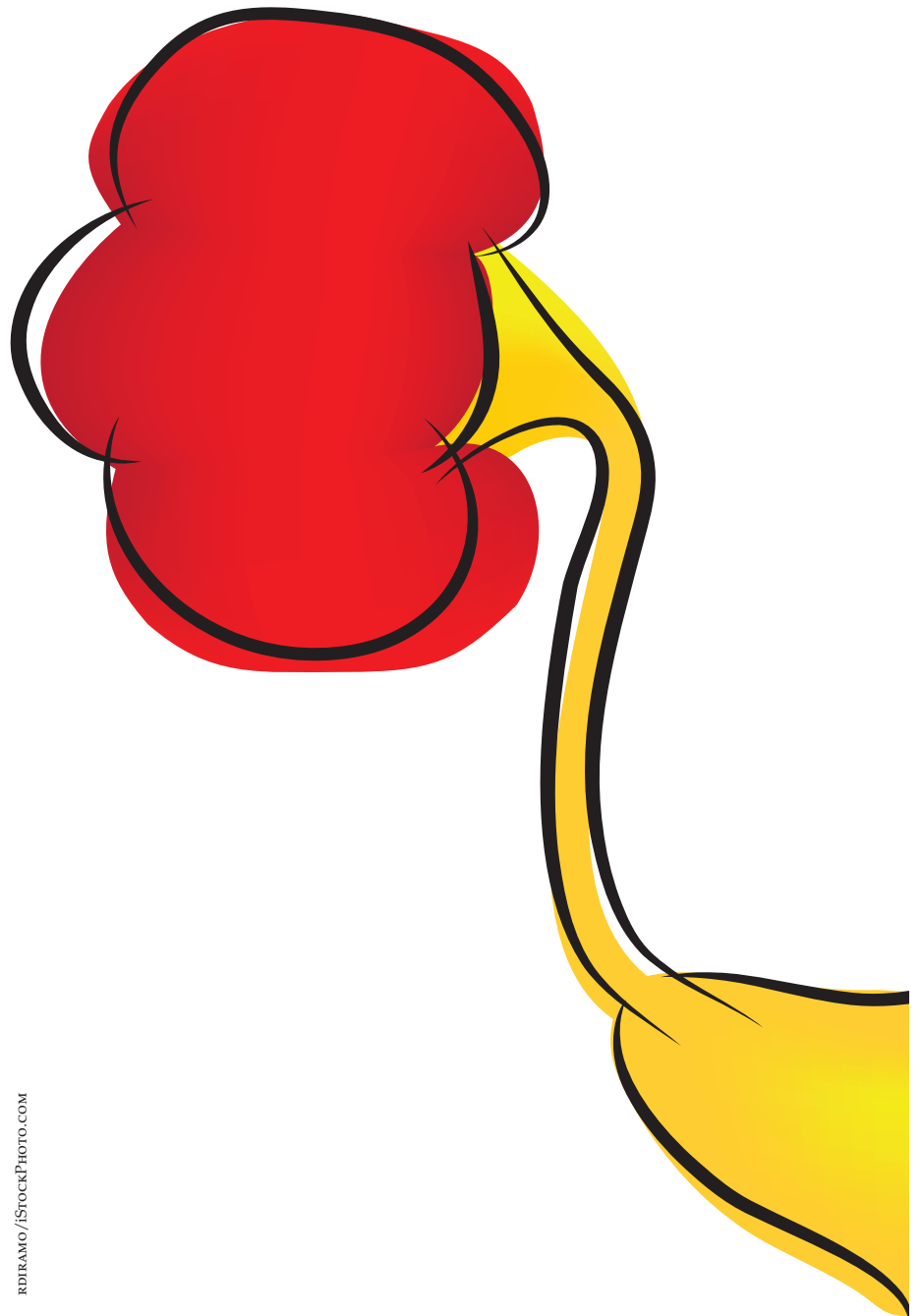
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What happens when the kidneys aren't functioning properly? What are the symptoms and treatment of kidney dysfunction? In this article, we'll explore these questions as we discuss normal kidney function, acute kidney injury (AKI), chronic kidney disease (CKD), dialysis, and kidney transplantation.

