Table 550.1 KDIGO Staging of Acute Kidney Injury

STAGE	SERUM CREATININE	URINE OUTPUT
1	1.5-1.9 times baseline, OR ≥0.3 mg/dL increase	<0.5 mL/kg/hr for 6-12 hr
2	2.0-2.9 times baseline	<0.5 mL/kg/hr for ≥ 12 hr
3	3.0 times baseline, OR SCr ≥ 4.0 mg/dL, OR Initiation of renal replacement therapy, OR eGFR < 35 mL/min per 1.73 m^2 (< 18 yr)	<0.3 mL/kg/hr for ≥ 24 hr, OR Anuria for ≥ 12 hr

Table 550.2 Common Causes of Acute Kidney Injury

PRERENAL
- Dehydration
- Gastroenteritis
- Hemorrhage
- Burns
- Sepsis
- Capillary leak
- Hypoalbuminemia
- Cirrhosis
- Abdominal compartment syndrome
- Cardiac failure
- Anaphylaxis

INTRINSIC RENAL
- Glomerulonephritis
  - Postinfectious/poststreptococcal
  - Lupus erythematosus
  - Henoch-Schönlein purpura
  - Membranoproliferative
  - Anti–glomerular basement membrane
- Hemolytic-uremic syndrome
- Acute tubular necrosis
- Cortical necrosis
- Renal vein thrombosis
- Rhabdomyolysis
- Acute interstitial nephritis
- Tumor infiltration
- Toxin and drugs (see Table 550.3)
- Tumor lysis syndrome
- Vasculitis

POSTRENAL
- Posterior urethral valves
- Ureteropelvic junction obstruction
- Ureterovesicular junction obstruction
- Ureterocele
- Tumors
- Urolithiasis
- Urethral strictures
- Hemorrhagic cystitis
- Neurogenic bladder
- Anticholinergic drugs

