

Acute kidney injury Renal replacement therapy

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Learning goals

- Student learns how to recognize and define acute kidney injury/failure.
- Student gains basic knowledge about the methods for renal functions replacement.

Lecture summary

- Definition and diagnostics of AKI
- Pathogenesis of AKI
- Treatment of AKI
- Methods of renal replacement therapy in AKI
- Vascular access
- Anticoagulation in CRRT

Definition and diagnostics of AKI

- abrupt/prompt deterioration of the glomerular filtration rate (GFR)
- several reasons (pre-renal, renal, post-renal)
- AKI (acute kidney injury)

- occurrence in 50-60% adult patients in critical care
- 5-10% in children
- confirmed independent factor of mortality – 50-60%
- approx. 70% of patients with AKI requires renal replacement therapy (RRT)

Definition and diagnostics of AKI

– ischemic injury

- sepsis (most common reason for AKI onset)
- pancreatitis
- trauma
- part of multiorgan dysfunction syndrome (MODS)

– toxic injury

– exogenous

drugs (gentamicin, amphotericin, cisplatin, etc.)

toxins (heavy metals, plants, mushrooms, contrast media, etc.)

– endogenous

hem pigments (rhabdomyolysis)

uric acid (tumor lysis syndrome)

Definition and diagnostics of AKI

- Clinical problems in AKI:
 - **Oliguria** (<400 ml urine/day in adults, <0,5 ml/kg/h in children)
 - **fluid overload** and hypertension
 - complications with nutrition and drugs administration
 - **Reduced renal clearance**
 - electrolyte imbalances (**hyperkalemia**)
 - **metabolic acidosis** with high anion gap (HAGMA)
 - azotemia (BUN and creatinine elevation)

Pathogenesis of AKI

- pathophysiology of AKI:
 - pre-renal
 - renal
 - post-renal

Pathogenesis of AKI

– Pre-renal reasons for AKI:

- **hypovolemia** from bleeding (surgical intervention, trauma, GIT bleeding), gastrointestinal losses (diarrhea, vomiting), renal losses (diuretics, diabetes insipidus) or skin losses (burns)
- **decrease in effective perfusion pressure** and/or effective circulating volume in heart failure, shock or liver cirrhosis
- **combined pathology**, e.g. sepsis (hypovolemia + changes on cellular level + drug toxicity, e.g. antibiotics)

Pathogenesis of AKI

- Renal reasons for AKI:
 - **vascular** – thrombosis, HUS, vasculitis, malignant hypertension
 - **infection** – sepsis, glomerulonephritis
 - **tubular and interstitial pathology** – acute tubular necrosis, rhabdomyolysis, hemoglobinuria, sepsis, pancreatitis

Pathogenesis of AKI

- Post-renal reasons for AKI:
 - bilateral obstruction of urine passage
 - relative rare in children (posterior urethral valve)
 - other reasons may include **tumor, compression from outside** or **trauma**

Treatment of AKI

- basis common principles of AKI treatment:
 - restore/retain electrolyte homeostasis (Na, K, Ca, P) and fluid balance
 - diuretics (furosemide) – only to solve fluid overload and hyperkalemia, not the treatment of AKI itself
 - proper nutritional support
 - prevention of life-threatening complications
 - elimination of endogenous and exogenous toxins as fast as possible
 - treatment of underlying illness
 - dose reduction of administered drugs (e.g. antibiotics)

Methods of renal replacement therapy in AKI

- **intracorporeal** – peritoneal dialysis (sometimes in children)
- **extracorporeal** – intermittent hemodialysis (iHD), continual elimination methods (CRRT, continual renal replacement therapy)