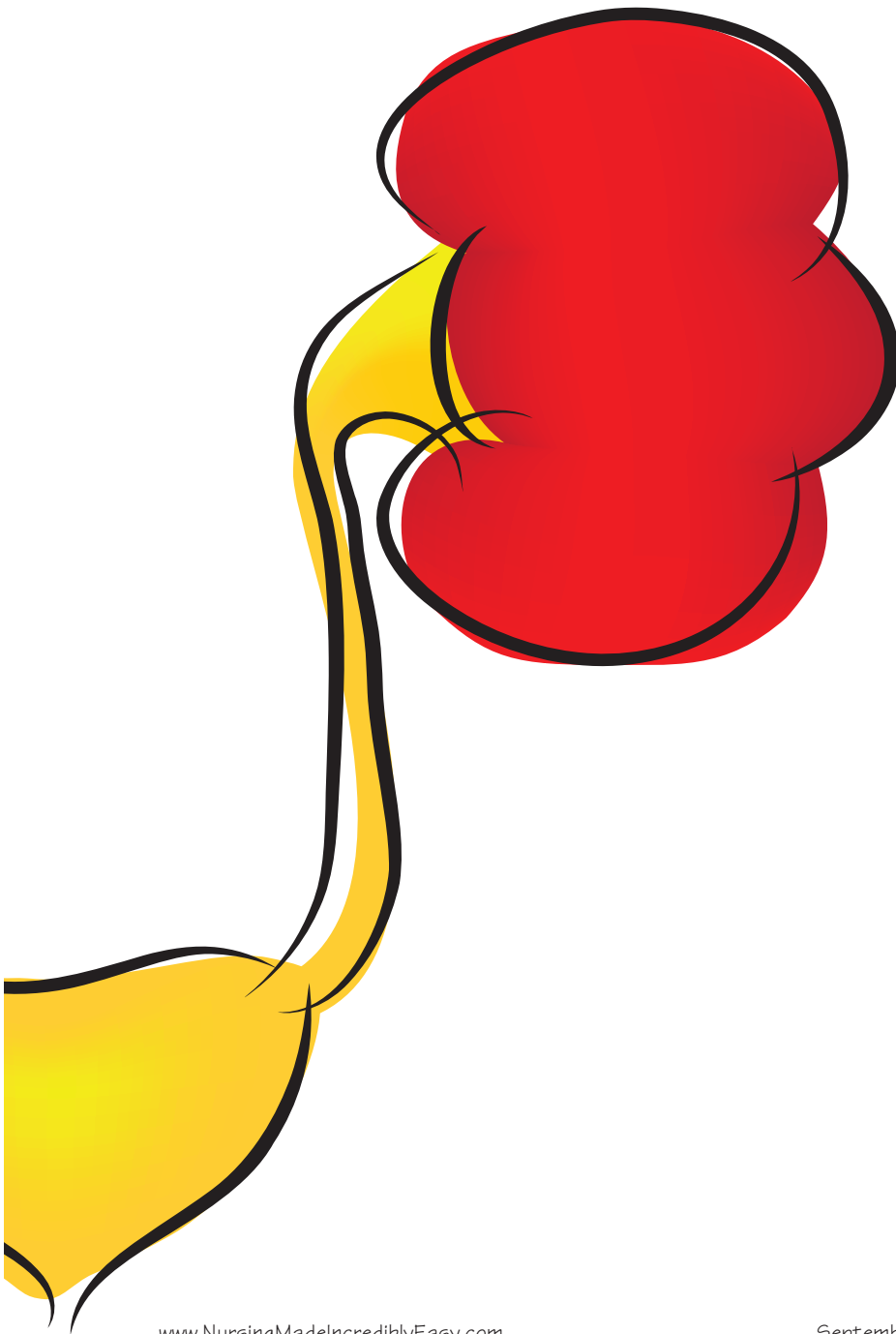
The renal system at a glance

The renal (or urinary) system comprises the kidneys, ureters, bladder, and urethra (see *Urinary system components*). The primary



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Kidney disease



function of the kidneys is regulating the volume and composition of extracellular fluid. Additionally, the kidneys assist in the control of BP, erythropoietin production, vitamin D activation, and acid-base regulation. The kidneys secrete dietary and waste products that aren't eliminated by other organs through the formation of urine. Urine is formed within the kidneys, drains to the ureters, then to the bladder, and out of the body through the urethra (see *Inside scoop: Kidney regions*).

Nephrons are the functional units of the kidneys, and each kidney has over 1 million nephrons (see *Inside scoop: The nephron*). Each nephron has two major portions: the renal corpuscle and the renal tubules. The renal corpuscle consists of the glomerulus and Bowman's capsule where the production of filtrate takes place. The renal tubules consist of the proximal convoluted tubule, nephron loop, and distal convoluted tubule. The proximal convoluted tubule is responsible for the reabsorption of 80% of electrolytes and water, all glucose and amino acid reabsorption, and the secretion of creatinine. The nephron loop is divided into the descending limb and the ascending limb. The reabsorption of water takes place in the descending limb; the reabsorption of sodium and chloride takes place in the ascending limb. The distal convoluted tubule is the site of active secretion of potassium, hydrogen, and ammonia and the reabsorption of sodium, calcium ions, and water.

The **renin-angiotensin-aldosterone system** (RAAS) is a regulatory mechanism that helps the body control vascular fluid volume, BP, and serum sodium concentrations. When the kidneys receive a signal of low BP, low renal blood flow, or low serum sodium concentra-

tions, the RAAS is activated. These signals are sensed by the juxtaglomerular apparatus, which then secretes renin. When renin encounters angiotensinogen, it acts as an enzyme that promotes the conversion of angiotensinogen into angiotensin I.

