

Acute kidney injury & Renal replacement therapy

By

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Acute kidney injury

Acute kidney injury is defined : An abrupt (within 48 hrs.) decrease in renal function that is clinically significant (i.e., can have adverse consequences).

- 70% of ICU patients have some degree of acute renal dysfunction,
- 5% require renal replacement therapy.

Diagnostic Considerations

Diagnostic Criteria

1. An increase in **serum creatinine** of ≥ 0.3 mg/dL within 48 hours, or
2. An increase in **serum creatinine** of $\geq 50\%$ within 48 hours, or
3. A decrease in **hourly urine output** to **40 mEq/L**. (**urinary sodium excretion (Na+ <40 mEq/L)**)

EXCEPTIONS: A prerenal disorder can be associated with a high urine sodium if there is ongoing diuretic therapy, or the patient has chronic renal disease with an obligatory sodium loss in urine.

Initial Management

- The following recommendations apply to the initial management of a patient who develops acute kidney injury (AKI),, especially when associated with **oliguria**.

A. What to Do

- I. **Reduce fluid intake** as much as possible.
- II. Discontinue potentially **nephrotoxic drugs**.
- III. Adjust the dose of **drugs that are excreted in the urine**.

B. What not to do

1. Do not give furosemide to correct oliguria

Intravenous furosemide does not improve renal function in AKI, and does not convert oliguric to non-oliguric renal failure.

Furosemide can increase urine output during the recovery phase of AKI, and can be used at that time if volume overload is a problem.

2. Do not use low-dose dopamine to increase renal blood flow in AKI.

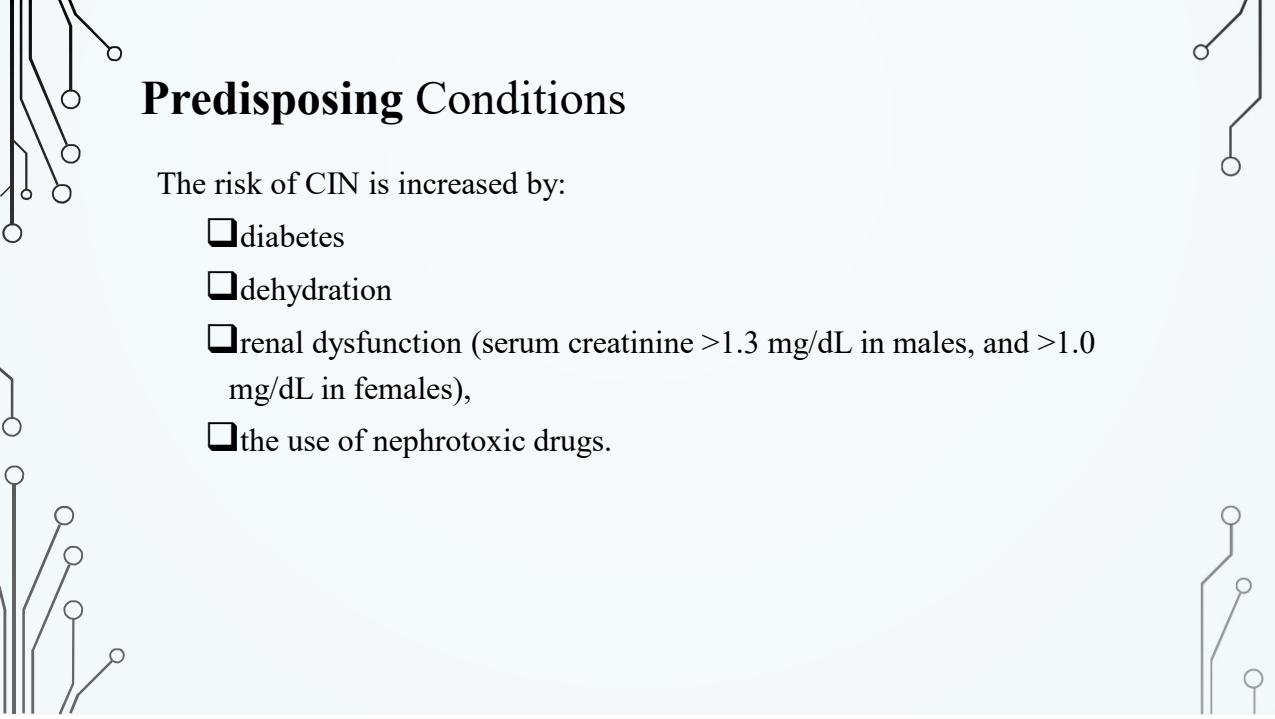
Low-dose dopamine does not improve renal function in patients with AKI, and it can have deleterious effects (e.g., decreased splanchnic blood flow, inhibition of T-cell lymphocyte function).

