

ACUTE RENAL FAILURE AND RENAL REPLACEMENT THERAPY

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OBJECTVES OF LEARNING

- RECOGNITION OF DEFINITION OF ARF
- RECOGNITION OF CAUSE OF ARF
- RECOGNITION OF PATHOGENESIS OF ARF
- RECOGNITION OF EVALUATION OF ARF
- RECOGNITION OF MANAGEMENT OF ARF
- RECOGNITION OF INDICATION OF CRRT
- RECOGNITION OF PRINCIPLES OF CRRT
- RECOGNITION OF ADVANTAGES OF CRRT

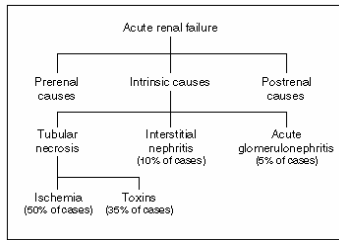
ACUTE RENAL FAILURE

- Defined as a **sudden** and **sustained** loss of renal function (over several hours to several days), ARF results in derangements in extracellular fluid balance, acid base, electrolytes, and divalent cation regulation.

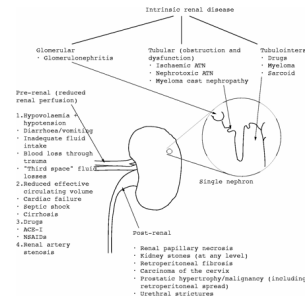
ACUTE RENAL FAILURE

- An increased serum creatinine concentration, accumulation of other nitrogenous-based waste products, and often a decline in urinary output are the hallmarks of ARF.

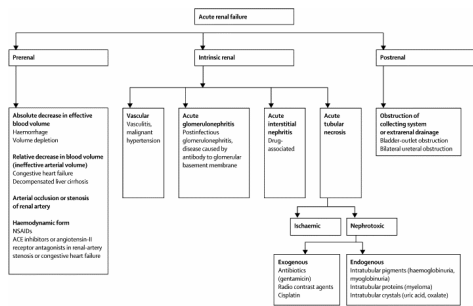
ACUTE RENAL FAILURE



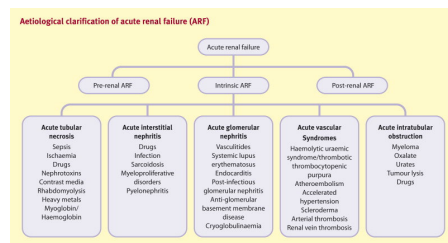
CAUSES OF ARF



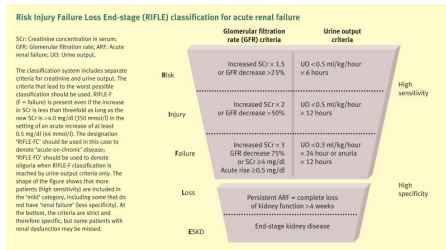
CLASSIFICATION OF ARF



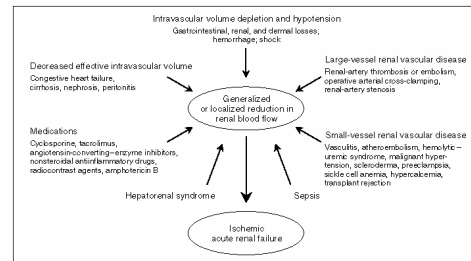
ETIOLOGICAL CLASSIFICATION OF ARF



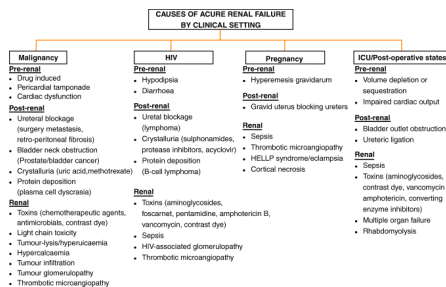
RIFLE CRITERIA CLASSIFICATION OF ARF



CONDITIONS CAUSE THE ISCHEMIC ARF



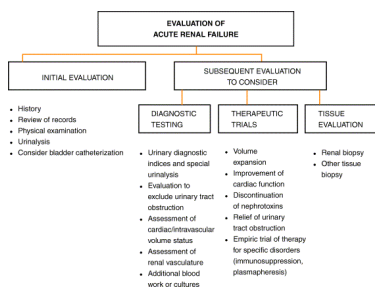
CAUSES OF ARF BY CLINICAL SETTING



SEPSIS AND KIDNEY

- Sepsis is one of the most common causes of ARF in critically ill patient
- ARF is a marker on the road to the loss of life

EVALUATION OF ACUTE RENAL FAILURE



EVALUATION OF ARF

- **INTRAVASCULAR VOLUME STATUS IS THE MOST IMPORTANT FACTOR IN THE EVALUATION OF ARF.**
- **A DECREASE IN URINARY VOLUME IS OFTEN ONE OF THE INITIAL CLINICAL FINDING IN ARF.**

EVALUATION OF ARF

- The most important laboratory test for a patient with ARF is **urinalysis**.
- Both **urinary sediment** and **urinary indices** in combination with **serum values** can often be extremely helpful in determining the **cause** of ARF.