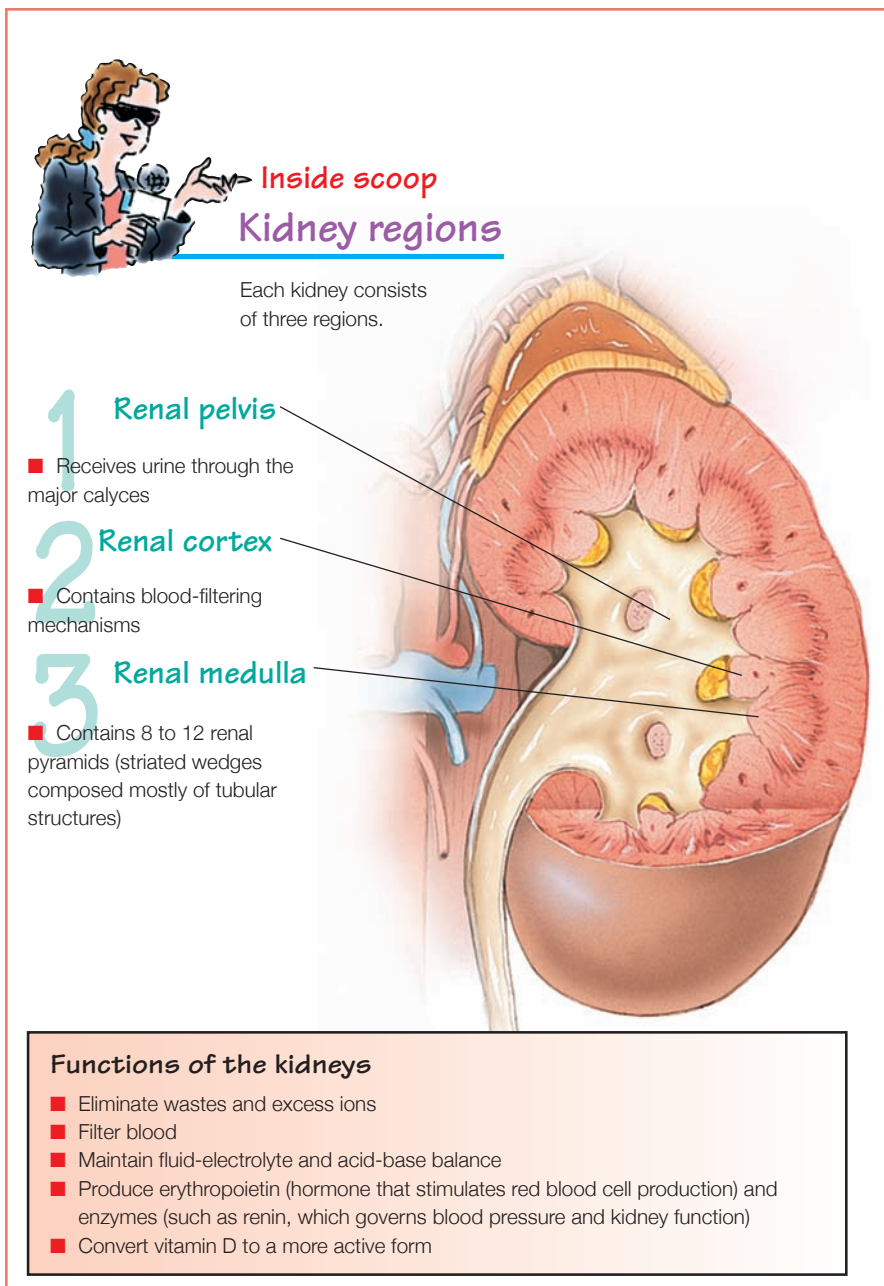
In the lungs, angiotensin I encounters angiotensin-converting enzyme and is converted from angiotensin I to angiotensin II. Angiotensin II has two main functions: (1) to act as a potent vasoconstrictor, resulting in increased BP and (2) to stimulate the release of aldosterone from the adrenal glands. In the adrenal glands, angiotensin II triggers release of aldosterone by the adrenal cortex.

Aldosterone acts on the kidney at the distal tubules and promotes the reabsorption of sodium and water into the blood. This increases the volume of fluid in the body, which also increases BP.

Active in the bone marrow, **erythropoietin** is a glycoprotein produced by the kidney in response to decreased oxygen levels. Functionally, erythropoietin may also have a protective effect on various organs, including the kidneys. In CKD, erythropoietin production is supplemented to avoid chronic anemia.

Receptors in the kidney turn **vitamin D** into its active form—1,25-dihydroxyvitamin D or calciferol. In its active form, vitamin D balances calcium and phosphorus by controlling their absorption from food and regulates the parathyroid hormone.

The kidneys maintain **acid-base balance** by reabsorbing bicarbonate from and excreting



hydrogen ions into urine. The kidneys filter roughly 5,000 mmol of bicarbonate, but only 2 mmol is expelled through the urine daily. This action takes place in the distal portion of the nephrons; however, the transport of hydrogen ions takes place in the proximal tubule. If the kidneys fail to filter hydrogen ions, this will result in metabolic acidosis.

Urine concentration is controlled by the hypothalamus and the posterior pituitary gland. When there's an increase in blood osmolality, the posterior pituitary gland secretes antidiuretic hormone, which causes water reabsorption in the kidney and urine to become more concentrated. In healthy kidneys, urine osmolality ranges from 50 to 1,200 mmol/kg, depending on hydration status.