Specific CONDITIONS

A. Contrast-Induced Nephropathy

Iodinated contrast agents can damage the kidneys in several ways, including

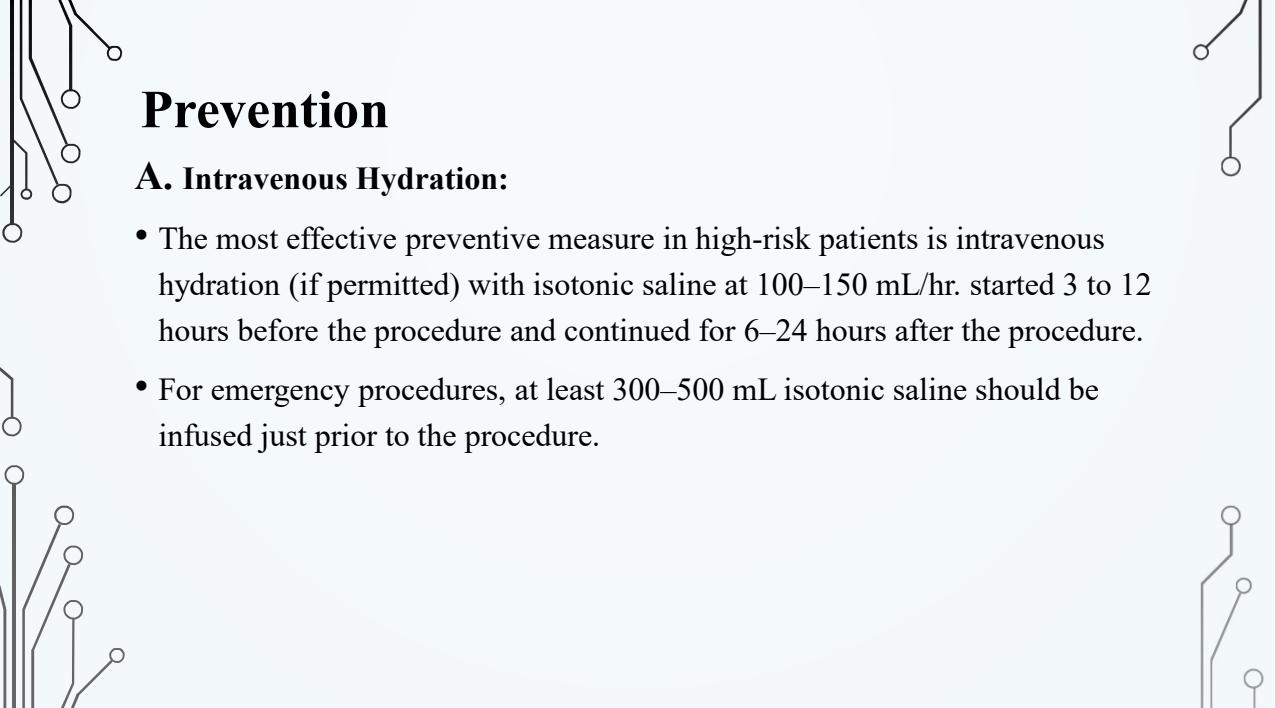
- direct renal tubular toxicity
- renal vasoconstriction
- the generation of toxic oxygen metabolites.
- The incidence of contrast-induced nephropathy (CIN) is 8– 9%. CIN appears within 72 hours after the contrast study, and most cases resolve within two weeks without renal replacement therapy.



Predisposing Conditions

The risk of CIN is increased by:

- diabetes
- dehydration
- renal dysfunction (serum creatinine >1.3 mg/dL in males, and >1.0 mg/dL in females),
- the use of nephrotoxic drugs.



Prevention

A. Intravenous Hydration:

- The most effective preventive measure in high-risk patients is intravenous hydration (if permitted) with isotonic saline at 100–150 mL/hr. started 3 to 12 hours before the procedure and continued for 6–24 hours after the procedure.
- For emergency procedures, at least 300–500 mL isotonic saline should be infused just prior to the procedure.