GUIDELINE OF URINARY INDICES

Table 1
Guidelines for urinary indices whereby established ARF can be distinguished from renal vasoconstriction with intact tubular function (prerenal azotemia)

Laboratory test	Prerenal azotemia	ARF
Urine osmolality (mOsm/kg)	>500	<400
Urine sodium level (mEq/l)	<20	>40
Urine/plasma creatinine ratio	>40	<20
Fractional excretion of sodium (%)	<1	>2
Fractional excretion of urea (%)	<35	>35
Urinary sediment	Normal; occasional hyaline or fine granular casts	Renal tubular epithelial cells; granular and muddy brown casts

Osm, osmole; Eq, equivalent.

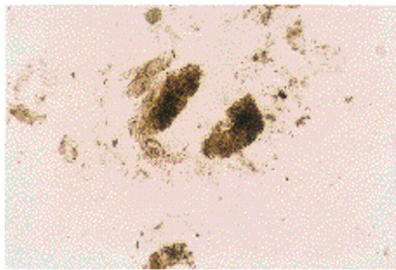
CAUSES OF ABNORMAL BUN/CREATININE RATIO

BUN:Cr > 15
 Increased formation of urea
 High intake of protein
 Catabolic states
 Fever
 Diuretic therapy
 Corticosteroid use
 Tetraacyclines
 Squirts
 Decreased elimination of urea
 Volume loss
 Decreased cardiac output
 Obstructive uropathy
BUN:Cr < 15
 Decreased formation of urea
 Starvation
 Advanced liver disease
 Hereditary deficiency of urea cycle enzymes
 Relative increased removal of urea
 Post-dialysis
 Increased formation of creatinine
 Rhabdomyolysis
 Decreased secretion of creatinine
 Creatinins
 Trimethoprim
 Pyrazinamide
 Interference with assay
 Ketones
 Ceftriaxone
 Ascorbic acid
 Nifedipine
 Fluocytosine
 Barbiturates

URINALYSIS IN ARF

Normal
 Post-renal
 Pre-renal
 High plasma ureotic pressure
Abnormal
RBC, WBC, casts, proteinuria
 Glomerulonephritis
 Vasculitis
 Thrombotic microangiopathy
WBC, WBC casts
 Pyelonephritis
 Interstitial nephritis
 Escherichia
 Allergic interstitial nephritis
ATN
 Acute tubular necrosis
 Pigmented casts, renal tubular epithelial cells
ATN
 Myoglobinuria
 Hemoglobinuria
 Crystalluria
 Urine acid
 Drug/toxins
 Non-oliguric prerenaluria
 Phagocytic dyscrasia

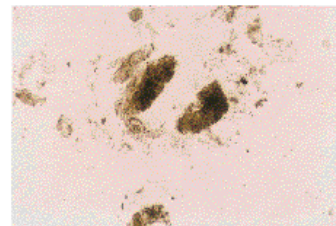
Photomicrograph of Urinary Sediment Obtained from a Patient with Acute Tubular Necrosis (x200)



Klahr S and Miller S. N Engl J Med 1998;338:671-675



URINARY SEDIMENT OF ATN



Typical Urine Findings in Conditions That Cause Acute Renal Failure

Table 2. Typical Urine Findings in Conditions That Cause Acute Renal Failure.

Condition	Diagnosis Test	Suggested Analysis	Urine	Fractional
			Osmo-	Excretion
			OSMOLALITY	OF SODIUM
			mOsm/kg	%
Prerenal azotemia	Trace or no proteinuria	A few hyaline casts possible	>500	<1
Renal azotemia				
Tubular injury				
Ischemia	Mild-to-moderate proteinuria	Pigmented granular casts	<350	>1
Nephrotoxicity*	Mild-to-moderate proteinuria	Pigmented granular casts	<350	>1
Acute interstitial nephritis	Mild-to-moderate proteinuria; hemoglobin, leukocytes	White cells and white-cell casts; eosinophils and eosinophil casts; m/d cells	<350	>1
Acute glomerulonephritis†	Moderate-to-severe proteinuria; hemoglobin, leukocytes	Red cells and red-cell casts; red cells can be dysmorphic	>500	<1
Postrenal azotemia‡	Trace or no proteinuria; can have hemoglobin, leukocytes	Crystals, red cells, and white cells possible	<350	>1

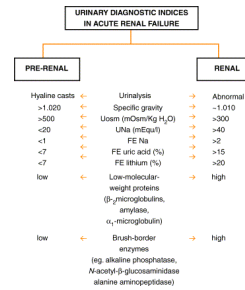
*In some conditions that lead to nonoliguric acute renal failure (e.g., response to radiopaque agents and rhabdomyolysis), the initial fractional excretion of sodium can be <1 percent.