Measuring normal kidney function

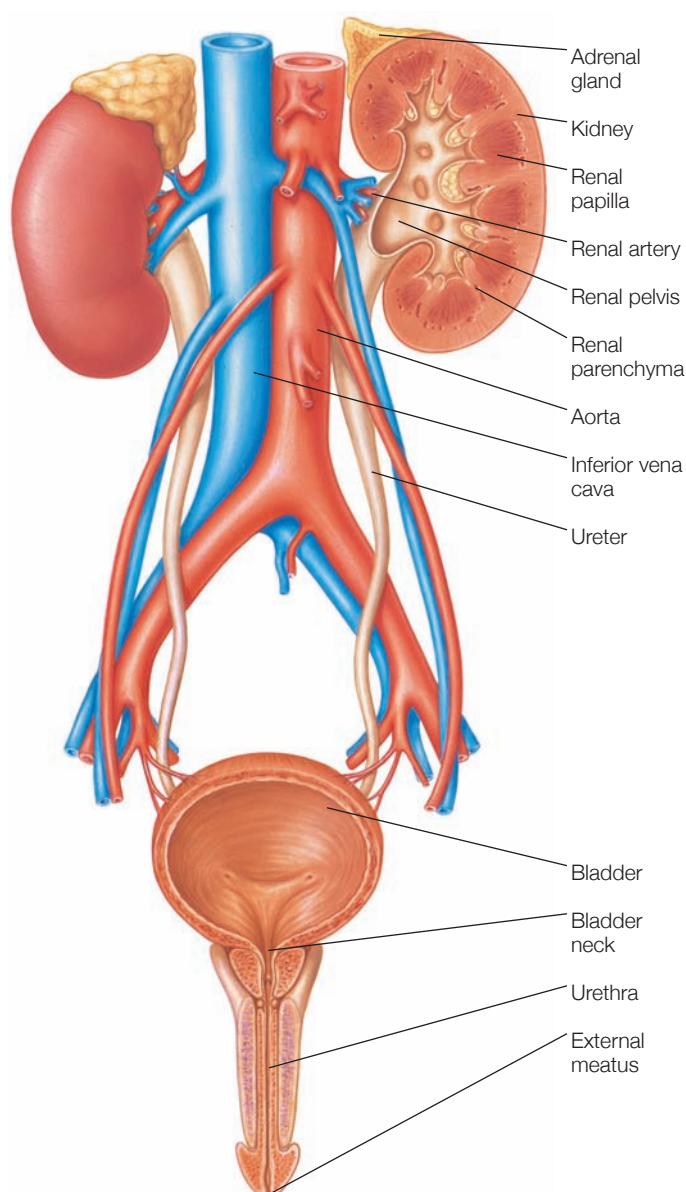
Several lab tests are used to monitor kidney function, including serum creatinine, serum blood urea nitrogen (BUN), glomerular filtration rate (GFR), urine specific gravity, and urine osmolality.

Creatinine is the end product of muscle and protein metabolism. The body generates approximately 1.8 g of creatinine each day, and virtually all of it is excreted in urine. Normal creatinine levels are 0.5 to 1.1 mg/dL for women and 0.6 to 1.2 mg/dL for men, but levels are affected by muscle mass. The more muscle, the higher the creatinine level. Therefore, you can expect a healthy young man to have a higher creatinine level than an elderly or adolescent man.

BUN is the concentration of urea in the blood, regulated by the rate at which the kidney excretes urea. Normal findings are 10 to 30 mg/dL, but the GFR is normally the best indicator of overall kidney function.

The **GFR** is an estimation of the actual clearance of filtrate in the glomerulus. To calculate GFR, one formula you can use is serum creatinine multiplied by age in years multiplied by 0.742 if female or 1.21 for Black patients. Normal GFR levels vary according