B. N-acetylcysteine

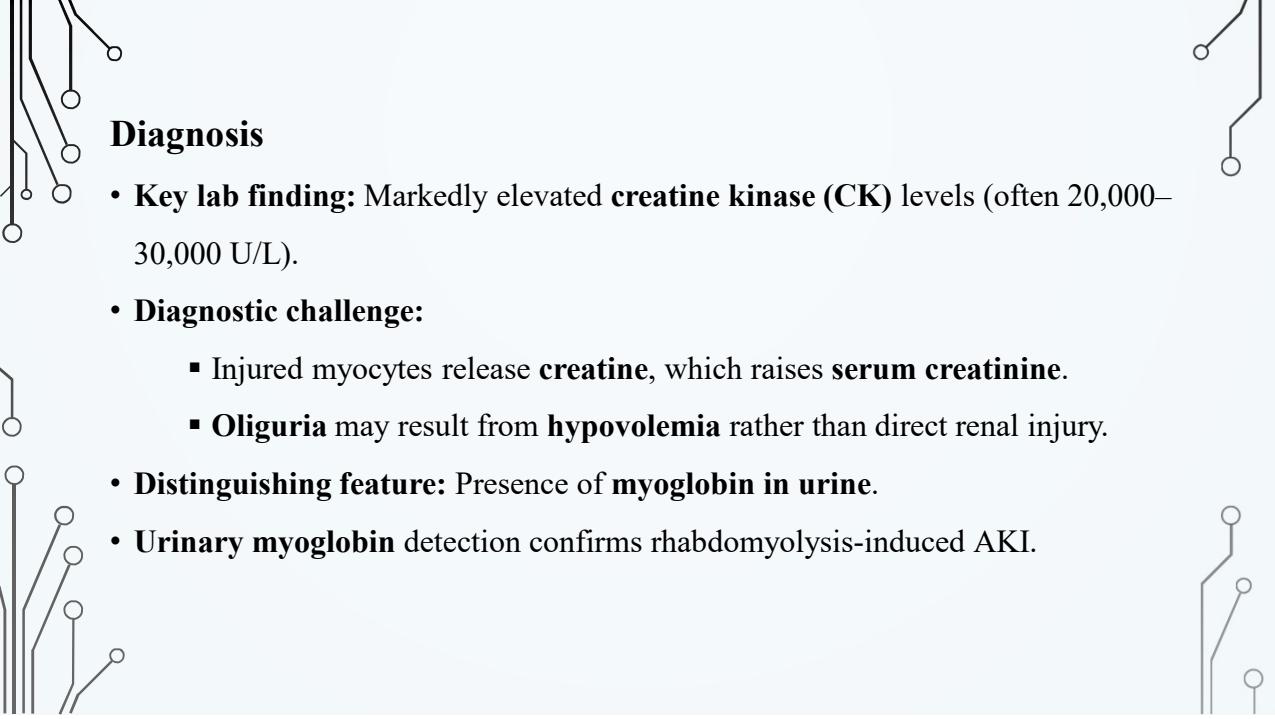
- N-acetylcysteine (NAC) is a glutathione surrogate with antioxidant actions that has had mixed results as a protective agent for CIN.
- The high-dose NAC regimen is **1,200 mg orally twice daily for 48 hours, beginning the night before the contrast procedure.**

Drug-Induced Acute Interstitial Nephritis (AIN):

- Often associated with **hypersensitivity signs**: fever, rash, eosinophilia (not always present).
- **Renal injury onset**: usually **several weeks** after first exposure, but can occur **within days after a second exposure**.

