Acute Renal Failure in the SICU
nephron
the functional unit of the kidney
• capable of forming urine
• has two major components:
  glomerulus
  tubule:
    proximal
    loop of Henle
    distal
    collecting
structural organization

renal parenchyma
cortex
medulla

nephrons
cortical
juxtamedullary
renal blood supply:

- the kidneys receive 20% of the cardiac output

vascular supply:

- renal arteries
- interlobar arteries
- arcuate arteries
- interlobular arteries
- afferent arterioles
- glomerular capillaries
- efferent arterioles
- peritubular capillaries
renal circulation

• there are two capillary beds arranged in series

• the efferent arteriole helps to regulate the hydrostatic pressures in both sets of capillaries
steps in urine formation

- filtration (glomerular function)
- reabsorption and secretion (tubular function)

98% of the ultrafiltrate is reabsorbed

tubular reabsorption is quantitatively more important than tubular secretion in the formation of urine, but secretion determines the amount of K+ and H+ ions that are excreted
glomerular filtration rate (GFR)

GFR depends on the interplay between hydrostatic and oncotic pressures within the nephron

- *hydrostatic pressure* is usually higher in the glomerulus than within the tubule, forcing filtrate out of the capillary bed into the tubule
- *oncotic pressure* is generated by non-filtered proteins: it helps to retain fluid in the intravascular space

- **GFR**: Kf* (hydrostatic pressure – oncotic pressure)
- **Normal GFR**: 100 ml/min/1.72m²

*Kf filtration coefficient in the glomerulus*
determinants of Glomerular Filtration Rate (GFR)

net filtration pressure:
hydrostatic + colloid osmotic pressure

Glomerular hydrostatic pressure
Glomerular colloid osmotic pressure

Bowman’s capsule pressure
Determinants of renal blood flow (RBF)

\[ RBF = \frac{\text{renal artery pressure} - \text{renal vein pressure}}{\text{total renal vasculature resistance}} \]
autoregulation

a feedback mechanism that keeps renal blood flow (RBF) and glomerular filtration rate (GFR) constant despite changes in arterial blood pressure.
autoregulation of GFR

- As renal blood flow increases, GFR increases, leading to an increase in NaCl delivery to the macula densa.

- A feedback loop through the macula densa to the juxtaglomerular cells of the afferent arteriole results in increased vascular tone, decreased renal blood flow and a decrease in GFR.

- NaCl to the macula densa then decreases leading to relaxation of the afferent arteriole (increasing glomerular hydrostatic pressure) and increases renin release from juxtaglomerular cells of afferent and efferent arterioles.

- Renin increases angiotensin I, then converted to angiotensin II which constrict efferent arteriole increasing hydrostatic pressure returning GFR to normal.
Macula densa feedback mechanism for autoregulation

afferent arteriolar resistance

arterial pressure

glomerular hydrostatic pressure

GFR

macula densa NaCl

renin

angiotensin II

afferent arteriolar resistance

Proximal tubule NaCl reabsorption

GFR
Tubular Function

- proximal tubule
  - 70% of Na is reabsorbed in the proximal tubule
Tubular Function

- loop of Henle
  - 20% of Na, Cl and K reabsorbed
  - urine concentration and dilution occurs in the loop of Henle through an osmotic gradient provided by the countercurrent mechanism (vasa recta)
  - urine flow rate is regulated by NaCl, prostaglandins, adenosine and urine volume presented to the macula densa
Tubular Function

- distal tubule
  - secretes K and bicarbonate
  - proximal segment of distal tubule is impermeable to water (urine dilution)
  - distal segment (cortical collecting tubule): K and bicarbonate secretion
Tubular Function

- collecting duct
  - regulates final urine concentration
  - aldosterone receptors regulate Na uptake and K excretion
  - ADH increases water reabsorption. In the absence of ADH, the collecting duct is impermeable to water
major sites of solute and water movement across the nephron
Clinical Assessment of Renal Function

- Acute deterioration of renal function is common in the ICU → significant contributor to Mortality and Morbidity
- Serum creatinine concentration underestimates the decrease in Glomerular Filtration rate [GFR]
- Utilizing equations to estimate renal function should be routine in the ICU
Glomerular Filtration rate [GFR]—standard measure of renal function
  ◦ Reflects overall renal functional capacity
  ◦ In renal failure, correlates with structural damage to the kidney
Renal Blood Flow