Methods of renal replacement therapy in AKI

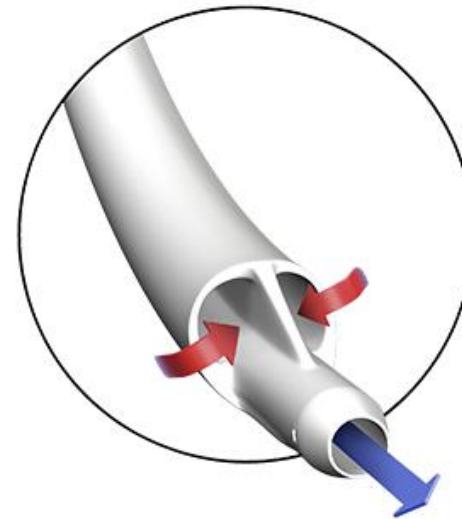
- **peritoneal dialysis**: sometimes used in children when CRRT not available, less effective in changing the ions concentration and body water volume, well tolerated in circulatory instability, simplest, serious risk of infection
- **iHD**: fastest changes in the ions concentration and body water removal compared to other methods, oldest, widely used, most effective, very complicated in critical care patients, mainly because of circulatory instability
- **CRRT**: several modalities (CVVD, CVVHD, CVVHDF), slow and continuous speed of ions and water removal, well tolerated in circulatory compromised patients