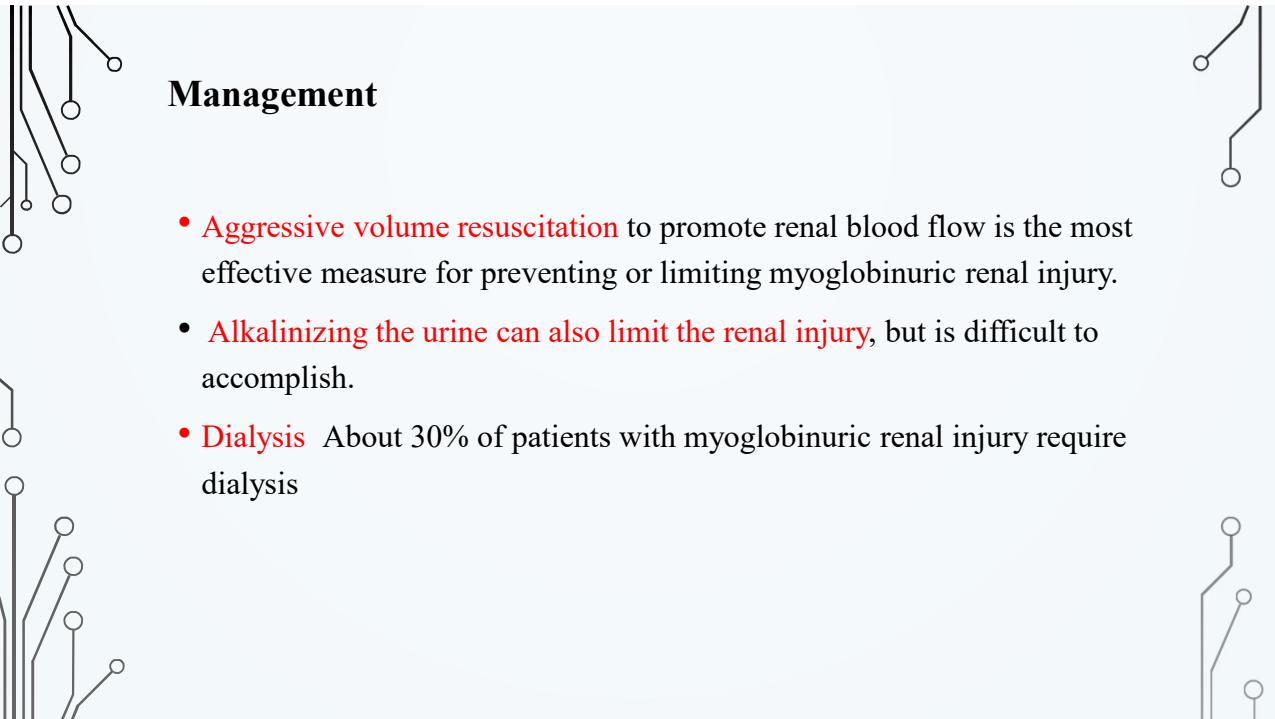
Myoglobinuric Renal Injury

- AKI develops in one-third of patients with diffuse muscle injury (rhabdomyolysis).
- The culprit is myoglobin, which is released by the injured muscle, and can damage the renal tubular epithelial cells.



Diagnosis

- **Key lab finding:** Markedly elevated **creatine kinase (CK)** levels (often 20,000–30,000 U/L).
- **Diagnostic challenge:**
 - Injured myocytes release **creatine**, which raises **serum creatinine**.
 - **Oliguria** may result from **hypovolemia** rather than direct renal injury.
- **Distinguishing feature:** Presence of **myoglobin in urine**.
- **Urinary myoglobin** detection confirms rhabdomyolysis-induced AKI.



Management

- **Aggressive volume resuscitation** to promote renal blood flow is the most effective measure for preventing or limiting myoglobinuric renal injury.
- **Alkalinating the urine can also limit the renal injury**, but is difficult to accomplish.
- **Dialysis** About 30% of patients with myoglobinuric renal injury require dialysis

Abdominal Compartment Syndrome

- An increase in intraabdominal pressure (IAP) can adversely affect renal function by decreasing both renal perfusion pressure and the net filtration pressure across the glomerulus.
- As a result, **oliguria** is one of the first signs of intraabdominal hypertension (IAH).
- When IAH is associated with organ dysfunction, the condition is called **abdominal compartment syndrome (ACS)**.

Predisposing Conditions

- ACS is traditionally associated with abdominal trauma, but several conditions can raise the IAP and predispose to ACS, including:
 - Gastric distension
 - Bowel obstruction
 - Ileus
 - Ascites
 - Bowel wall edema
 - Hepatomegaly
 - Positive-pressure breathing
 - Upright body position
 - Obesity.

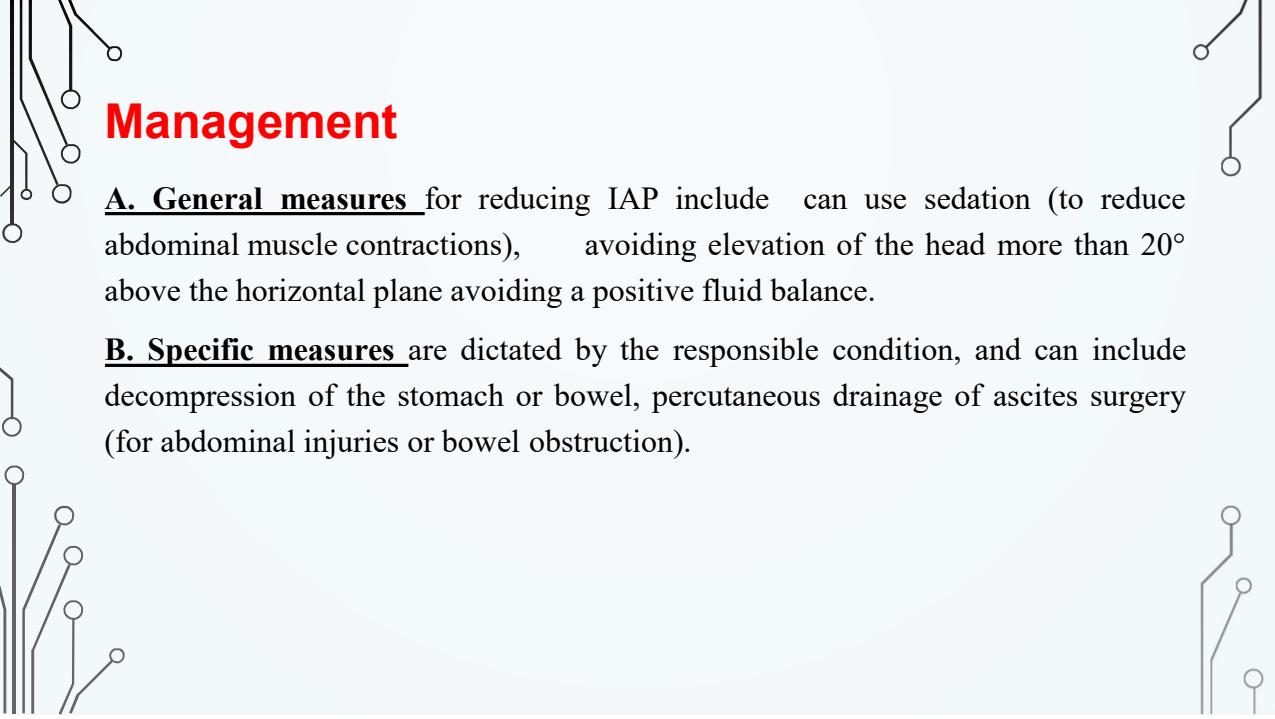
- Several of these factors can **co-exist** in critically ill patients, which explains why IAH is discovered in as many as 60% of patients in medical and surgical ICUs.