- 20% of Cardiac output [only 0.5% of mass]
- High flow rate
  - Arrangement of renal vasculature → low resistance vessels
- Major resistance vessels = afferent and efferent arterioles
- Autoregulation – constancy of blood flow & GFR over a wide range of perfusion pressures
- Renal circulation mediated by:
  - Neural, hormonal, and/or intra-renal factors
Renin–Angiotensin pathway

- Renin– [JG cells] – Released in response to
  - Decrease in renal perfusion
  - Altered sodium chloride delivery to the ascending limb and macula densa of the nephron
  - Increased renin → augmented Angiotensin 2 [A2] at local nephron level

- A2–
  - Increases renal vascular resistance by effecting both afferent and efferent arterioles [E>A]
**Renal eicosanoids**
[**protective effect on kidney function**]

- Physiologically active fatty acid products—synthesized by the kidney→ local release and effect on kidney vasculature
  - COX pathway→ produces
    - prostaglandins[PgE2, PgI2—vasodilator effect], endoperoxidases, and thromboxane A2 [vasoconstrictor]
  - Lipooxygenase pathway
    - leukotrienes
Measurement of renal blood flow

- Measured by clearance of Para- amino Hippurate [PAH]
- PAH is cleared almost totally from plasma by filtration and secretion
- PAH test used rarely—research purposes only
- Replaced by:
  - Selective angiography
  - Duplex U/S – preferred
  - External radionuclide scanning – preferred
GFR

- Major function of kidney
- 120–130 ml /min/m²
- Estimation or direct measurement of GFR – widely used in clinical practice
Measurement of GFR

- Clearance of Inulin [fructose polymer]—rarely used
- Normal values of GFR—for patients age 13–35
- After 35 years of age—GFR declines in most individuals
  - Rate of 10 ml/min/decade
Creatinine Clearance

- Useful measurement to estimate GFR
- Usually 24 hour urine collection
- Shorter time periods seem to show just as accurate results [4 hrs, 6 hrs]
- If GFR below 40 ml/min then creatinine clearance may overestimate GFR [creatinine is not just filtered but secreted into the urine–problem corrected by the use of cimetidine]
Serum Creatinine

- Creatinine
  - Formed non-enzymatically from creatine and phosphocreatine in muscle
  - Production closely related to muscle mass
  - Rise in serum creatinine may take several days after insult [time to approach steady state level]
FIGURE 126-1. Relationship between creatinine clearance and serum creatinine. In the steady state, the serum creatinine should increase twofold for each 50% reduction in creatinine clearance. Inset represents an enlarged view of the changes in serum creatinine as creatinine clearance decreases from 120 to 50 mL/min. If serum creatinine is 0.8 mg/dL when the creatinine clearance is 120 mL/min, creatinine clearance can decrease by 33% such that the increased serum creatinine is still within the normal range.
<table>
<thead>
<tr>
<th>Equation</th>
<th>Description</th>
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</table>
| **Cockcroft-Gault** $(C_F \times BSA/1.73 \, m^2)$ | For men: $C_F = \frac{[(140 - \text{age}) \times \text{weight (kg)}/S_{Cr}]}{72}$  
For women: $C_F = \frac{[(140 - \text{age}) \times \text{weight (kg)}/S_{Cr}]}{72} \times 0.85$ |
| **MDRD (1)** | $GFR = 170 \times S_{Cr}^{0.996} \times [\text{age}]^{-0.176} \times [0.762 \text{ if patient is female}] \times [1.18 \text{ if patient is black}] \times [\text{BUN}]^{-0.176} \times [\text{Alb}]^{0.318}$ |
| **MDRD (2)** | $GFR = 186 \times S_{Cr}^{-1.154} \times [\text{age}]^{-0.203} \times [0.742 \text{ if patient is female}] \times [1.212 \text{ if patient is black}]$ |
| **Jelliffe (1)** $(C_F \times BSA/1.73 \, m^2)$ | For men: $(98 - [0.8 \times (\text{age} - 20)])/S_{Cr}$  
For women: $(98 - [0.8 \times (\text{age} - 20)])S_{Cr} \times 0.90$ |
| **Jelliffe (2)** | For men: $(100/S_{Cr}) - 12$  
For women: $(80/S_{Cr}) - 7$ |
| **Mawer** | For men: weight $\times [29.3 - (0.203 \times \text{age})] \times [1 - (0.03 \times S_{Cr})]$  
For women: weight $\times [25.3 - (0.175 \times \text{age})] \times [1 - (0.03 \times S_{Cr})]$ |
| **Bjornsson** | For men: $(27 - (0.173 \times \text{age})) \times \text{weight} \times 0/S_{Cr}$  
For women: $(25 - (0.175 \times \text{age})) \times \text{weight} \times 0.07/S_{Cr}$ |
| **Gates** | For men: $(89.4 \times S_{Cr}^{-1.2}) + (55 - \text{age}) \times (0.447 \times S_{Cr}^{-1.1})$  
For women: $(89.4 \times S_{Cr}^{-1.2}) + (55 - \text{age}) \times (0.447 \times S_{Cr}^{-1.1})$ |
| **Salazar-Corcoran** | For men: $(137 - \text{age}) \times [(0.285 \times \text{weight}) + (12.1 \times \text{height}^2)]/(51 \times S_{Cr})$  
For women: $(146 - \text{age}) \times [(0.287 \times \text{weight}) + (9.74 \times \text{height}^2)]/(60 \times S_{Cr})$ |
Serum Urea Nitrogen [BUN]