Vascular access

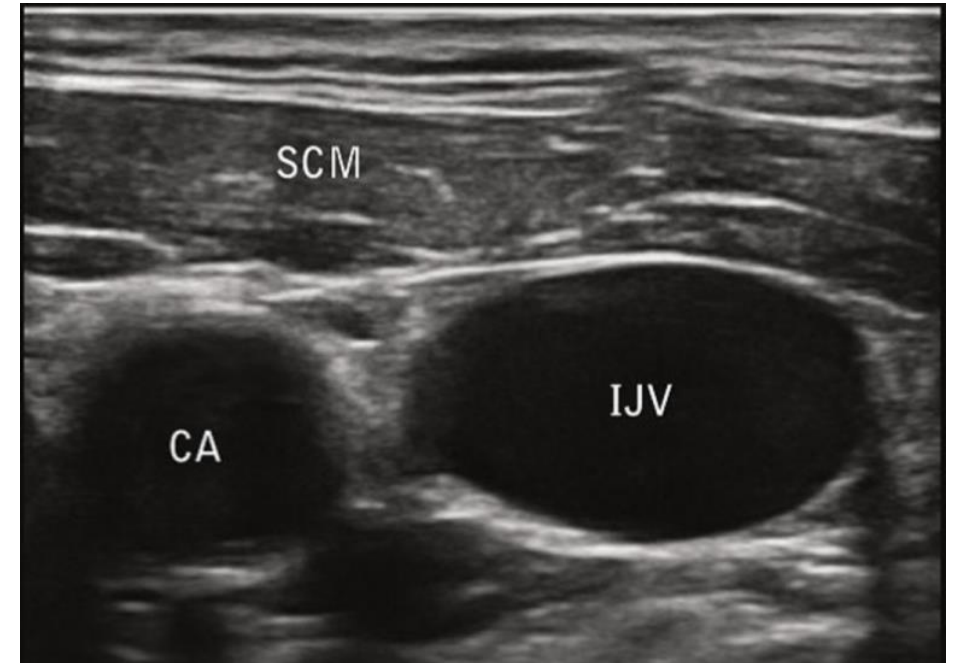
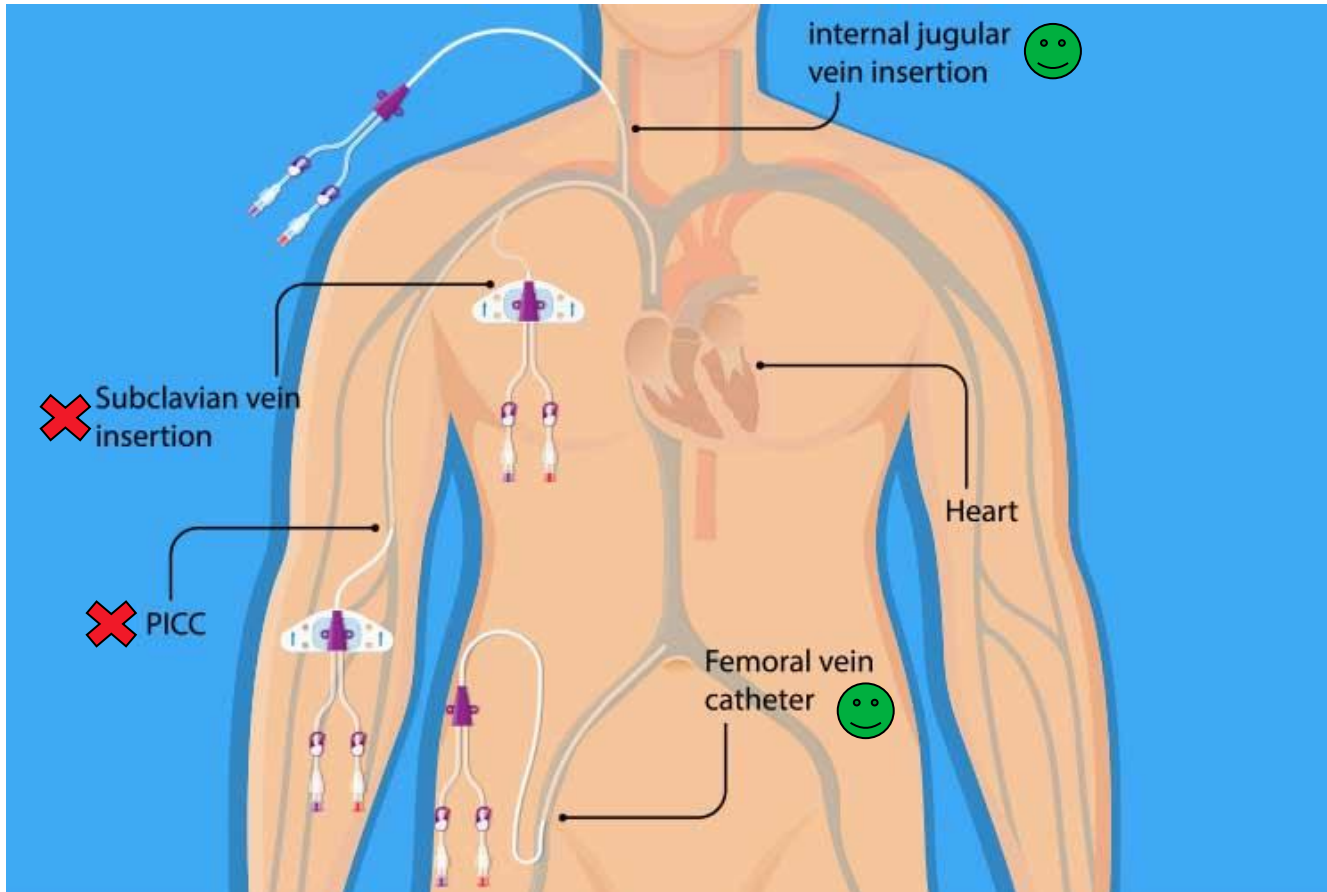
— vascular access