- **Large volume resuscitation:**

- Can raise IAP by promoting edema in the abdominal organs (particularly the bowel).
- One report of icu patients with a positive fluid balance >5 liters over 24 hours found that 85% of the patients had iah, and 25% had acs.

Diagnostic criteria

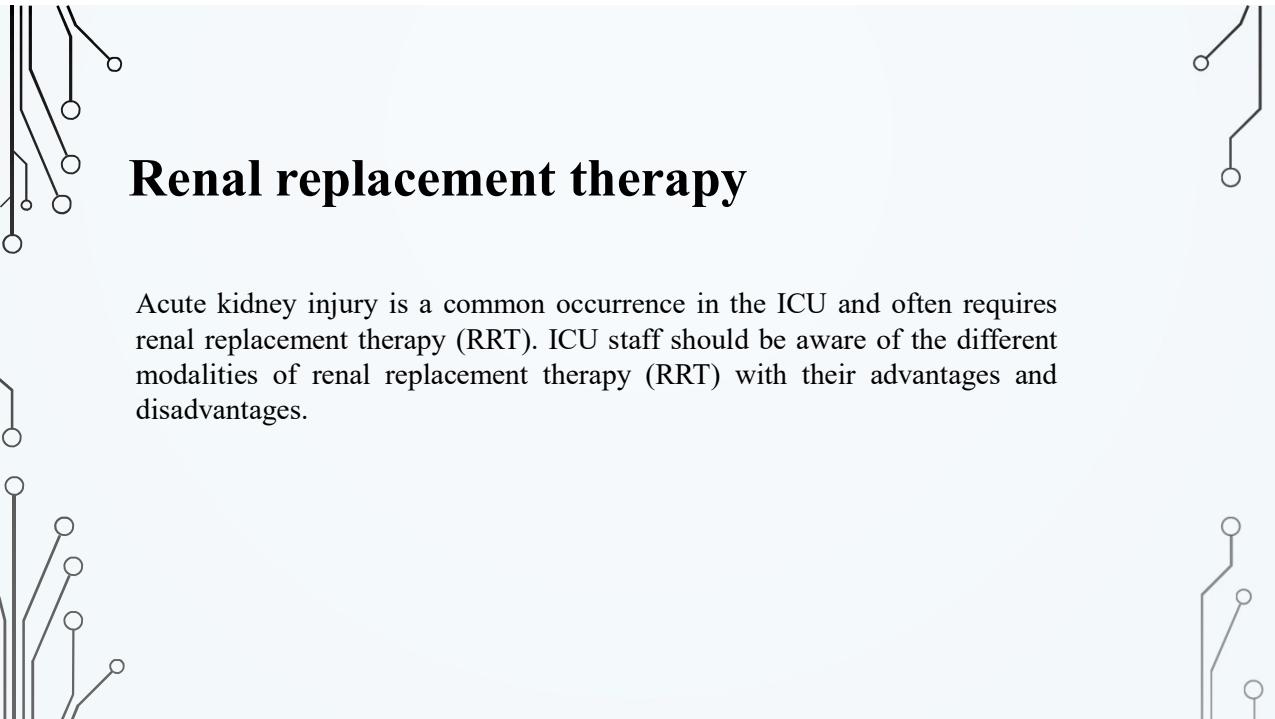
- The normal IAP is 5–7 mm Hg in the supine position.
- IAH is defined as a sustained **IAP ≥ 12 mm Hg**.
- ACS is defined as an **IAP ≥ 20 mm Hg** plus acute organ dysfunction.