†When glomerulonephritis (e.g., post-streptococcal glomerulonephritis) is associated with tubulointerstitial abnormalities, the urine osmolality is <350 mOsm per kilogram and the fractional excretion of sodium is >1 percent.

‡Even in the course of obstruction, before tubular damage has occurred, the fractional excretion of sodium can be <1 percent.



URINARY DIAGNOSTIC INDICES IN ARF



SIGN OF ECF DEPLETION

- TACHYCARDIA
- ORTHOSTATIC BLOOD PRESSURE CHANGES
- DRYNESS OF MUCOCUTANEOUS MEMBRANE
- HYPOTENSION
- OLIGURIA (< 400mL/D.)
- ANURIA (< 50mL/D.)

PRERENAL CAUSES OF ARF

- > 60% of cases of community acquired ARF are due to **prerenal** conditions.
- These disorders are caused by excessive, nonreplaced fluid deficits due to GI, renal, or cutaneous losses. (**hypovolemia**)

PRERENAL CAUSES OF ARF

- Hospital-acquired prerenal ARF is primarily due to **decreased effective renal perfusion**.
- Often, **CHF, cirrhosis, or sepsis** is noted.
- A continuum exists between prerenal renal failure and ischemic acute tubular necrosis (ATN).

CAUSES OF LOW PERFUSION STATES

Table 2. Causes of Low-Perfusion States.
Hypovolemic causes
Fluid loss to the third space
Tissue damage (e.g., pancreatitis)
Hypoalbuminemia (e.g., the nephrotic syndrome)
Bowel obstruction
Blood loss
Fluid loss to the outside
Gastrointestinal causes
Renal causes (e.g., diuretics, adrenal insufficiency, hypercalcemia)
Dermal causes (e.g., burns, sweating)
Cardiovascular causes (congestive heart failure)
Myocardial causes (e.g., infarction, cardiomyopathy)
Pericardial causes (e.g., tamponade)
Pulmonary vascular causes (e.g., embolism)
Arrhythmia
Valvular disease
Distributive causes (reduced vascular resistance)
Sepsis
Hepatorenal syndrome
Overdose of drugs (e.g., barbiturates)
Vasodilators (e.g., nitrates, antihypertensive agents)
Local renal hypoperfusion
Renal artery stenosis (atherosclerosis or fibromuscular hyperplasia)
Malignant hypertension

FACTORS INCREASING SUSCEPTIBILITY TO RENAL HYPOPERFUSION

Table 1. Factors Increasing Susceptibility to Renal Hypoperfusion.
Failure to decrease arteriolar resistance
Structural changes in renal arterioles and small arteries
Old age
Atherosclerosis
Chronic hypertension
Chronic kidney disease
Malignant or accelerated hypertension
Reduction in vasodilatory prostaglandins
Nonsteroidal antiinflammatory drugs
Cyclooxygenase-2 inhibitors
Afferent glomerular arteriolar vasoconstriction
Sepsis
Hypercalcemia
Hepatorenal syndrome
Cyclosporine or tacrolimus
Radiocontrast agents
Failure to increase efferent arteriolar resistance
Angiotensin-converting-enzyme inhibitors
Angiotensin-receptor blockers
Renal-artery stenosis

PRERENAL CAUSES OF ARF

- OF IMPORTANCE, MANY **MEDICATIONS** INDUCE A FUNCTIONAL PRERENAL STATE BECAUSE OF ALTERATIONS IN **RENAL PERFUSION AND GLOMERULAR HEMODYNAMICS**.

MEDICATIONS CAUSE A FUNCTIONAL DECREASE IN RENAL PERFUSION

- NSAIDs
- ACE inhibitor
- Cyclosporine
- Cocaine

GLOMERULAR PRESSURE AUTOREGULATION

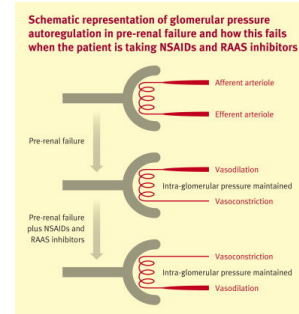
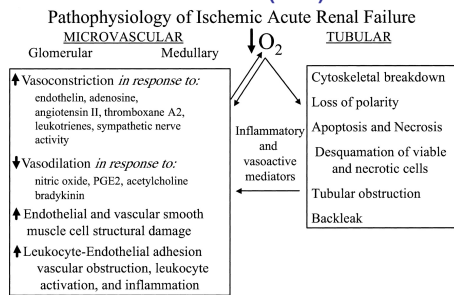


Figure 1. Interacting microvascular and tubular events contributing to the pathophysiology of ischemic acute renal failure (ARF)