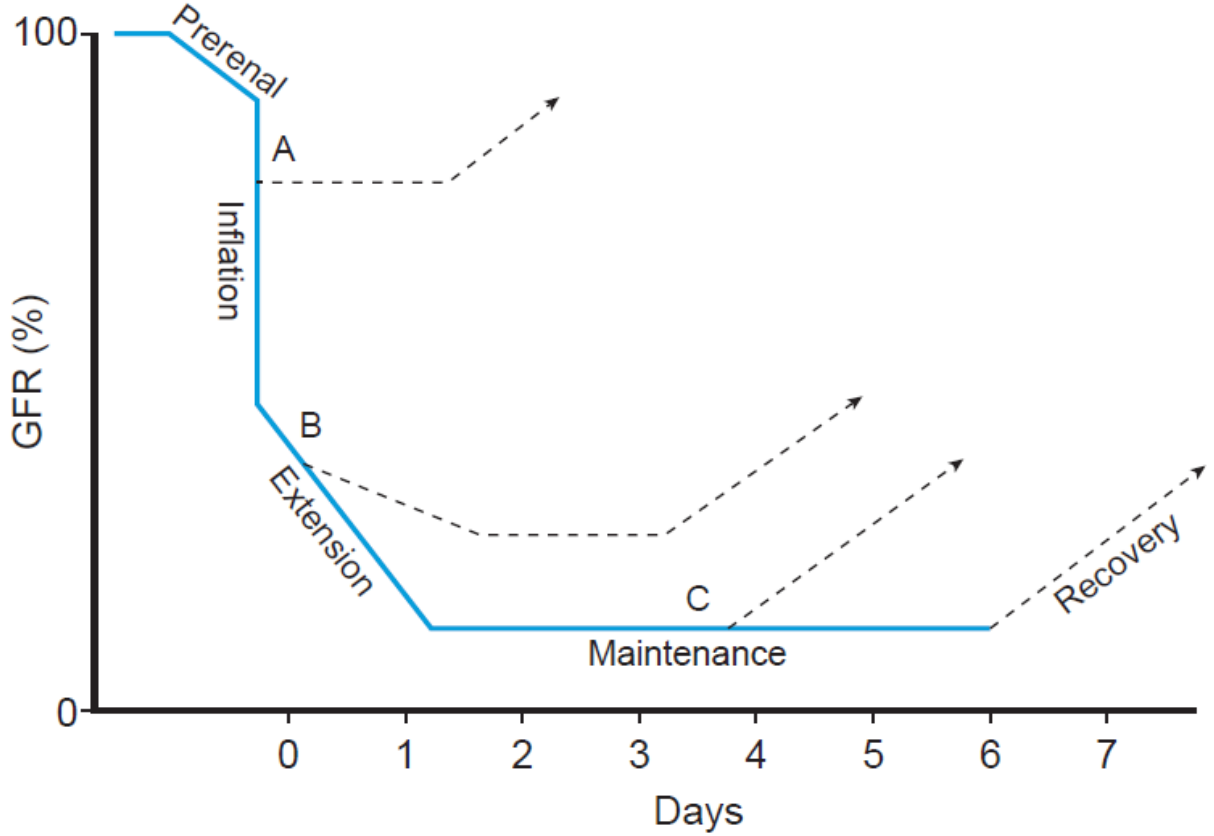

Fig. 550.1 Phases of acute kidney injury. GFR, glomerular filtration rate. *(From Sutton TA, Fisher CJ, Molitoris BA: Microvascular endothelial injury and dysfunction during ischemic acute renal failure, Kidney Int 62:1539-1549, 2002.)*

Table 550.3 Major Endogenous and Exogenous Toxins Causing Acute Tubular Injury

ENDOGENOUS TOXINS	EXOGENOUS TOXINS
MYOGLOBULINURIA	**ANTIBIOTICS**
Muscle breakdown—trauma, compression, electric shock, hypothermia, hyperthermia, seizures, exercise, burns	Aminoglycosides
Metabolic—hypokalemia, hypophosphatemia	Amphotericin B
Infections—tetanus, influenza	Antiviral agents—acyclovir, cidofovir, indinavir, foscarnet, tenofovir
Toxins—isopropyl alcohol, ethanol, ethylene glycol, toluene, snake and insect bites, cocaine, heroin	Pentamidine
Drugs—HMG-CoA reductase inhibitors (statins), amphetamines, fibrates	Vancomycin
Inherited disease—deficiency of myophosphorylase, phosphofructokinase, carnitine palmitoyltransferase	**CHEMOTHERAPY**
Autoimmune—polymyositis, dermatomyositis	Cisplatin
	Ifosfamide
	Plicamycin
	5-Fluorouracil
	Cytarabine
	6-Thioguanine
	Methotrexate
HEMOGLOBINURIA	**CALCINEURIN INHIBITORS**
Mechanical—prosthetic valves, microangiopathic hemolytic anemia, extracorporeal circulation	Cyclosporine
Drugs—hydralazine, methyldopa	Tacrolimus
Chemicals—benzene, arsine, fava beans, glycerol, phenol	**ORGANIC SOLVENTS**
Immunologic—transfusion reaction	Toluene
Genetic—G6PD deficiency, PNH	Ethylene glycol
	POISONS
	Snake venom
	Paraquat
	MISCELLANEOUS
	Radiocontrast media
	Intravenous immune globulin
	Nonsteroidal antiinflammatory drugs
	Oral phosphate bowel preparations
INTRATUBULAR OBSTRUCTION FROM CRYSTALLURIA OR PARAPROTEINS	
Tumor lysis syndrome	
HGPT deficiency	
Multiple myeloma	
Oxalate (ethylene glycol)	

G6PD, Glucose-6-phosphate dehydrogenase; HGPRT, hypoxanthine-guanine phosphoribosyltransferase; HMG-CoA, 3-hydroxy-3-methylglutaryl–coenzyme A; PNH, paroxysmal nocturnal hemoglobinuria.