Management

A. General measures for reducing IAP include can use sedation (to reduce abdominal muscle contractions), avoiding elevation of the head more than 20° above the horizontal plane avoiding a positive fluid balance.

B. Specific measures are dictated by the responsible condition, and can include decompression of the stomach or bowel, percutaneous drainage of ascites surgery (for abdominal injuries or bowel obstruction).



Renal replacement therapy

Acute kidney injury is a common occurrence in the ICU and often requires renal replacement therapy (RRT). ICU staff should be aware of the different modalities of renal replacement therapy (RRT) with their advantages and disadvantages.

initiate resuscitation and decide on RRT

- Along with resuscitation measures with ventilatory and hemodynamic support, RRT should be considered in patients with acute kidney injury.
- Optimal timing of starting RRT remains controversial, and a joint decision between the nephrologist and the intensivist should be taken.
- The usual indications of commencing RRT are the following (Fig 47.1):
 - Volume overload/pulmonary edema.
 - Refractory hyperkalemia (>6.5 mEq/L).
 - Severe metabolic acidosis ($pH < 7.1$).
 - Anuria (rising urea and creatinine)
 - Uremic encephalopathy.
 - Uremic pericarditis.

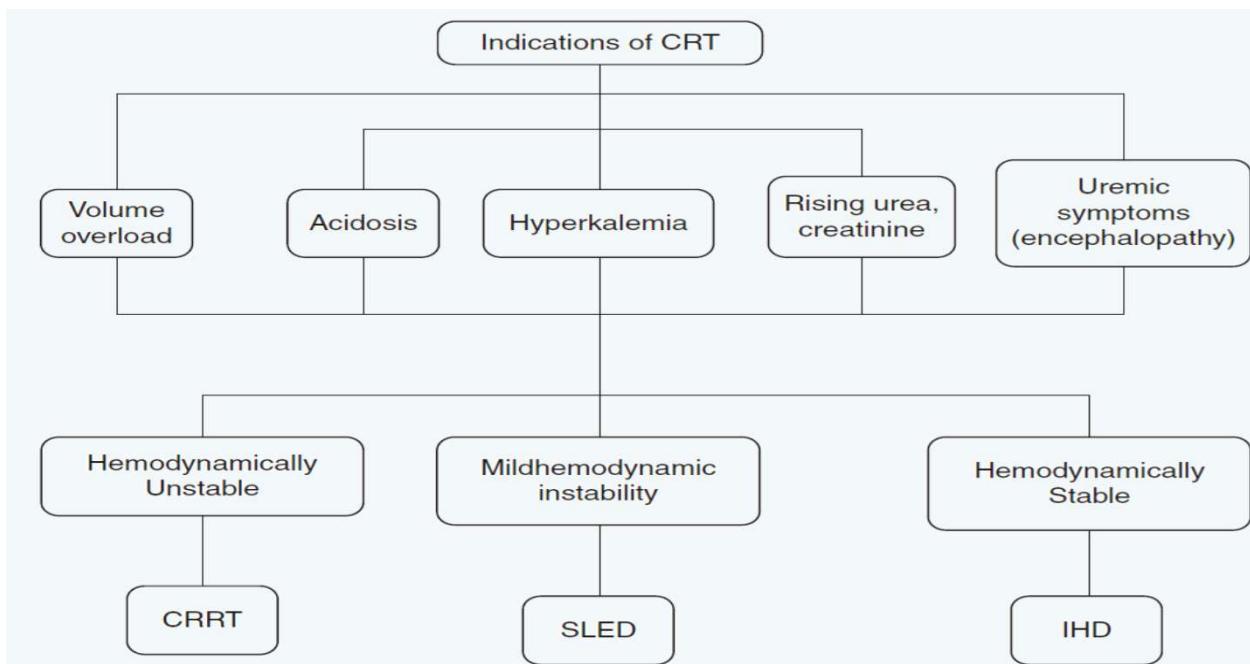


Fig. 47.1 Algorithm for choosing RRT modalities

Decide On Appropriate Modality Of RRT

Renal replacement therapy (RRT) can be performed using Intermittent Hemodialysis (IHD), Sustained Low-Efficiency Dialysis (SLED), or Continuous Renal Replacement Therapy (CRRT) all via a venovenous circuit.