Urinary system components



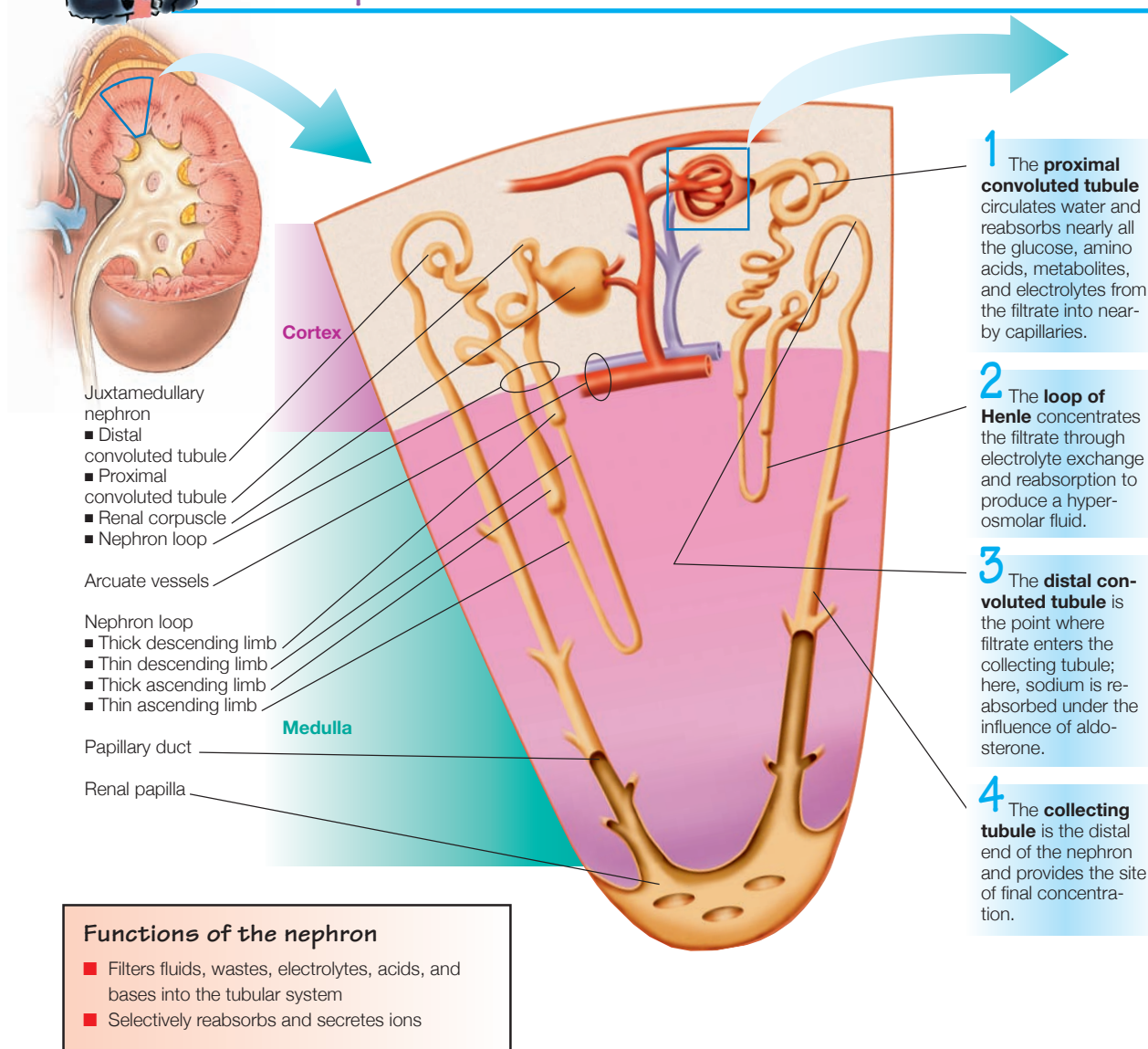
to a patient's age, sex, ethnicity, and body mass index. As a general rule, normal GFR is approximately 100 mL/min/1.73m².

Urine specific gravity indicates the concentrating ability of the kidneys. Low specific gravity indicates dilute urine and possibly excessive diuresis. High specific gravity indi-

cates dehydration. A fixed specific gravity of about 1.010 indicates renal inability to concentrate urine, suggesting progression to end-stage renal disease (ESRD). **Urine osmolality** (more accurate than specific gravity) determines the diluting and concentrating ability of the kidneys. Findings indicate if the



Inside scoop The nephron



kidneys have lost their ability to concentrate or dilute urine. Normal findings are 300 to 1,300 mOsm/kg.

On the lookout for AKI

AKI is the sudden loss of the kidney's ability to regulate volume, remove waste products from the body, release hormones, or maintain the body's acid-base balance. AKI was once known as acute renal failure, which implies failure of the entire renal system, not a very accurate description. In 2004, the Acute Dialysis Quality Initiative was created with the task of developing a definition of AKI and evidence-based guidelines for treatment and prevention. The acronym RIFLE (risk, injury, failure, loss, end-stage) was developed to define the increasing severity of AKI.

The two most common causes of AKI are prolonged renal ischemia and nephrotoxic injury leading to tubular necrosis. AKI is categorized into prerenal, intrinsic, and postrenal causes (see *Mechanism of AKI*).

Prerenal causes of AKI are usually external, which in turn reduces blood flow (hypoperfusion) to the kidneys. When blood flow is reduced, so is oxygen delivery, which leads to decreased glomerular perfusion and filtration. Poor perfusion can result from renal vasoconstriction, hypotension, hypovolemia, or inadequate cardiac output. For patients in prerenal AKI, urinalysis is typically nonspecific or with hyaline casts, urine sodium is low (less than 1%), and urine osmolality is high.

Intrinsic causes of AKI are usually conditions that lead to parenchyma (renal tissue) damage, resulting in impaired nephron function. Acute tubular necrosis (ATN), normally caused by ischemia or nephrotoxins, is the most common cause of intrinsic AKI. ATN caused by ischemia usually occurs after surgery, but is also associated with sepsis, severe burns, or trauma. Nephrotoxic ATN can point to several causative agents, such as poisoning, overdose, and some therapeutic drugs (particularly

did you know?

Current research includes regenerating kidney tissue using stem cells to restore function to damaged kidneys. Nanotechnology is also being developed to shrink dialysis filtration systems to coffee cup size.

aminoglycosides and nonsteroidal anti-inflammatory drugs). Contrast media may also be nephrotoxic.