From Sharfuddin AA, Weisbord SD, Palevsky PM, Molitoris BA: Acute kidney injury. In Skorecki K, Chertow GM, Marsden PA, et al (eds): Brenner & Rector's the kidney, 10/e, Philadelphia, 2016, Elsevier, Tab 31-5.

Table 550.4 Urinalysis, Urine Chemistries, and Osmolality in Acute Kidney Injury

	HYPOVOLEMIA	ACUTE TUBULAR NECROSIS	ACUTE INTERSTITIAL NEPHRITIS	GLOMERULONEPHRITIS	OBSTRUCTION
Sediment	Bland, may have hyaline casts	Broad, brownish granular casts	White blood cells, eosinophils, cellular casts	Red blood cells, red blood cell casts	Bland or bloody
Protein	None or low	None or low	Minimal but may be increased with NSAIDs	Increased, > 100 mg/dL	Low
Urine sodium (mEq/L)*	<20	>40	>30	<20	<20 (acute) >40 (few days)
Urine osmolality (mOsm/kg)	>400	<350	<350	>400	<350
Fractional excretion of sodium %†	<1	>2‡	Varies	<1	<1 (acute) >1 (few days)

*The sensitivity and specificity of urine sodium of < 20 mEq/L in differentiating prerenal azotemia from acute tubular necrosis are 90% and 82%, respectively.
†Fractional excretion of sodium is the urine:plasma (U:P) ratio of sodium divided by U:P of creatinine ×100. The sensitivity and specificity of fractional excretion of sodium of < 1% in differentiating prerenal azotemia from acute tubular necrosis are 96% and 95%, respectively.
‡The fractional excretion of sodium may be < 1% in acute tubular necrosis secondary to radiocontrast material or rhabdomyolysis.
NSAIDs, nonsteroidal antiinflammatory drugs.

Table 550.5 Common Complications of Acute Kidney Injury

METABOLIC	CARDIOPULMONARY	GASTROINTESTINAL	NEUROLOGIC	HEMATOLOGIC	INFECTIOUS	OTHER
Hyperkalemia Metabolic acidosis Hyponatremia Hypocalcemia Hyperphosphatemia Hypermagnesemia Hyperuricemia	Pulmonary edema Arrhythmias Pericarditis Pericardial effusion Hypertension Myocardial infarction Pulmonary embolism	Nausea Vomiting Malnutrition Hemorrhage	Neuromuscular irritability Asterixis Seizures Mental status changes	Anemia Bleeding	Pneumonia Septicemia Urinary tract infection	Hiccups Elevated parathyroid hormone level Low total triiodothyronine and thyroxine levels Normal thyroxine level

Table 550.6 Comparison of Peritoneal Dialysis, Intermittent Hemodialysis, and Continual Renal Replacement Therapy

	PD	IHD	CRRT
BENEFITS			
Fluid removal	+	++	++
Urea and creatinine clearance	+	++	+
Potassium clearance	++	++	+
Toxin clearance	+	++	+
COMPLICATIONS			
Abdominal pain	+	–	–
Bleeding	–	+	+
Dysequilibrium	–	+	–
Electrolyte imbalance	+	+	+
Need for heparinization	–	+	+/–
Hyperglycemia	+	–	–
Hypotension	+	++	+
Hypothermia	–	–	+
Central line infection	–	+	+
Inguinal or abdominal hernia	+	–	–
Peritonitis	+	–	–
Protein loss	+	–	–
Respiratory compromise	+	–	–
Vessel thrombosis	–	+	+

PD, peritoneal dialysis; IHD, intermittent hemodialysis; CRRT, continual renal replacement therapy.