Source: <https://www.teleflexvascular.com/products/cd-13902>

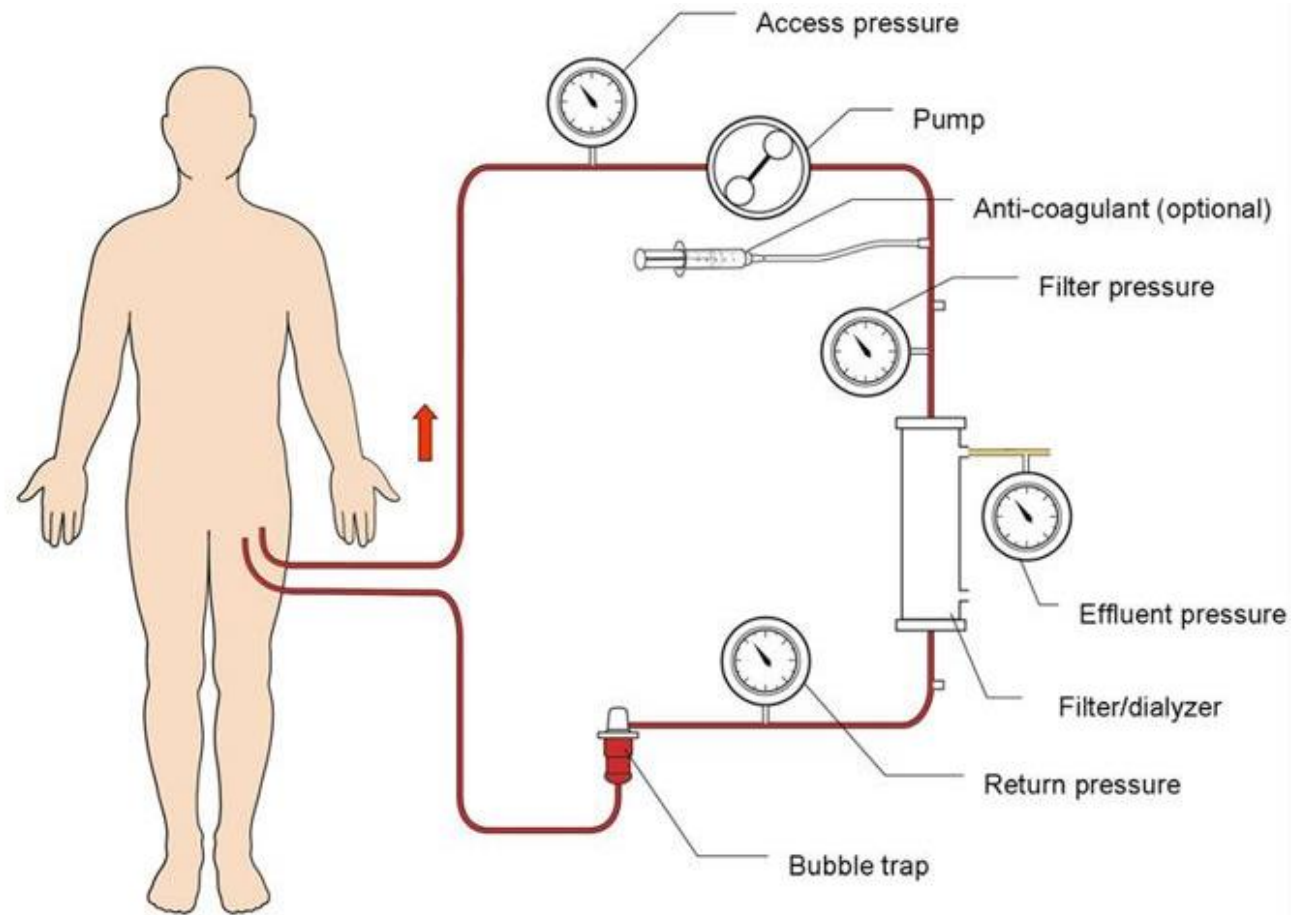


Vascular access



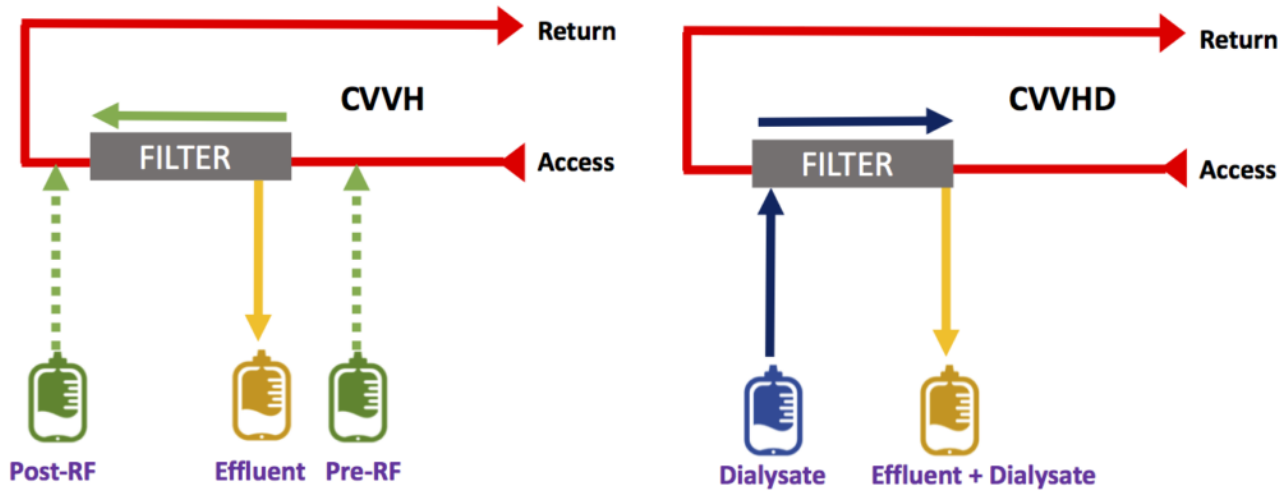
Source:
<http://www.emdocs.net/unlocking-common-ed-procedures-never-let-go-a-review-of-central-venous-access-placement/>
<https://www.ausmed.com.au/cpd/articles/-central-venous-catheters>

Design of RRT

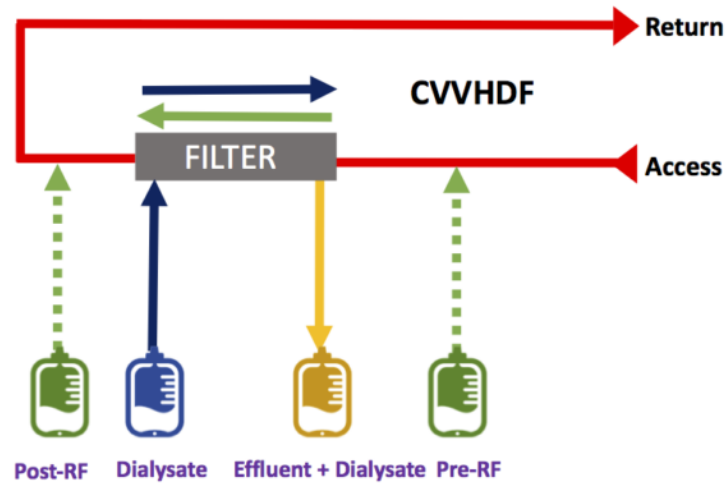