Bonventre, J. V. et al. J Am Soc Nephrol 2003;14:2199-2210

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JASN

PREDISPOSING FACTORS FOR NSAID-INDUCED ARF

- CHF
- CIRRHOSIS OF LIVER
- NEPHROTIC SYNDROME
- CHRONIC RENAL FAILURE
- ATHEROSCLEROTIC DISEASE OF RENAL ARTERY
- HYPOVOLEMIA

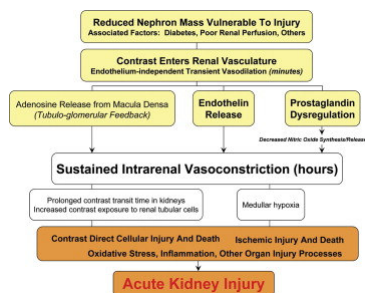
ACE inhibitor associated ARF

- ACUTE RENAL FAILURE ASSOCIATED WITH ACE INHIBITORS SHOULD PROMPT INVESTIGATION TO RULE OUT **BILATERAL RENAL ARTERY STENOSIS OR SEVERE RENAL ARTERY STENOSIS** IN A SOLITARY FUNCTIONING KIDNEY.

CONTRAST- INDUCED NEPHROPATHY

- Contrast-induced nephropathy causes ARF predominantly via **acute vasoconstriction**.

Pathophysiology of contrast-induced acute kidney injury



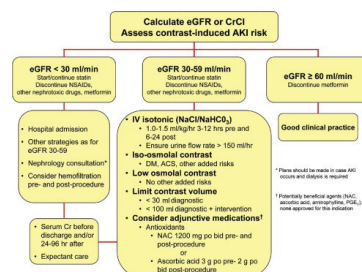
RISK FACTORS FOR CONTRAST-INDUCED NEPHROPATHY

- GFR < 35mL/min.
- Diabetic nephropathy
- Severe CHF
- Administration of large amount of contrast
- Preexisting hypokalemia or hypotension

PREVENTION OF CONTRAST-INDUCED NEPHROPATHY

- Administration of **crystalloid** (1-1.5mL/Kg/hr for 8-12 hours before a procedure) remains the safest, most efficacious, and cost-effective method of preventing contrast-induced nephropathy.
- Administration of **acetylcysteine** in two 600mg doses the day along with saline infusion for patients with stable chronic renal insufficiency ($Cr > 2.0$ mg/dL)

Algorithm for management of contrast-induced nephropathy



Renal causes of ARF

- The **most common cause** of ARF in hospitalized patient is **intrinsic renal failure** due to **ATN**.
- Four anatomic compartment of Intrinsic ARF: vasculature, interstitium, glomerulus, tubule

Renal causes of ARF

- Polypharmacy** is common among most patients with chronic diseases and hospitalized patients.
- Allergic interstitial nephritis** should be strongly considered in all patients with ARF.

MEDICATION CAUSES OF ALLERGIC INTERSTITIAL NEPHRITIS

- Penicillines
- Cephazsporines
- Sulfonamides
- Rifampin
- Ciprofloxacin
- NSAIDs
- Thiazide diuretics
- Loop diuretics
- Cimetidine
- Phenytoin
- Allopurinol
- Chinese herb

Drugs Associated with Acute Renal Failure

Table 1. Drugs Associated with Acute Renal Failure.

Mechanism	Drugs
Reduction in renal perfusion through alteration of intrarenal hemodynamics	NSAIDs, angiotensin-converting-enzyme inhibitors, cyclosporins, tacrolimus, re- ducant agents, amphotericin B, su- bcutaneous*
Direct tubular toxicity	Aminoglycoside antibiotics, radioccontrast agents, cisplatin, cyclosporins, tacro- limus, amphotericin B, methotrexate, foscarnet, pentamidine, organic sol- vents, heavy metals, intravenous im- mune globulin†
Heme pigment-induced tubular toxicity (thaldomycin)	Cocaine, ethanol, levamisole
Intravascular obstruction by pre- cipitation of the agent or its metabolites or by products	Acyclovir, sulfonamides, ethylene gly- col, chemotherapeutic agents, metho- trexate
Allergic interstitial nephritis‡	Penicillins, cephalosporins, sulfonamides, rifampin, ciprofloxacin, NSAIDs, thia- zide diuretics, furosemide, cimetidine, phenytoin, allopurinol
Hemolytic-uremic syndrome	Cyclosporins, tacrolimus, mitomycin, co- caine, quinine, conjugated estrogen

*Subcutaneous produces a capillary leak syndrome with volume contraction.
 †The mechanism of this agent is unclear but may be due to additives.
 ‡Acute renal failure is most likely to occur when levamisole is given in combination with cyclosporins.
 §Ethylene glycol-induced toxicity can cause calcium oxalate crystals.
 ¶The acid crystals form as a result of heavy metal.
 ¶Many other drugs in addition to the ones listed can cause renal failure by this mechanism.