Table 550.9 Etiologies of Pediatric Chronic Kidney Disease

NONGLOMERULAR	GLOMERULAR
Aplastic, hypoplastic, and dysplastic kidneys	Chronic glomerulonephritis (including focal segmental glomerulonephritis [FSGS])
Cystinosis	Congenital nephrotic syndrome (CNS)
Medullary cystic kidney disease/juvenile nephronophthisis	Hemolytic-uremic syndrome (HUS)
Obstructive uropathy (e.g., PUV, cloaca, neurogenic bladder)	Henoch-Schönlein nephritis (HSP nephritis)
Oxalosis	Idiopathic crescentic glomerulonephritis
Autosomal dominant and autosomal recessive polycystic kidney disease (ADPKD, ARPDK)	IgA nephropathy (IGAN)
Pyelonephritis/interstitial nephritis/reflux nephropathy	Membranoproliferative glomerulonephritis (MPGN)
Renal infarct	Membranous nephropathy
Syndrome of agenesis of abdominal musculature (Eagle-Barrett syndrome)	Sickle cell nephropathy
Wilms tumor	Systemic immunologic disease (e.g., SLE, Wegener granulomatosis)
	Hereditary nephritis (Alport syndrome)

Table 550.7 Criteria for Definition of Chronic Kidney Disease (NKF KDOQI Guidelines)

Patient has chronic kidney disease (CKD) if either of the following criteria are present:

1. Kidney damage for ≥ 3 mo, as defined by structural or functional abnormalities of the kidney, with or without decreased GFR, manifested by one or more of the following features:
   - Abnormalities in the composition of the blood or urine
   - Abnormalities in imaging tests
   - Abnormalities on kidney biopsy
2. GFR < 60 mL/min/1.73 m^2 for ≥ 3 mo, with or without the other signs of kidney damage described above

NKF KDOQI, National Kidney Foundation Kidney Disease Outcomes Quality Initiative.

Table 550.8 Standardized Terminology for Stages of Chronic Kidney Disease (NKF KDOQI Guidelines)

STAGE	DESCRIPTION	GFR (mL/min/1.73 m^2)
1	Kidney damage with normal or increased GFR	≥90
2	Kidney damage with mild decrease in GFR	60-89
3	Moderate decrease in GFR	30-59
4	Severe decrease in GFR	15-29
5	Kidney failure	<15 or on dialysis

GFR, glomerular filtration rate; NKF KDOQI, National Kidney Foundation Kidney Disease Outcomes Quality Initiative.

Table 550.10	Pathophysiology of Chronic Kidney Disease
MANIFESTATION	**MECHANISMS**
Accumulation of nitrogenous waste products	Decrease in glomerular filtration rate
Acidosis	Decreased ammonia synthesis Impaired bicarbonate reabsorption Decreased net acid excretion
Sodium wasting	Solute diuresis Tubular damage
Urinary concentrating defect	Solute diuresis Tubular damage
Hyperkalemia	Decrease in glomerular filtration rate Metabolic acidosis Excessive potassium intake Hyporeninemic hypoaldosteronism
Renal osteodystrophy	Impaired renal production of 1,25-dihydroxycholecalciferol ($1,25OH_2D$) Hyperphosphatemia Hypocalcemia Secondary hyperparathyroidism
Growth retardation	Inadequate caloric intake Renal osteodystrophy Metabolic acidosis Anemia Growth hormone resistance

Continued

Anemia	Decreased erythropoietin production Iron, folate, and/or vitamin B_{12} deficiency Decreased erythrocyte survival
Bleeding tendency	Defective platelet function
Infection	Defective granulocyte function Impaired cellular immune functions Indwelling dialysis catheters
Decreased academic achievement, attention regulation, or executive functioning	Hypertension Low birth weight
Gastrointestinal symptoms (feeding intolerance, abdominal pain)	Gastroesophageal reflux Decreased gastrointestinal motility
Hypertension	Volume overload Excessive renin production
Hyperlipidemia	Decreased plasma lipoprotein lipase activity Abnormal HDL-C
Cardiomyopathy	Hypertension Anemia Fluid overload
Glucose intolerance	Tissue insulin resistance