- Urea production more variable than creatinine
- Related to protein intake, hypercatabolism, etc.
- Even at constant GFR– BUN may rise or fall due to other clinical issues
Sodium balance & extra-cellular fluid volume

- Fractional excretion of Sodium [FeNa]
  - Can be measured from a random sample of urine and plasma obtained simultaneously
  - In general
    - FeNa – less than 1% = Pre-renal
    - FeNa – 1–4 % = Indeterminate
    - FeNa – greater than 4 % = renal problem
    - Without the use of Diuretics
Acute Renal Failure (ARF)
acute renal failure: definition

ARF is an abrupt decline in glomerular and tubular function, resulting in the failure of the kidneys to excrete nitrogenous waste products and to maintain fluid and electrolyte homeostasis.
Azotemia is a consistent feature of acute renal failure (ARF), oliguria **is not**.

anuria ::: urine output < 0.5 ml/kg/h
acute renal failure: pathophysiology

Increase in NaCl delivered to macula densa. Damage to proximal tubule cells increases NaCl delivery to distal nephron. This causes disruption of feedback mechanism.

Obstruction of tubular lumen. Casts (necrosis of tubular cells and sloughed basement membrane) clog the lumen. This will increase the tubular pressure and then GFR will fall.

Backleak of fluid through the tubular basement membrane.
Acute Renal Failure

- Pre-renal causes
  - Pre-renal azotemia accounts for:
    - 70% of Community acquired ARF
    - 40% of Hospital acquired ARF
  - 2 issues
    - Decreased blood volume [vomiting, bleeding, dehydration]
    - Reduced arterial blood volume [CHF, Cirrhosis]
  - No cellular injury → reversible with correction of cause
  - Bland urine sediment & FeNa less than 1%
Lack of Urine output in the acutely hypovolemic patient is “renal success” not “renal failure”
ARF–Post Renal causes

- Occurs with obstruction to urinary flow
- Uncommon cause of ARF in the ICU
  - Most easily correctable
- Evaluation =
  - Renal U/S
  - Post void residual check [normal less than 50 ml]
ARF—Intra-renal causes

• Defined according to anatomic location of injury [glomerulus, tubule, interstitium, or vasculature]

• In ICU—Acute Tubular Necrosis [ATN]—most common form of ARF [includes both tubular and vascular injury]

• DDX: Pre-renal azotemia
  – Examine sediment [brown casts → ATN]
  – Check FeNa [greater than 1 %]
Epidemiology

- ARF—Common complication [Up to 25% of ICU patients]
- Multi-factorial etiology
  - HTN
  - Sepsis
  - Drugs
- High mortality—up to 80% 
- Risk of developing ARF increases with
  - Age, Presence of baseline Chronic Kidney Disease, Oliguria, Sepsis
ARF– Definition

• Qualitatively– abrupt reduction in GFR
• Clinically– defined in terms of small solute clearance
  – BUN/ Creatinine:
    • Most common parameters measured
    • Not sensitive indicators of renal dysfunction
• No Standard Defn of ARF
  – 50 % increase in serum creatinine
  – S.creatinine greater than 2 mg/dl
• Measurement of injury markers
Renal Failure