DRUG AND TOXIN ASSOCIATED ARF

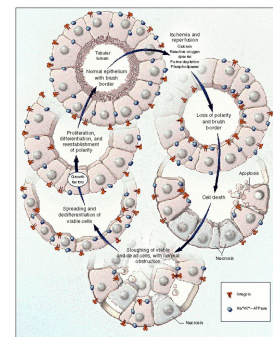
Increased renal perfusion
 NSAIDs, ACE inhibitors, sartans, nifedipine, angiotensin II, cyclosporin, tacrolimus

Direct tubular injury
 Aminoglycosides, contrast media, amphotericin B, methotrexate, cisplatin, foscarnet, pentamidine, heavy metals, nifedipine, tacrolimus, intravenous immune globulin, HIV protease inhibitors

Interstitial edema
 Contrast media, methotrexate, acyclovir, nephrotoxic drugs, uric acid, cocaine, levamisole

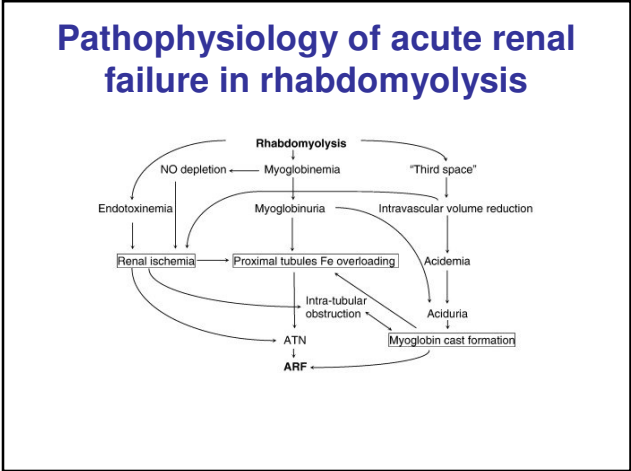
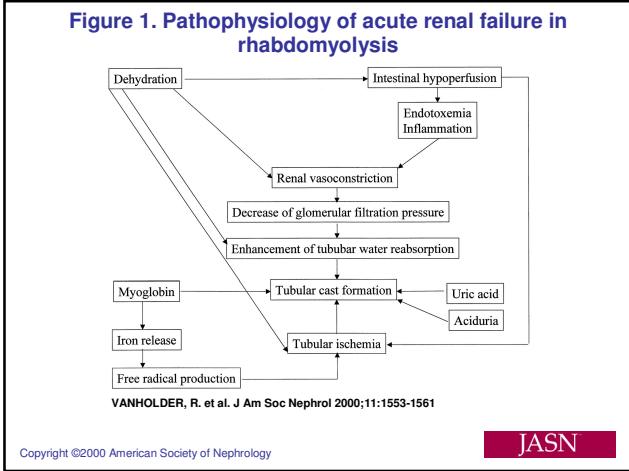
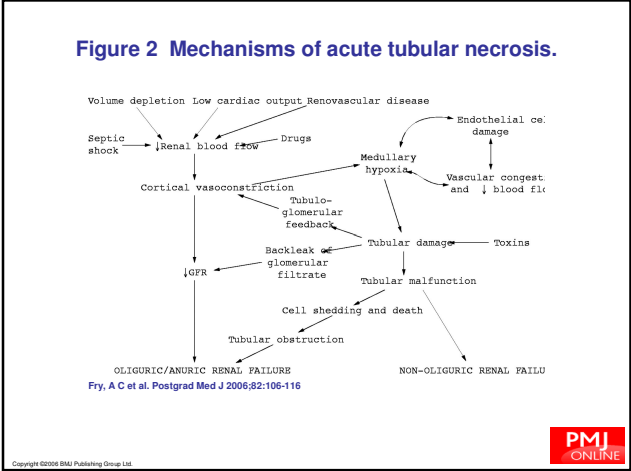
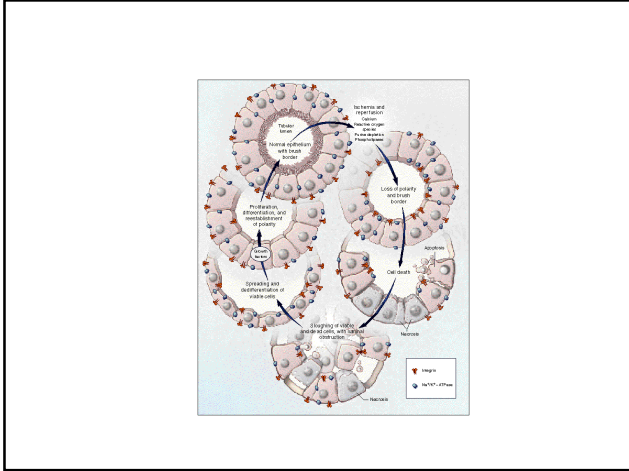
Immunological-inflammatory
 Penicillins, cephalosporins, allopurinol, NSAIDs, sulfonamides, diuretics, rifampin, ciprofloxacin, cimetidine, trimoprim, phenytoin