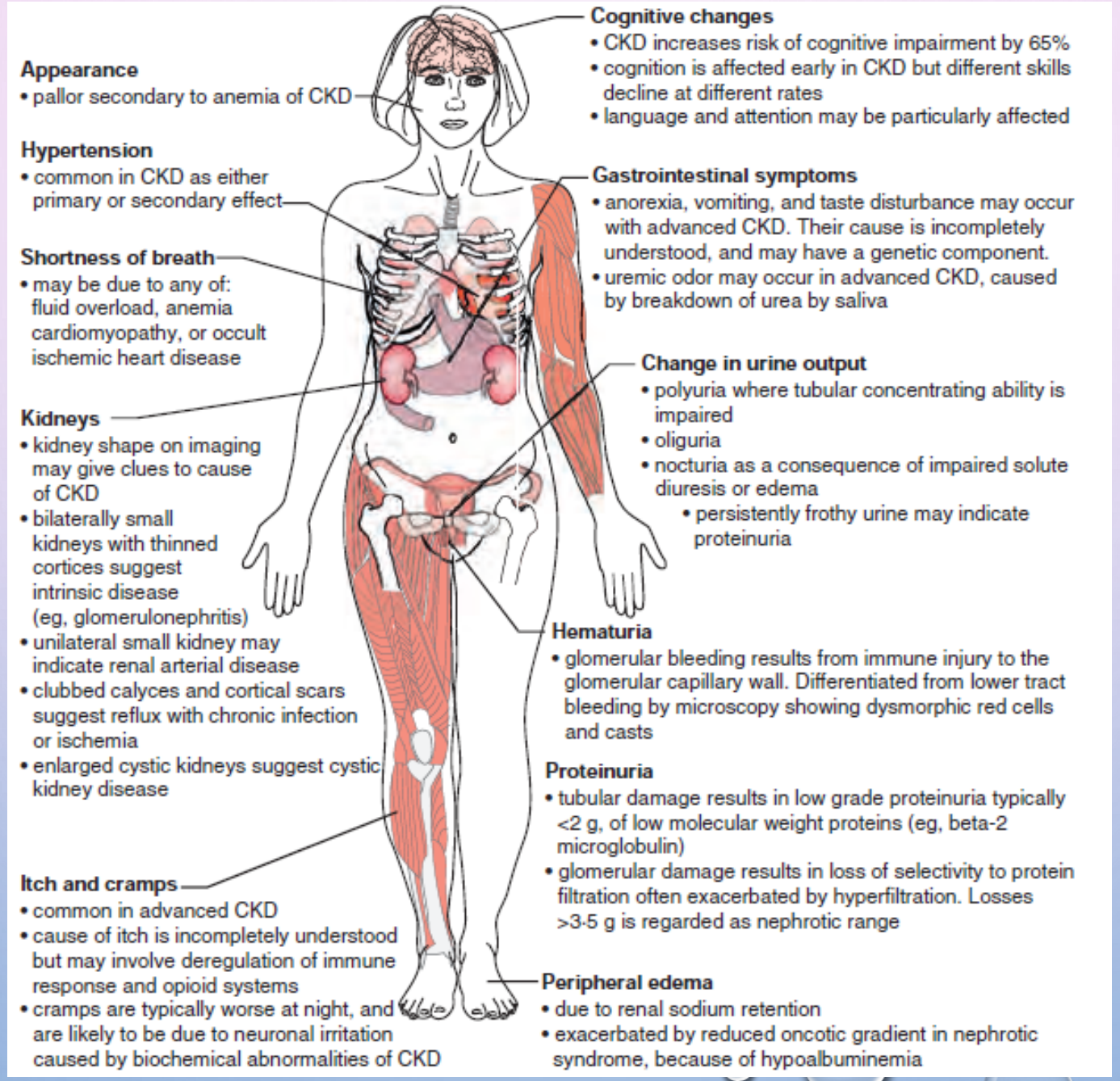

Fig. 550.2 **Symptoms and signs of CKD**.