- Diseases effecting primarily
  - Glomerulus (glomerulo-nephritis)
  - Interstitium (Interstitial nephritis)
  - Blood vessels (Vasculitis)
  - Tubules (ATN)—Most common hosp. acquired ARF.
Acute Tubular Necrosis

- Toxic
- Ischemic
Toxic ATN

- **Drugs**
  - **Aminoglycosides**
    - Risk factors (Age/Hypovolemia/Hypokalemia/Other nephrotoxic drugs/Short dosing interval.)
    - Routine use of Peak & Trough does not reduce likelihood of ATN.
    - Dose adjusted by creatinine clearance. (Body wt. [kg]/Serum creatinine.)
Radiographic contrast agents

- Risk factors (Pre-existing renal insufficiency/Diabetes/Poor LV function/Multiple studies in 24x hours/Volume of contrast used greater than 1.5 cc/kg).
- Non-ionic contrast may be less toxic.
- Volume expanding with crystalloid solutions (500–1000)---best prophylaxis.
- Nephrotoxicity not lessened by (Mannitol/Lasix/Ca\(^{++}\) channel blockers).
- Theophylline may help.
Rhabdomyolysis

- Drugs (Heroin, Cocaine, Lovastatin)
- Crush injury
- Alcohol
- Seizures
- Risk factors (Decreased ECV, Shock)
acute renal failure: prevention

- recognize patients at risk (postoperative states, cardiac surgery, septic shock)

- prevent progression from prerenal to renal

- preserve renal perfusion
  - isovolemia, cardiac output, normal blood pressure
  - avoid nephrotoxins (aminoglycosides, NSAIDS, amphotericin)
hemoglobinuria + myoglobinuria

hemoglobinuria:
transfusion reactions, HUS, ECMO

myoglobinuria:
crush injuries, rhabdomyolysis
urine (+) blood but (-) red blood cells
↑ CPK ↑ K+

treatment
aggressive hydration + urine alkalinization
mannitol / furosemide
acute renal failure: management

- treat the underlying disease
- strictly monitor intake and output (weight, urine output, insensible losses, IVF)
- monitor serum electrolytes
- adjust medication dosages according to GFR
- avoid highly nephrotoxic drugs
- attempt to convert oliguric to non-oliguric renal failure (furosemide x 3)
**acute renal failure: fluid therapy**

If patient is fluid overloaded
- fluid restriction (insensible losses)
- attempt furosemide 1–2 mg/kg
- Renal replacement therapy (see later)

If patient is dehydrated:
- restore intravascular volume first
- then treat as euvolemic (below)

If patient is euvolemic:
- restrict to insensible losses (30–35 ml/100kcal/24 hours) + other losses (urine, chest tubes, etc)
Most patients have dilutional hyponatremia which should be treated with fluid restriction.

Severe hyponatremia (Na< 125 mEq/L) or hypernatremia (Na> 150 mEq/L): dialysis or hemofiltration.
Oliguric renal failure is often complicated by hyperkalemia, increasing the risk in cardiac arrhythmias.

**Treatment of hyperkalemia:**

- Sodium bicarbonate (1-2 mEq/kg)
- Insulin + hypertonic dextrose: 1 unit of insulin/4 g glucose
- Sodium polystyrene (Kayexalate): 1 gm/kg. Can be repeated qh. (Hypernatremia and hypertension are potential complications)
- Dialysis
For the prevention of contrast induced ARF in patients at risk:
- Hydration with NS – most beneficial
- Role of N-Acetyl Cystiene – undetermined

No role for Dopamine in ARF

Diuretics may be used in the initial mgmt of ARF → If no response → D/C and Start RRT
Early goal directed management—may reverse adverse hemodynamics → Before tissue injury → Better outcome
Recognition of Pseudo-ARDS & mgmt with U/F → Improved outcome
Optimal fluid resuscitation → in general crystalloids – consider colloids in Capillary leak syndromes [sepsis]
When vasopressors indicated → systemic hemodynamic priorities
In sepsis—consider vasopressin [splanchnic vasoconstiction]
Nutritional Support

- ARF → increased Protein catabolism due to insulin resistance [Kwashiorkor]
- Enteral nutrition is recommended
- Caloric supplementation [non–protein calories] 25–30 kcal/kg/d
- PROTEIN RESTRICTION HAS NO ROLE IN MANAGEMENT OF ARF
Nephrology Consultation

- Early consultation may lead to earlier recognition of ARF → earlier institution of RRT → better outcomes
Renal Replacement Therapy [RRT]