- **CRRT (Continuous RRT (CRRT):** This consists of **CVVHD (Continuous venovenous hemodialysis)**, **CVVHF (Continuous venovenous hemofiltration)**, or **CVVHDF (Continuous venovenous hemodiafiltration)**: Preferred for hemodynamically unstable patients, those on multiple vasopressors, with MAP < 70 mmHg, high fluid needs (e.g., TPN), or raised intracranial pressure.
- **SLED:** Suitable if MAP > 70 mmHg and the patient requires only low-dose vasopressors.
- **IHD:** Used in hemodynamically stable patients without major volume overload.
- The final choice depends on clinical status, resource availability, cost, and physician preference.

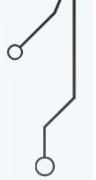
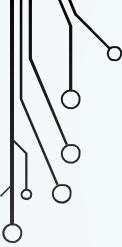
Understand Different Modalities Of RRT

RRT includes several modalities, each suited to specific patient conditions and resources.

1. Continuous Renal Replacement Therapy (CRRT)

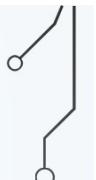
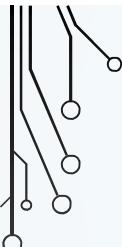
Types:

- **CVVHD:** Diffusive solute removal effective for **highly catabolic** patients.
- **CVVHF:** Convective removal of large solutes.
- **CVVHDF:** Combines both preferred in **septic shock** for removing inflammatory mediators.



Advantages:

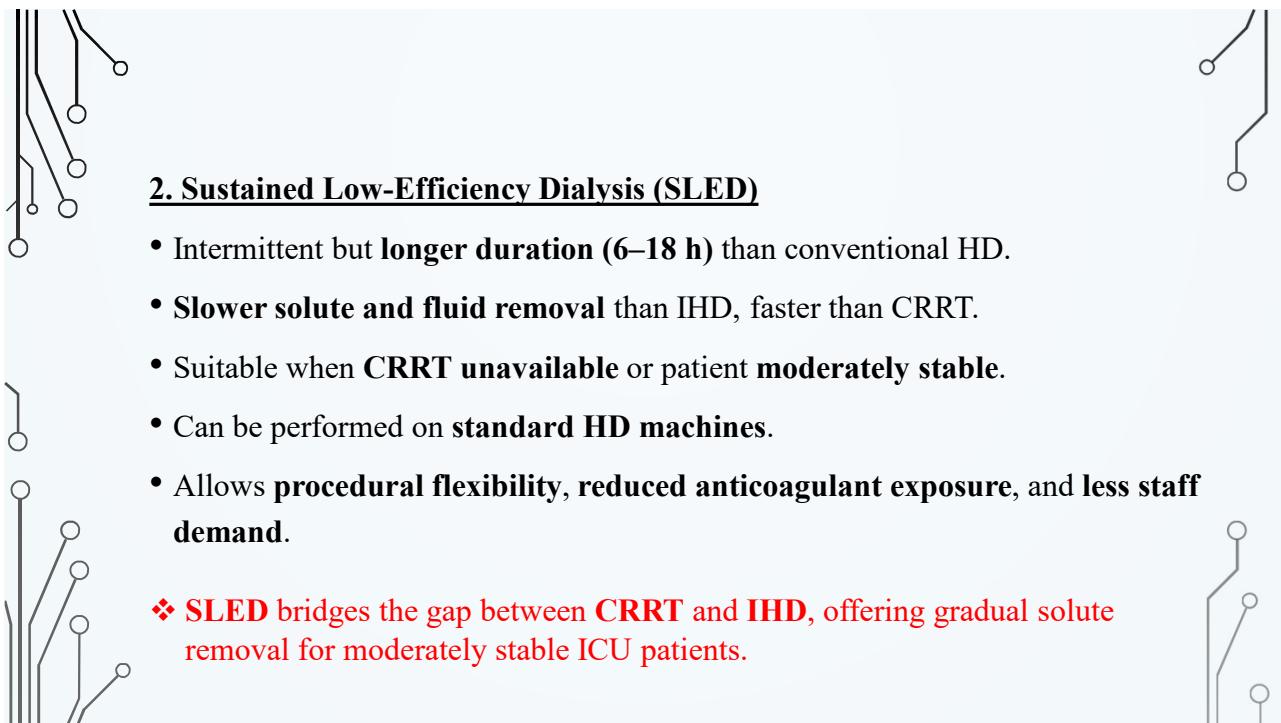
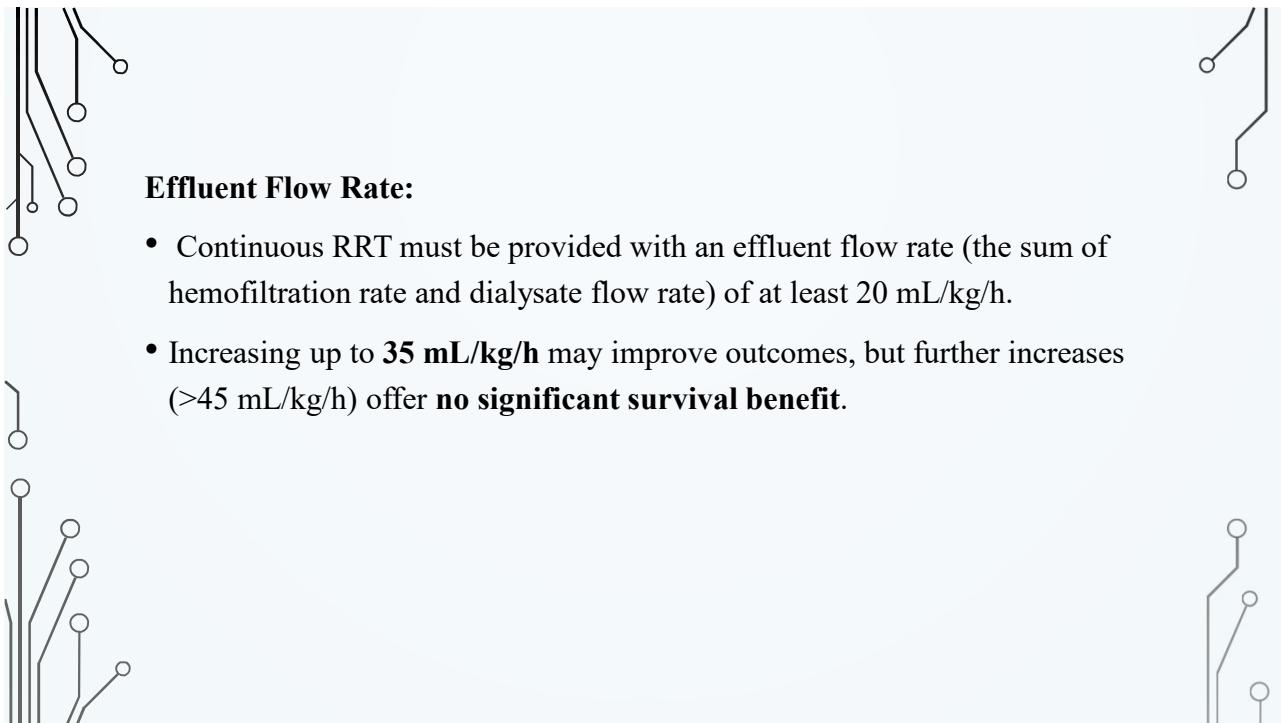
- Mimics normal kidney function with **gradual, 24-hour solute and fluid removal**.
- Improves **hemodynamic stability** and prevents **fluid overload**.
- Supports **nutritional therapy** (TPN 2–3 L/day).
- Gentler correction of **electrolytes and osmolality**, reducing risk of **ICP changes**.
- Preferred in **septic shock**.