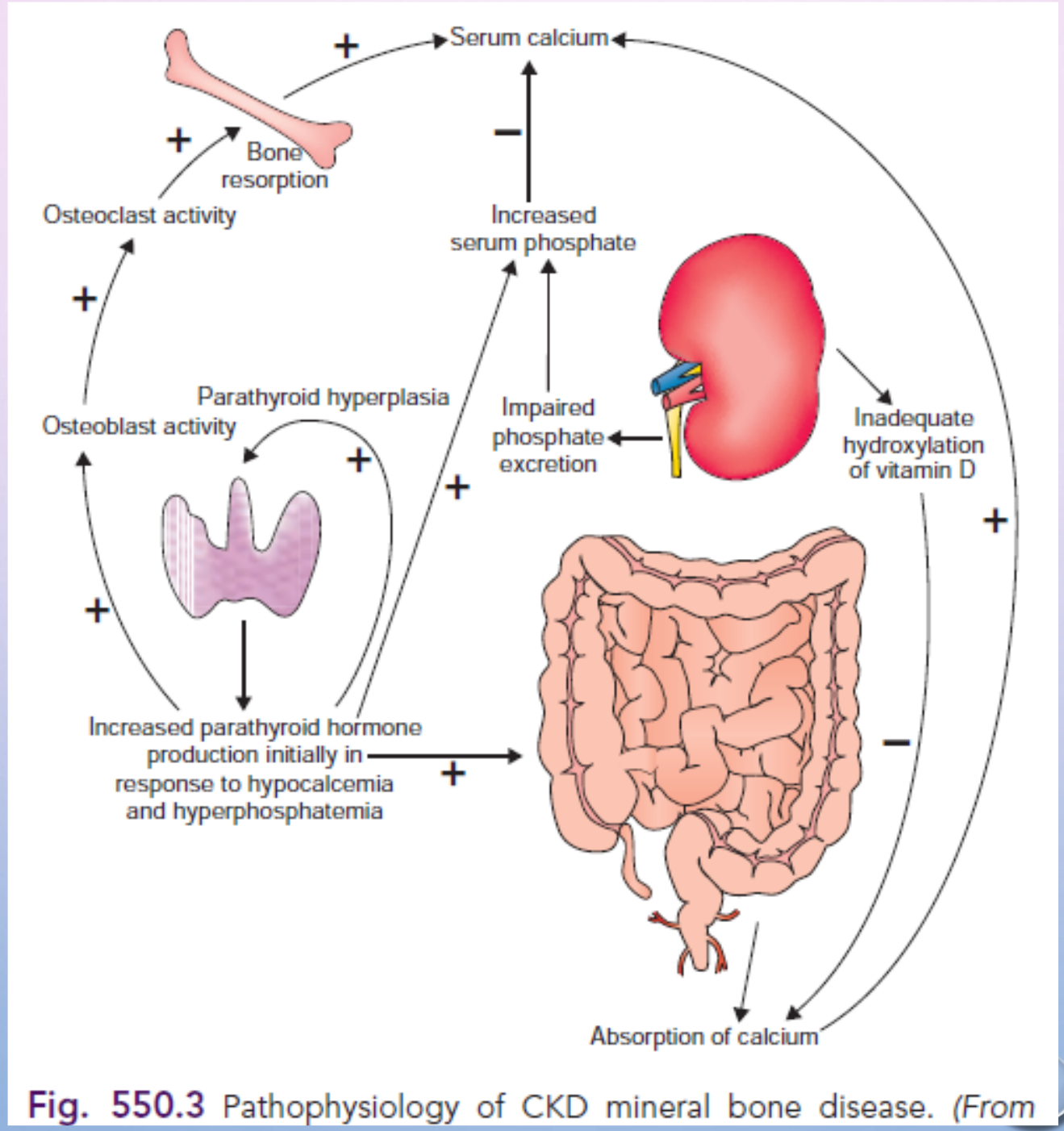

Fig. 550.3 Pathophysiology of CKD mineral bone disease. *(From*