Tubular-Cell Injury and Repair in Ischemic Acute Renal Failure



Thadhani R et al. N Engl J Med 1996;334:1448-1460





DIFFERENTIAL DIAGNOSIS OF ACUTE RENAL FAILURE

Pre renal (40-50%)
 Volume loss or maldistribution
 Decreased cardiac output
 Hypotension
 Renal (5-15%)
Intrarenal
 Cystitis
 Prostatitis
 Interstitial
 Pyelitis
 Ureteric
 Obstruction
 Nephritis
Renal (10-30%)
 Vascular disorders:
 Small vessel
 Large vessel
 Glomerulonephritis
 Interstitial disorders
 Tubulonephrosis
 Spinal-cord-injury process
 Tubular necrosis
 Ischaemia
 Toxins
 Pigmenturia

Therapy of ARF

- The **goal** of any focused evaluation of ARF is **immediate correction** of its **reversible causes**.
- **Recognition and relief** of urinary outlet obstruction should be given the **highest priority**, especially for patient with **anuria**.

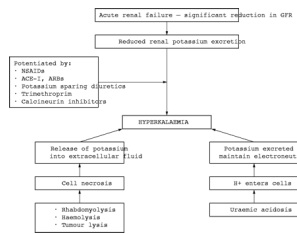
Therapy of ARF

- Support of **renal perfusion** with either volume infusion or therapeutics that improve renal oxygen delivery should be considered before any attempt to improve urinary flow.
- **Urinary indices** should be examined before diuretic intervention.

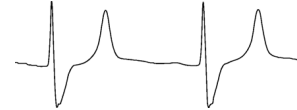
INDICATION OF RENAL REPLACEMENT THERAPY

- Anuria or oliguria (urine output <200 ml/12 hours)
- Hyperkalaemia (K^+ >6.5 mmol/l)
- Severe acidaemia (pH >7.1)
- Azotaemia (urea >30 mmol/l)
- Clinically significant organ oedema (particularly lung)
- Uraemic encephalopathy
- Uraemic pericarditis
- Uraemic neuropathy/myopathy
- Severe dysnatraemia ($[Na^+]$ >160 or <115 mmol/l)
- Hyperthermia
- Drug overdose with a dialysable product

MECHANISM OF HYPERKALEMIA IN ARF



EKG OF HYPERKALEMIA



CRRT USED ON ICU

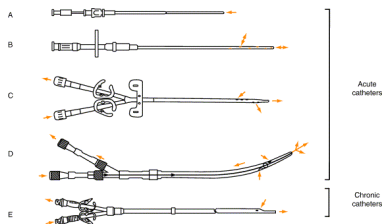
Continuous renal replacement therapies used on ICUs

Mode of therapy	Principal method of solute clearance
Continuous veno-venous haemofiltration (CVVH)	Convection
Continuous veno-venous haemodiafiltration (CVVHDF)	Convection and diffusion
Continuous veno-venous haemodialysis (CVVHD)	Diffusion
Slow continuous ultrafiltration (SCUF)	Fluid removal by ultrafiltration
High flux dialysis (HFD)	Convection and diffusion

CRRT MACHINE

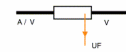


DIFFERENT TYPES OF CRRT CATHETER



CRRT

SCUF - Slow continuous ultrafiltration (AV or VV)



Technique used for fluid control only
Convective mechanism
Ultrafiltrate iso-osmotic to blood
Used in arteriovenous or venovenous mode
Qb = 50 - 100 ml/min
Ultrafiltration rate controlled

CVVH - Continuous veno-venous haemofiltration



Convective blood purification through high permeability membrane
Ultrafiltration rate controlled
Ultrafiltrate replaced by replacement solution
Qb = 50 - 200 ml/min Qd = 8-25 ml/min
K = 12 - 30 L/24h
Can be used in arteriovenous mode

CVVHD - Continuous veno-venous haemodialysis



Dilutional blood purification through low permeability dialyser
Dialysate solution in countercurrent flow
No replacement fluid used
Qb = 50 - 200 ml/min Qd = 2-4 ml/min
Qd = 10 - 20 ml/min K = 14 - 26 L/24h
Small molecule clearance only
Can be used in arteriovenous mode

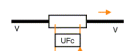
CRRT

CVVHDF - Continuous veno-venous haemodiafiltration



Diffusive and convective blood purification
Countercurrent dialysate flow
High permeability membrane utilized, thus small and middle molecules removed
Qb = 50 - 200 ml/min Qd = 8 - 12 ml/min
Qd = 10 - 20 ml/min K = 20 - 40 L/24h