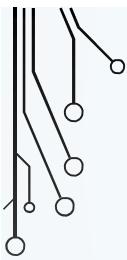


Anticoagulation in CRRT:

- **Heparin:** 1000–2000 U bolus, then 300–500 U/h.
- **Regional citrate anticoagulation (RCA):** Better filter life, less bleeding; contraindicated in **liver failure or lactic acidosis**.

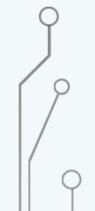
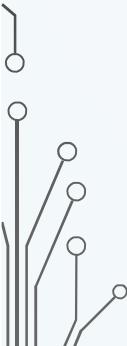






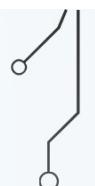
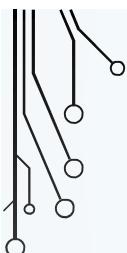
3. Intermittent Hemodialysis (IHD)

- Conventional form of dialysis.
- Used in **hemodynamically stable** patients without major fluid overload.



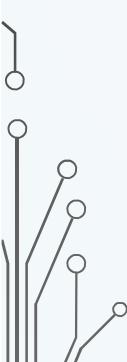
4. Slow Continuous Ultrafiltration (SCUF)

- Focuses on **fluid removal only** (no dialysate/replacement fluid).
- Used for **gradual volume removal**, e.g., in heart failure.
- UF rate \approx **up to 2 L/h**; blood flow **100–180 mL/min**.



5. Peritoneal Dialysis (PD)

- Uses **peritoneal membrane** as a natural filter.
- Solute removal by **diffusion**; fluid removal by **osmosis** (glucose-based dialysate).
- Suitable for **non-catabolic, non-hypotensive** patients or those with **bleeding risk** (no anticoagulation needed).
- **Gentle and continuous**, but **infection risk** limits its use.



*Have a wonderful
day,
my students
keep working hard and
believing in
yourselves!*

THANK YOU