Source:
<https://derangedphysiology.com/main/required-reading/renal-failure-and-dialysis/Chapter%203.1.6/cvvhf-circuit-diagram>

Design of RRT



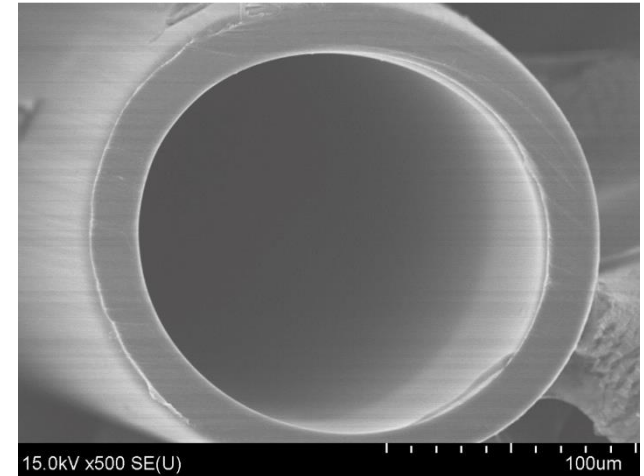
CVVH: Continuous veno-venous hemofiltration
 CVVHD: Continuous veno-venous hemodialysis
 CVVHDF: Continuous veno-venous hemodiafiltration
 Post-RF: post-dilutional replacement fluid
 Pre-RF: pre-dilutional replacement fluid