CVVHDF - Continuous high flux dialysis



Diffusive and convective blood purification through a highly permeable membrane
Back diffusion occurs in membrane
Dialysate in countercurrent flow
Accessory pumps to control ultrafiltration
Replacement not required because fine regulation of filtration and backfiltration
Qb = 50 - 200 ml/min Qd = 2 - 8 ml/min
Qd = 50 - 200 ml/min K = 40 - 60 L/24h

CPFA - Continuous plasmafiltration adsorption



Highly permeable plasmafilter filters plasma allowing it to pass through a bed of adsorbent material (carbon or resin)
Fluid balance maintained
Can be coupled with CVVH or CVVHDF
Qb = 50 - 200 ml/min P1 = 20 - 30 ml/min

PRINCIPLE OF CRRT

Size of molecules cleared by continuous renal replacement therapies (CRRT)

Type of molecule	Size	Example	Mode of removal
Small	< 500 Da	Urea, creatinine, uretic acids	Convective, diffusive
Middle	500-2000 Da	Pharmaceuticals, amino acids	Convective, diffusive, adsorption
Size molecule weight proteins	2000-10000 Da	β ₂ microglobulin, cytokines, complement	Convective or adsorption only (if filter)
Large proteins	> 10000 Da	Albumin	Only removed adsorbed by standard CRRT

Da = Daltons

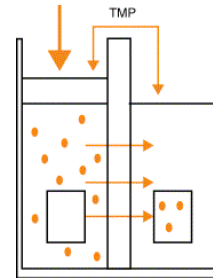
UREMIC SOLUTES

Table 1. Uremic Solutes.*

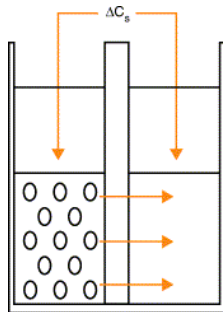
Solute Group	Example	Source	Characteristics
Peptides and small proteins	Beta ₂ -microglobulin	Shed from MHC	Poorly dialyzed because of large size
Guanidines	Guanidinosuccinic acid	Arginine	Increased production in uremia
Phenols	p-Cresol sulfate	Phenylalanine, tyrosine	Protein bound, produced by gut bacteria
Indoles	Indican	Tryptophan	Protein bound, produced by gut bacteria
Aliphatic amines	Dimethylamine	Choline	Large volume of distribution, produced by gut bacteria
Furans	CMPF	Unknown	Tightly protein bound
Polyols	Myoinositol	Dietary intake, cell synthesis from glucose	Normally degraded by the kidney rather than excreted
Nucleosides	Pseudouridine	tRNA	Most prominent of several altered RNA species
Dicarboxylic acids	Oxalate	Ascorbic acid	Formation of crystal deposits
Carbonyls	Glyoxal	Glycolytic intermediates	Reaction with proteins to form advanced glycation end products

* Uremic solutes may have multiple sources, although only one is listed. MHC denotes major histocompatibility complex, and CMPF 3-carboxy-4-methyl-5-propyl-2-furanpropionic acid.

CONVECTION PRINCIPLE

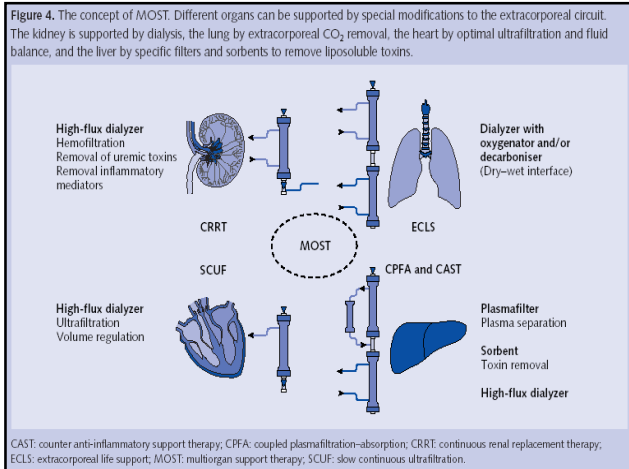
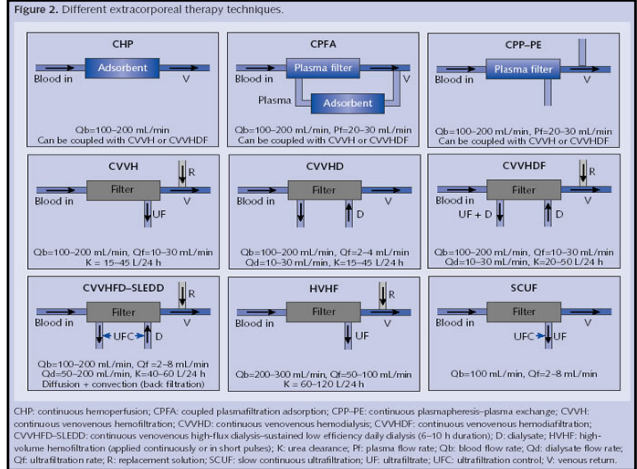
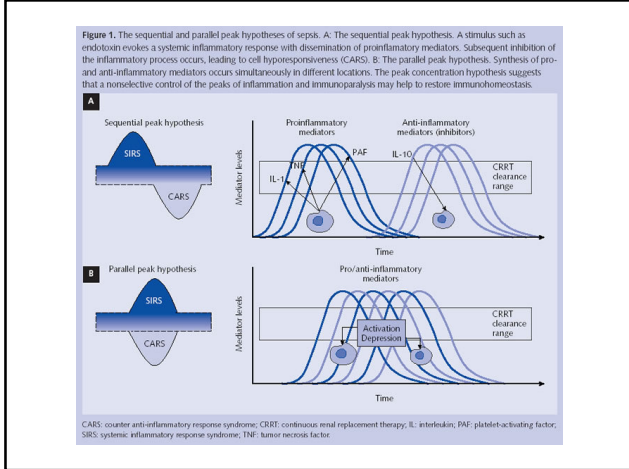