Postrenal causes of AKI normally involve some type of obstruction of urinary outflow in the bladder, ureters, or urethra. The obstruction causes an increase in intraluminal pressure, with a gradual fall in GFR. This can occur after catheterization of the ureters or from stones or congenital anomalies. Patients present with anuria and flank pain.

AKI may progress through four phases: initiation, oliguric, diuretic, and recovery.

- The **initiation phase** of AKI usually begins at the time of injury and continues until the signs and/or symptoms become apparent—usually hours to days.
- The **oliguric phase** is the reduction of urine output to 400 mL in 24 hours, usually within 1 to 7 days of the causative agent.
- The **diuretic phase** usually starts with a urine output of 1 to 3 L per day, sometimes increasing to 3 to 5 L or more per day. This high urine volume is caused by osmotic diuresis from the high urea concentration in the glomerular filtrate and the inability of the tubules to concentrate the urine. In this phase, the kidneys have acquired their ability to excrete waste but not concentrate urine. With the large losses of fluid and electrolytes, the patient must be monitored for hyponatremia, hypokalemia, and dehydration. The diuretic phase may last from 1 to 3 weeks.
- The **recovery phase** begins when the filtration rate increases, allowing the BUN and serum creatinine levels to decrease and electrolyte balance to be

Acute kidney injury can usually be resolved.



Mechanism of AKI

Prerenal failure

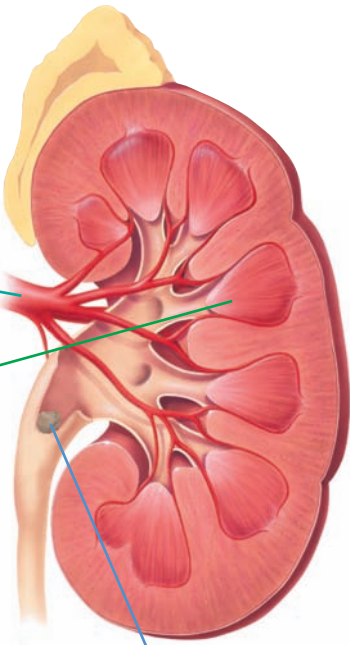
(marked decrease in renal blood flow)

- Antihypertensives
- Arrhythmias that cause reduced cardiac output
- Arterial embolism
- Arterial or venous thrombosis
- Ascites
- Burns
- Cardiac tamponade
- Cardiogenic shock
- Dehydration
- Disseminated intravascular coagulation
- Diuretic overuse
- Eclampsia
- Heart failure
- Hemorrhage
- Hypoalbuminemia
- Hypovolemic shock
- Malignant hypertension
- Myocardial infarction
- Pulmonary embolism
- Sepsis
- Trauma
- Tumor
- Vasculitis

Intrarenal failure

(damage to the structures of the kidney)

- Acute glomerulonephritis
- Acute tubular necrosis
- Acute pyelonephritis
- Bilateral renal vein thrombosis
- Crush injuries
- Malignant nephrosclerosis
- Myopathy
- Nephrotoxins
- Obstetric complications
- Papillary necrosis
- Polyarteritis nodosa
- Poorly treated prerenal failure
- Renal myeloma
- Scleroderma
- Sickle cell disease
- Systemic lupus erythematosus
- Transfusion reaction
- Vasculitis



Postrenal failure

(obstruction of urine outflow from kidney [renal calculi])

- Bladder obstruction
- Prostatic hypertrophy
- Ureteral obstruction
- Urethral obstruction

maintained. However, completely normal renal function may take up to 12 months to return.

Older adults are more susceptible to AKI than younger adults because the number of functioning nephrons decreases as the kidney ages, so it's less able to compensate for changes in fluid volume, solute load, and cardiac output. Common causes of AKI in older adults include dehydration, hypotension, diuretic therapy, and obstruction.

When taking care of patients with AKI, be vigilant in monitoring vital signs (particularly BP and heart rate) and intake and output of all fluids. Patients must be weighed daily and should be on a high-calorie, low-protein, low-potassium, low-sodium diet. Assess the patient's general appearance, including skin color, peripheral edema, neck vein distension, and bruises (see *AKI: What to look for*). Maintain normal electrolyte balance, particularly serum potassium levels. Teach your patient to avoid high-potassium

foods and sports drinks. The overall goal should be for the individual to recover completely without any loss of kidney function.

Prerenal and postrenal AKI can be resolved quickly when the cause is corrected. However, in some cases, the patient may not recover from AKI and CKD results.

Do you see CKD?