- Likely that early initiation of RRT [BUN less than 60 mg/dl] is beneficial
- Indications for RRT
  - Volume overload
  - Hyperkalemia
  - Acidosis
  - Pericarditis
- In moderately severe illness, increased dose of RRT→ improved outcome
  - With CVVH→ U/F rates approaching 35 ml/kg should be attained
  - With intermittent HD, daily dialysis should be initiated for catabolic patients
Modality – RRT

- Delayed recovery from ATN → fresh areas of necrosis within kidney → exacerbated by Dialysis Associated Hypotension
- CRRT provides better CV stability than Intermittent HD
- CRRT allows for superior metabolic control in catabolic patients & improved fluid management in pts receiving TPN / Blood products
- CRRT NOT associated with Improved Survival
- Drawbacks of CRRT = increased nursing care & need for anti-coagulation
Dialysis membranes

- Interaction between blood & dialysis membrane → inflammatory response
- Biocompatible membranes → slight improvement in outcomes [not all studies]
- Ability to bind cytokines → may improve outcomes with synthetic membranes
Lactate buffer is associated with hyperlactatemia in pts with Hypotension or Liver dysfunction ➞ elevated serum lactate ➞ contributes to protein catabolism

Lactate buffer associated with decreased hemodynamic stability

Bicarbonate buffer is now Standard
Medication dosing

- In critical illness—both volume of distribution and extent of protein binding of drugs changes
- Consider degree of renal dysfunction when determining medication dosing ➔ potential toxicities
## Laboratory & Microscopic findings in Pre-renal azotemia & ATN

<table>
<thead>
<tr>
<th>Lab Test</th>
<th>Pre-Renal Azotemia</th>
<th>ATN</th>
</tr>
</thead>
<tbody>
<tr>
<td>Urine Osmolality</td>
<td>Greater than 500</td>
<td>Less than 400</td>
</tr>
<tr>
<td>Urine Na</td>
<td>Less than 20</td>
<td>Greater than 40</td>
</tr>
<tr>
<td>FeNa</td>
<td>Less than 1 %</td>
<td>Greater than 2 %</td>
</tr>
<tr>
<td>Urine sediment</td>
<td>Normal, Occasional hyaline casts</td>
<td>Renal tubular epithelial cells, Granular and muddy brown casts</td>
</tr>
</tbody>
</table>
Risk factors for developing ARF

- Age greater than 65
- Infection on admission
- CV failure
- Cirrhosis
- Respiratory failure
- Chronic Heart Failure
- Lymphoma, Leukemia
Risk factors for Mortality in ARF

- High severity of illness index score
- Age greater than 65 years
- Male gender
- Oliguric acute renal failure
- CV failure
- Mechanical ventilation
- Prior decreased health status
Risk factors for Contrast Induced Nephropathy

- Pre-existing renal impairment
- DM
- Decrease in effective arterial volume [CHF, Cirrhosis, Volume depletion]
- High dose contrast media
- Concurrent use of nephrotoxic drugs [NSAIDs, ACE inhibitors]
Preventive strategies

- Especially in pts with significant CKD
  - eGFR less than 60 ml/min/m²
  - Other evidence of renal disease— (Proteinuria, etc)
  - If eGFR less than 30→ CIN rates 30–40 % & ARF requiring dialysis→ 2–8 %
Preventive strategies

4 basic concepts
- Hydration
- Choice and quality of contrast
- Pre, intra, and post procedure end-organ protection with drugs
- Post procedure monitoring and expectant care
Hydration

- Volume supplementation with NS or sodium bicarbonate solution starting at least 3–12 hours before procedure:
  - 1–2 ml/Kg/hr
  - 100–150 ml/hr
  - Minimum 300 ml to 500 ml pre-procedure
  - If concerned about CHF place SG catheter

- After procedure target urine output = 150 ml/hr:
  - If greater consider volume replacement
  - Usually 150 ml/hr for at least 6 hours after procedure
Ionic strength of contrast

- Lower ionic strength → less CIN
- Limit volume of contrast to less than 100ml if possible
- If staged procedures → consider 10 day interval
Lessons learned

- Diuretics (Loop and mannitol) can worsen CIN in the absence of volume replacement
- Renal protection with renal dose dopamine or Fenoldopam cannot be achieved routinely
- Renal toxic agents should be discontinued in the peri-procedural period (72 hours at least):
  - NSAIDs
  - Aminoglycosides
  - Cyclosporine
- **There are NO approved agents for the prevention of CIN**
Currently accepted approaches

