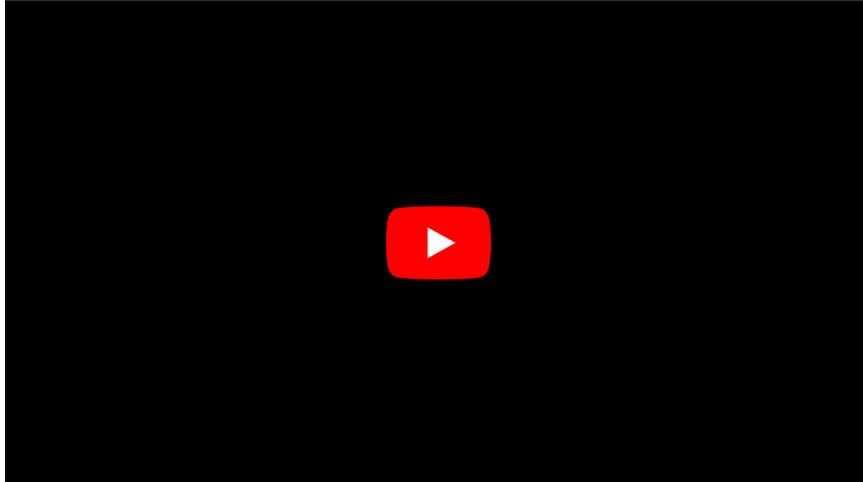


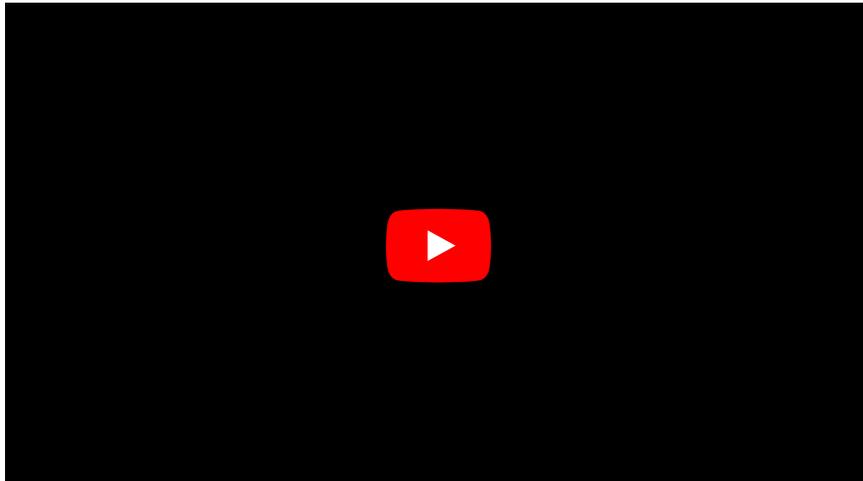
Acute Renal Failure

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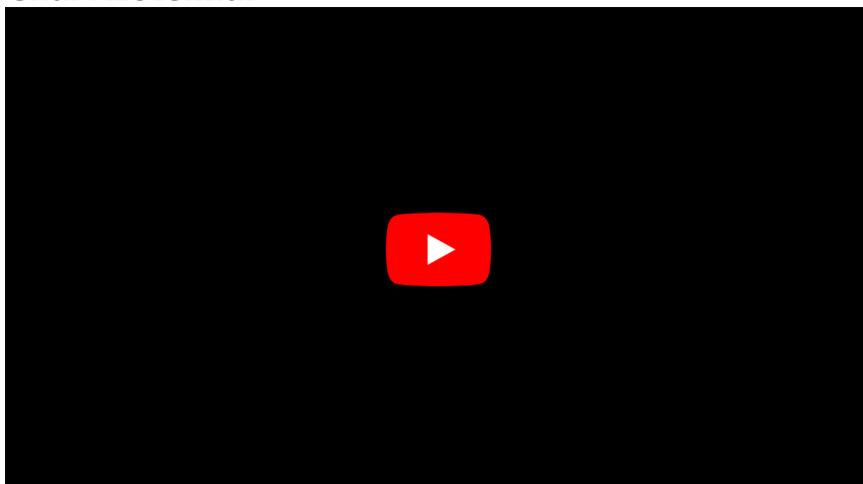
AKI (functions affected) / ARF (old term):



AKI (and ATN):



Prerenal vs Intrarenal Azotemia:



Definition of Disease Acute renal failure is also referred to as acute kidney injury and may result from renal parenchymal disease, renal obstruction or decreased renal circulation. The course of injury is reversible with medical intervention. Injury that is nonresponsive to treatment may advance to uremic syndrome, end-stage renal disease or death.