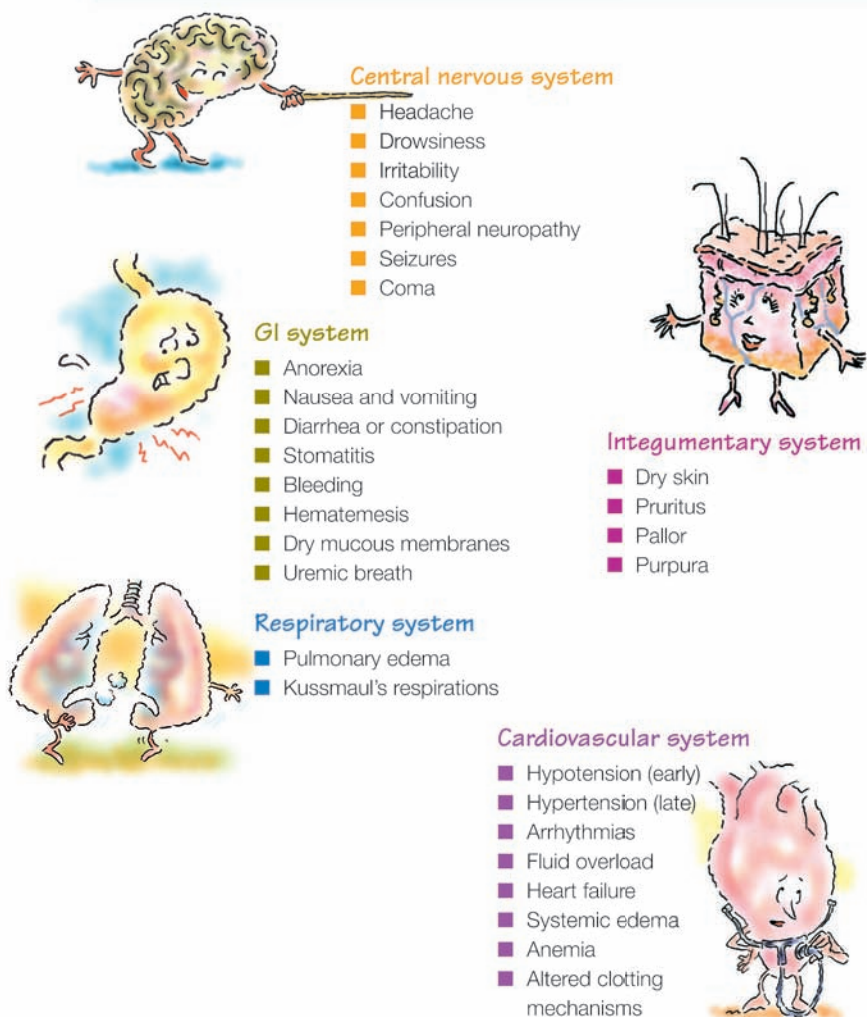
CKD begins with a slow decline in renal function and is irreversible. When damaged kidneys have been unable to process waste efficiently for longer than 3 months, as indicated by abnormal creatinine levels, the patient is considered to have CKD.

Unless preceded by AKI initially, there may not be any signs or symptoms of kidney disease, and substantial damage can occur before the disease is detected. In the early stages of CKD, serum creatinine levels may even be normal, but the kidneys begin leaking protein or red blood cells into the urine. By the time the disease is detected, the GFR can be substantially reduced.

Further testing is done to investigate the cause of the damage and formulate a treatment plan to control causative factors such as hypertension and high blood glucose levels. Diagnostic tests for the kidney may include measuring creatinine levels, urinalysis, magnetic resonance imaging, a computed tomography scan, ultrasound, or biopsy.

AKI: What to look for

AKI is a critical illness. Its early signs and symptoms are oliguria (decreased urine output), azotemia (excess levels of urea in blood) and, rarely, anuria (failure to secrete urine). Electrolyte imbalance, metabolic acidosis, and other severe effects follow as the patient becomes increasingly uremic and renal dysfunction disrupts other body systems.



CKD is staged from I to V based on the severity of the disease:

- **stage I**—indicates slightly diminished kidney function with kidney damage (normal or relatively high GFR of greater than or equal to 90 mL/min/1.73m²)

- **stage II**—indicates a mild reduction in GFR (60 to 89 mL/min/1.73m²) with kidney damage
- **stage III**—indicates a moderate reduction in GFR (30 to 59 mL/min/1.73m²)
- **stage IV**—indicates a severe reduction in GFR (15 to 29 mL/min/1.73m²)

Hemodialysis vs. peritoneal dialysis

Hemodialysis

Advantages

- May be done at home and/or at a dialysis center
- Removes fluid rapidly
- Excellent for potassium removal
- Less protein loss
- Lowers serum triglycerides quickly
- Quickly removes urea and creatinine
- Temporary access may be obtained quickly

Disadvantages

- Problems obtaining vascular access
- Very strict diet and fluid limits
- Heparin may be required to keep access open
- Blood loss, which contributes to anemia
- Trained personnel required
- Permanent access requires surgical procedure
- May take several hours to recover from treatment

Peritoneal dialysis

Advantages

- No need for vascular access
- Less dietary and fluid restriction
- Better BP control
- Not as complicated as hemodialysis
- Can be done at home
- Causes less cardiovascular problems
- Increased mobility
- Easier to manage for patients with diabetes

Disadvantages

- Infection risk (peritonitis)
- Nutritional complications (loss of protein)
- Chronic back pain or hernia
- Risk of hyperglycemia
- Surgery needed for catheter placement
- Catheter may migrate
- Contraindicated for patients with abdominal surgeries
- Transporting solutions and pump needed for travel
- Increased risk of hyperlipidemia
- Must be done daily

- **stage V**—indicates a GFR of less than 15 mL/min/1.73m² and the need for complete renal support.