- Optimal hydration (saline or bicarbonate)
- Iodixanol (low ionic contrast agent) with minimizing volume
- Oral or IV N-acetyl cysteine [NAC]—cyto-protective agent against oxidative injury
  - Standard regimen—NAC 600 mg PO BID for the day before and the day of the contrast study
  - Emergent procedures: NAC 1 gram PO 1 hour before and 4 hours after procedure
NAC dosing

- IV NAC can also be given
- Usually given in bolus before procedure and up to 4 hours after procedure
- Dose dependant protective effect
- Evidence based medicine:
  - Meta-analysis → Failed to show benefit of PO NAC in preventing CIN in all patients
  - May have benefit in patients with CKD
Other Agents/Approaches tried

- Animophylline
- Endothelin receptor antagonists
- Vitamin C
- Peri-procedural hemofiltration
- No benefit proven
TOP 10 check list for CIN risk stratification

- Calculate e GFR—high risk if less than 60
- Check DM status → 5 fold higher risk if +
- Discuss CIN in informed consent process
- Stop renal toxic drugs (NSAIDs, etc)
- Nephrology consult if e GFR < 15ml/min for dialysis plan after procedure
- Hold diuretics day before and day of procedure & Hydrate patient
TOP 10 check list for CIN risk stratification

- Ensure urine flow rate > 150 ml/hr after procedure
- Iodixanol – preferred contrast agent for high risk patients
- Limit contrast volume to less than 100 ml
- NAC 600 mg PO BID day before and day of procedure
Potential indications for RRT in ICU

- Non-obstructive oliguria [u/o less than 200 ml /12 hours] or anuric
- Severe acidemia
- Azotemia [BUN greater than 80 mg/dl]
- Hyperkalemia [K greater than 6.5 mmol/l]
- Uremia [encephalopathy, pericarditis, neuropathy, myopathy]
- Severe dysnatremia [Na greater than 160 mmol/l or less than 115 mmol/l]
Potential indications for RRT in ICU

- Hyperthermia [temp greater than 39.5 C]
- Clinically significant organ edema [especially lung]
- Drug overdose with dialyzable toxin
- Coagulopathy requiring large amount of blood products
Principles of RRT

- 2 fundamental principles
  - Water removal
  - Solute removal
Water removal

- Ultrafiltration
- Driving pressure
- Trans-membrane pressure greater than oncotic pressure
- Increasing osmolality of the dialysate with osmotic agents
Solute removal

- Create an electrochemical gradient across the membrane
- Diffusion—movement of a solute to reach the same concentration on each side of the membrane
Hemodialysis

Blood $\rightarrow$ Dialysate $\rightarrow$ Blood
Hemofiltration

Blood → Replacement fluid → Blood

Ultrafiltrate
Hemodiafiltration

Blood → Replacement fluid

Blood

Dialysate + Ultrafiltrate

Dialysate
Indications for RRT [acute renal failure]

- Different style and philosophy from RRT for chronic renal failure
- Critically Ill Patient
  - Initiate early
  - CRRT may also be justified—risks of HD are not relevant
Modern Criteria for the initiation of RRT in the ICU

- Oliguria [U/O less than 200 ml /24 hrs]
- Anuria [U/O 0–50 ml/12 hours]
- Serum Urea greater than 35 mmole/l
- Serum Creatinine greater than 400 micromoles/l
- K greater than 6.5 mmol/l or rapidly rising
- Pulmonary edema unresponsive to diuretics
- Uncompensated metabolic acidosis [pH<7.1]
- Na < 110 mmol/l or > 160 mmol/l
- Temp > 40 C
- Uremic complications–encephalopathy,etc
- Overdose with dialyzable toxin [eg. Lithium]
RRT

- Better uremic control = better survival
- Target Urea levels = 10–20 mmol/l
CRRT Vs HD

- No difference in survival
- Are we looking at the wrong outcome measure?
- CRRT hemodynamically less destabilizing
- More labor intensive
- Understanding of flow dynamics through filter is very important
- Anticoagulation essential to cost management
Conclusions

- No strict definition to ARF in ICU
- Prevention is better than treatment
- Understand pathophysiology of ATN –
  - Ischemic or nephrotoxic
- Watch the metabolic profile
- Attention to detail saves lives