Anticoagulation in CRRT

- blood of the patient comes in contact with the circuit tubing material – exogenous material, activation of coagulation

- possibilities:
 - no anticoagulation
 - heparin
 - citrate



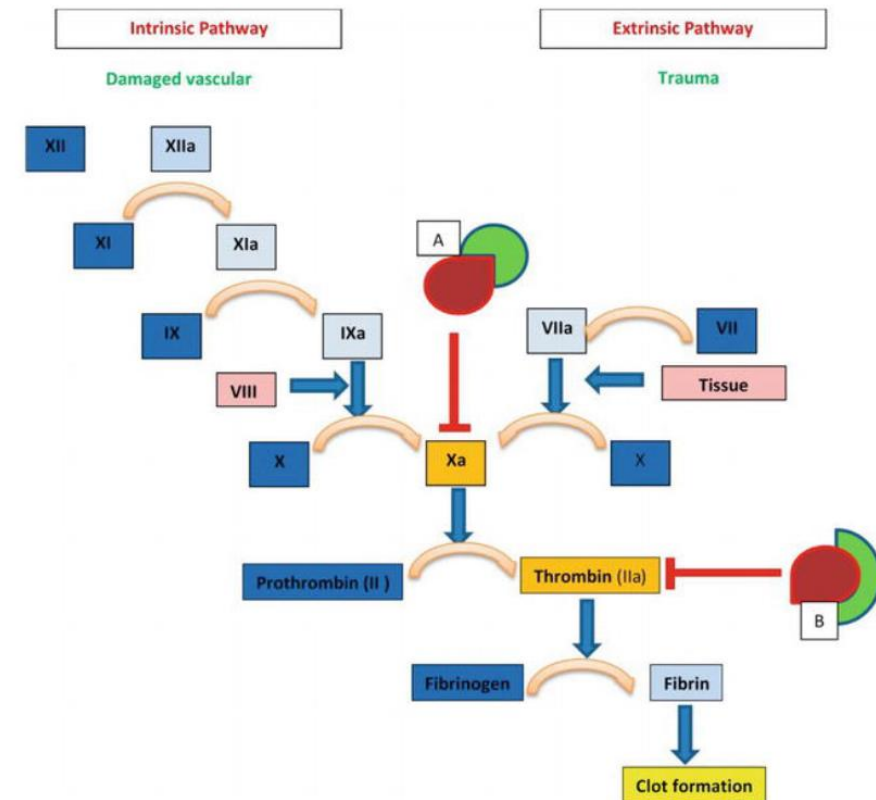
Source:

<https://www.freseniusmedicalcare.cz/cs/odborna-verejnost/akutni-terapie/filtry-pro-crrt-a-plazmaferezu/>

<https://www.intechopen.com/chapters/48020>

Anticoagulation in CRRT

- Heparin
- Advantages: used for the long time, extensive experience, well established protocols, intuitive usage, reliable function
- Disadvantages: systemic anticoagulation, needs frequent blood level monitoring, risk of bleeding, heparin-induced thrombocytopenia (HIT)



Source:

<https://www.pfizerhospitalus.com/products/heparin-sodium-injection>

<https://www.intechopen.com/chapters/68788>

Take home message

- acute renal injury/failure is relative common problem in critical care with high mortality
- accompanies most common ICU pathologies (e.g. sepsis)
- need to find out the reason of renal pathology (pre-renal, renal, post-renal)
- renal replacement therapy represents effective method of the treatment of AKI

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