DIFFUSION PRINCIPLE



CRRT

Technique	Diffusion	Convection	Replacement fluid	Dialysis fluid	Back filtration
Intermittent haemodialysis (IHD)	+++++	+	No	Yes	+
Daily haemodialysis (DHD)	+++++	+	No	Yes	+
Intermittent haemofiltration (HF)	+++++	+	Yes	Yes	+
Intermittent haemodiafiltration (HDF)	+++	+++	Yes	Yes	+
Intermittent high flux dialysis (HFD)	++++	++	No	Yes	++++
Sustained low efficiency dialysis (SLED)	+++	+	No	Yes	+
Continuous venovenous haemofiltration (CVVH)	+++++	+	Yes	Yes	+
Continuous venovenous haemodialysis (CVVHD)	+++++	+	No	Yes	+
Continuous venovenous haemodiafiltration (CVVHDF)	+++	+++	Yes	Yes	+
Continuous venovenous high flux dialysis (CVVHFD)	++++	++	No	Yes	++++
Intermittent ultrafiltration (UF)	+++++	+	No	Yes	+
Slow continuous ultrafiltration (SCUF)	+++++	+	No	Yes	+
Slow continuous ultrafiltration with dialysate (SCUF-D)	++	+++	No	Yes	+
High volume haemofiltration (HVHF)	+++++	+++++	+	+	+
Coupled plasmafiltration-absorption (CPFA)*	+	+	+	+	+



THE GOALS OF MOST (MULTIORGAN SUPPORT THERAPY)

- Blood purification and renal support.
- Temperature control.
- Acid-base control.
- Fluid balance control and cardiac support.
- Protective lung support and removal of carbon dioxide.
- Protection of the brain from fluid shifts.
- Blood detoxification and liver support.
- Sepsis therapy, immunomodulation, and endothelial support.

Pharmacokinetics of hemodialysis

Pharmacokinetics

Drugs removed on haemodialysis

- Salicylates
- Methanol
- Barbiturates
- Lithium
- Aminoglycosides
- Cephalosporins

Drugs not removed on haemodialysis

- Digoxin
- Tricyclic antidepressants
- Phenytoin
- Benzodiazepines
- β -blockers
- Oral hypoglycaemic agents

IHD VS CRRT

	Intermittent haemodialysis	Continuous renal replacement therapy
Advantages	<ul style="list-style-type: none"> Lower risk of systemic bleeding More time available for diagnostic and therapeutic interventions More suitable for severe hyperkalaemia Lower cost 	<ul style="list-style-type: none"> Better haemodynamic stability Fewer cardiac arrhythmias Improved nutritional support Better pulmonary gas exchange Better fluid control Better biochemical control Shorter stay in intensive-care unit
Disadvantages	<ul style="list-style-type: none"> Availability of dialysis staff More difficult haemodynamic control Inadequate dialysis dose Inadequate fluid control Inadequate nutritional support Not suitable for patients with intracranial hypertension No removal of cytokines Potential complement activation by non-biocompatible membranes 	<ul style="list-style-type: none"> Greater vascular access problems Higher risk of systemic bleeding Long-term immobilisation of patient More filter problems (ruptures, clotting) Greater cost

IHD vs. CRRT

- Randomized trial, observational studies unclear and limited because of patient populations and significant cross-over to CRRT.
- Meta-analysis unclear because of limitation of original studies.
- **LEVEL 2B SUGGESTION** of no difference between the use of IHD vs. CRRT as therapy for acute renal failure.

Renal Replacement

- **Absence of hemodynamic instability**
 - Intermittent hemodialysis and continuous venovenous filtration equal (CVVH)
- **Hemodynamic instability**
 - CVVH preferred

Grade B

THANK YOU FOR YOUR
ATTENTION