Risk factors for CKD include the following:

- **diabetes.** Almost 40 % of dialysis patients have diabetes. Tight glycemic control is the key to prevention, keeping the HbA1c at less than 7.

- **hypertension.** High BP stresses all vessels, and nephrons are no exception. Keep BP within the normal range (at or below 120/80 mm Hg).

- **inflammation.** Illnesses such as glomerulonephritis or an immune response to diseases such as strep throat are common causes of kidney dysfunction.

- **family history.** A positive family history of kidney disease dramatically increases the likelihood of CKD. Certain diseases, such as polycystic kidney disease, damage the kidney over time.

- **premature birth.** Premature babies born before 28 weeks' gestation are at higher risk for impaired kidney function due to immature kidneys.

- **age.** Changes in vasculature occur with aging, and the microcirculation of the kidney is particularly susceptible.

- **blockages.** Blockages within the renal system can cause urine to back up into the kidney instead of draining.

- **drugs.** Opioid analgesics and allergic reactions or other adverse reactions to certain drugs can damage the kidneys.

- **past treatment.** Treatment for all stages of kidney disease begins with finding and controlling the causes of the damage. Good control of blood glucose and BP are paramount, and diet restrictions of protein, potassium, sodium, and phosphorus become necessary as the disease progresses.

Black patients account for 30% of all kidney disease cases on record. Black Americans are four times more likely to develop CKD than White Americans. Diabetes and hypertension are the two leading causes of CKD among Black patients:

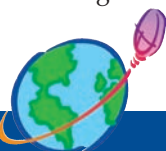
39% of all kidney disease in Black patients is linked to diabetes; 34% is linked to hypertension.

Long-term renal support

CKD describes a continuum from renal failure that doesn't resolve to ESRD—the complete or almost complete failure of the kidneys. When this happens, renal support, either by dialysis or by kidney transplant, is the only option left for survival.

Dialysis must be started when the GFR is less than 15 mL/minute. Hemodialysis and peritoneal dialysis are the two methods used to remove fluid, waste, and toxins (see *Hemodialysis vs. peritoneal dialysis*). Hemodialysis requires a vascular access site, such as an arteriovenous shunt, port, or fistula. Peritoneal dialysis requires access via an implanted port with a catheter placed in the anterior wall of the abdomen.

A transplanted kidney can come from either a living donor or a cadaver. The waiting list for transplant from deceased donors grows each year, but the supply of available organs remains fairly stable. Every person waiting for transplant is listed with the Organ Procurement and Transplantation Network, which maintains links to all regional organ-gathering organizations. Matching a donor organ to a recipient is based on matching blood type and human leukocyte antigen. The wait time for a deceased donor kidney varies based on availability but in 2009, 46% of persons on the waiting list age 60 or over died waiting for a cadaver kidney transplant.



On the web

- **American Diabetes Association:** <http://www.diabetes.org/living-with-diabetes/complications/kidney-disease-nephropathy.html>
- **Mayo Clinic:** <http://www.mayoclinic.com/health/kidney-failure/DS00682>
- **MedlinePlus:** <http://www.nlm.nih.gov/medlineplus/kidneydiseases.html>
- **National Kidney and Urologic Diseases Information Clearinghouse:** <http://www.kidney.niddk.nih.gov>
- **National Kidney Foundation:** <http://www.kidney.org>

A living donor can also donate one of his or her kidneys. The donation is generally between two relatives and has several distinct advantages: time (the donation occurs without the use of a registry and waiting list) and compatibility (in general, it's easier to find a close match among family members).

The transplant surgery is straightforward for the recipient. The donated organ with the ureter still attached is inserted into the abdomen, arteries and veins are attached, and the donor ureter is diverted into the urinary bladder. Frequently, the new kidney begins to function immediately, but there may be some delay of days or weeks before the new kidney becomes functional. The failed kidneys aren't usually removed.

Life after transplant includes antirejection medications to suppress the patient's immune system so the body doesn't reject the new kidney. Teach patients to:

- take medication exactly as ordered.
- avoid eating grapefruit or drinking grapefruit juice when taking tacrolimus
- notify the healthcare provider if they experience signs or symptoms of infectious disease while on immunosuppressive therapy
- wear sunscreen, examine their skin frequently for changes, and notify their healthcare provider if any new symptoms or concerns about their health arise.

The 3-year graft survival of a deceased donor kidney is 81%; for a living donor kidney, 90%. The 3-year survival rate for transplant patients is in the 90% range.

Keeping up the flow

Normal, healthy adults should consume 2 to 3 L of fluid a day to maintain adequate urine flow. A balanced, healthy diet plays a key role in maintaining healthy kidneys, as does maintaining tight blood glucose and BP control. Knowing how urine is created and the other functions of the kidney can help you keep your patients informed and aware of this complex system, both in the prevention and management of renal disease. ■

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