Detailed pathophysiology at cellular, tissue, organ, and system levels Acute renal failure may result from three separate etiologies: acute urinary tract obstruction, disease of renal interstitial tissue or decreased renal perfusion.

Acute obstruction of the urinary tract is an uncommon occurrence that normally affects both kidneys when it does occur. Obstructions such as tumors, neurogenic bladder, prostatic hypertrophy and bladder outlet problems increase the intraluminal pressure just prior to the point of obstruction. This results in a subsequent decrease in glomerular filtration rate.

Disorders of the interstitial renal tissues include post ischemic kidney injury due to hypotension, hypoperfusion or hypoxemia. Ischemia produces cellular edema and death. Invasion of the tissue by inflammatory cells such as neutrophils, macrophages and lymphocytes emit inflammatory cytokines that, in turn, damage the renal tubules. This damage impairs the proximal tubular epithelium and alters the transport of sodium. It is also responsible for the appearance of tubular granular casts that are observed in the urinalysis

Renal hypoperfusion is caused by a rapid and sudden decrease in arterial blood volume and is evidenced by an elevation in blood urea nitrogen and plasma creatinine. Decrease in arterial blood volume may result from hypovolemia, hemorrhage, renal vasoconstriction, microemboli, renal edema or renal artery thrombosis. Compensatory mechanisms preserve glomerular filtration rate (GFR) in the early stages of hypoperfusion through afferent arteriolar dilation and efferent arteriolar vasoconstriction. These mechanisms are soon overcome and GFR begins to decrease.

Genetics Evidence is inconclusive to support a genetic basis for acute renal failure.

Epidemiology Every year in the United States, approximately 100 out of every one million individuals experience acute renal failure. Acute renal failure is diagnosed in 1% of general hospital admissions and 20% of critical care unit admissions. Four percent of all individuals admitted to in-patient care experience hospital-acquired renal failure.

Disease described Acute renal failure is characterized by a precipitous decrease in renal function and glomerular filtration rate. Elevated blood urea nitrogen and plasma creatinine signal an accumulation of nitrogenous waste products in the blood.

Sign and Symptoms

- Oliguria or anuria
- Azotemia
- Electrolyte imbalance
- Metabolic acidosis
- Anorexia, nausea, vomiting
- diarrhea or constipation
- Stomatitis, uremia breath
- Dry mucous membranes
- Headache
- Drowsiness
- irritability, confusion
- Seizures, coma
- Peripheral neuropathy
- pulmonary edema, Kussmaul's respirations
- Dry skin, pruritus
- Pallor
- Purpura
- Uremic frost
- Fever and chills in the presence of infection

Diagnosis

- Blood urea nitrogen
- Serum creatinine
- Potassium level
- Hemoglobin and hematocrit level
- Urinalysis with urine osmolality
- Renal ultrasonography
- Kidney, ureter, bladder x-ray
- Urography, renal scan and nephrotomography

Treatment

- Correction of volume depletion or obstruction
- Discontinue use of kidney toxic medications
- Diet high in calories and low in sodium and potassium
- Fluid restriction
- Careful monitoring of electrolytes
- Correction of hyperkalemia
- Hemodialysis or peritoneal dialysis

Related articles

- [Kidney](#)

Links National Kidney Foundation

<http://www.kidney.org/>

Current Articles Dirkes, S. (2011). Acute kidney injury: not just acute renal failure anymore? *Critical Care Nurse*, 31(1), 37-50. doi: 10.4037/ccn2011946

References

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McCance, K.L. & Heuther, V.L. (2014). *Pathophysiology the biologic basis for disease in adults and children* (7th ed.). St. Louis, MO: Elsevier Mosby