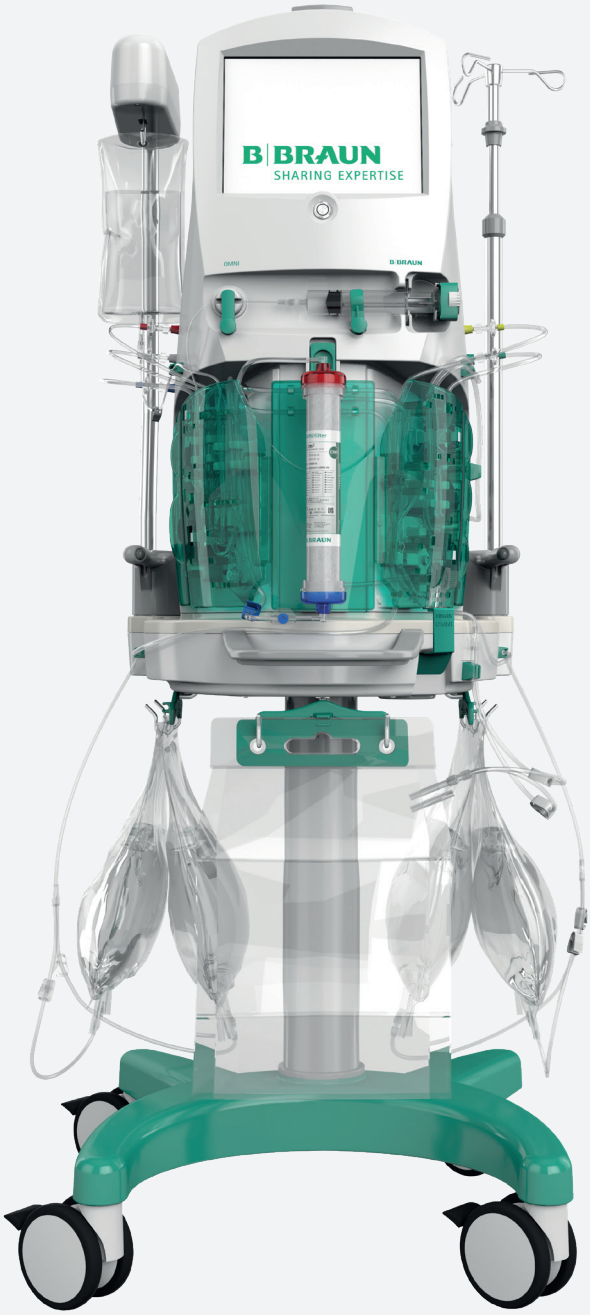
B BRAUN
SHARING EXPERTISE



B. BRAUN OMNI®

ACUTE BLOOD PURIFICATION

CONTINUOUS RENAL REPLACEMENT THERAPY (CRRT) PRE-COURSE READING MATERIAL[1]

Introduction

The kidneys are regulatory and excretory organs and play a vital role in maintaining homeostasis within the body. The primary function of the kidney is to produce urine, but it also has secondary functions including helping maintain blood pressure, producing erythropoietin (which is essential in red blood cell production) and activating Vitamin D. On account of the essential roles that the kidneys play, 15 - 25% of all cardiac output is directed to them. This amounts to approximately 625 ml/min. When patients experience renal pathology, there can be catastrophic outcomes, and intervention within the ITU may be essential in order to preserve life.

Anatomy of the Kidney[2,21]

The kidneys are bean shaped organs that lie on the posterior abdominal wall, one on each side of the spine, below the diaphragm. They are approximately 11 cm long, 6 cm wide and 3 cm thick – around the size of a human fist.

Functions of the kidney:

- Excretion of waste products, including urea, creatinine, drugs, and poisons.
- Regulation of fluid balance through ultrafiltration and reabsorption.
- Maintenance of acid base and electrolyte balance through reabsorption and excretion.
- Activation of Vitamin D, and regulation of calcium and phosphorus.
- Secretion of erythropoietin (EPO) for production of red blood cells.
- Regulation of blood pressure through secretion of renin, which causes vasoconstriction and helps to control sodium retention via the renin angiotensin system.

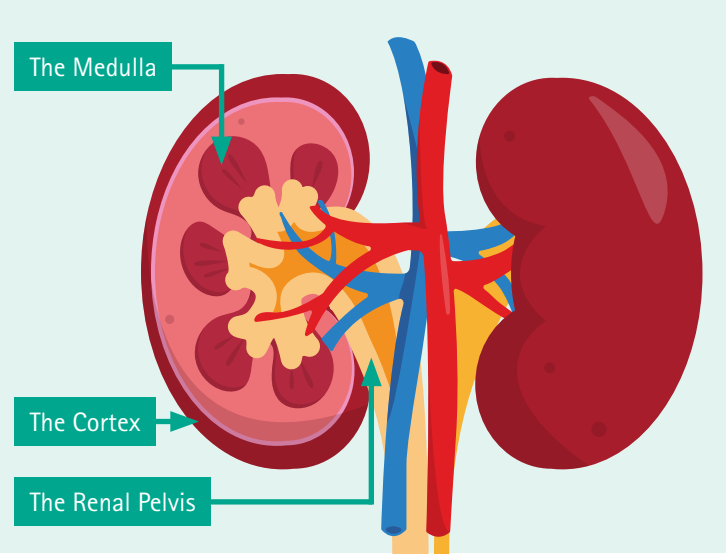
The kidneys consist of three layers:

1. The cortex (outer layer) - contains 80% of the nephrons
2. The medulla (inner layer) - contains 20% of the nephrons
3. The renal pelvis

The **nephron** is known as the functional unit of the kidney, they can be found primarily within the cortex (approximately 80%).

The **cortex** also contains the convoluted tubule structures. The remaining 20% of the nephrons can be found within the medulla. The **medulla** also houses the loop of Henle structures and the renal pyramids. Concentration of urine takes place within the medulla.

The **renal pelvis** is the beginning of the collecting system from which urine leaves the kidney and travels to the bladder via the ureter.



The Nephron

Around 1,000,000 nephrons can be found in each kidney. The structure is highly adapted in order to carry out ultrafiltration, excretion and reabsorption. Each nephron is composed of a renal corpuscle (glomerulus within Bowman's capsule), a proximal tubule, the loop of Henle, a distal convoluted tubule, a connecting tubule, and the collecting duct structures. The vasculature is very important in effective renal physiology.

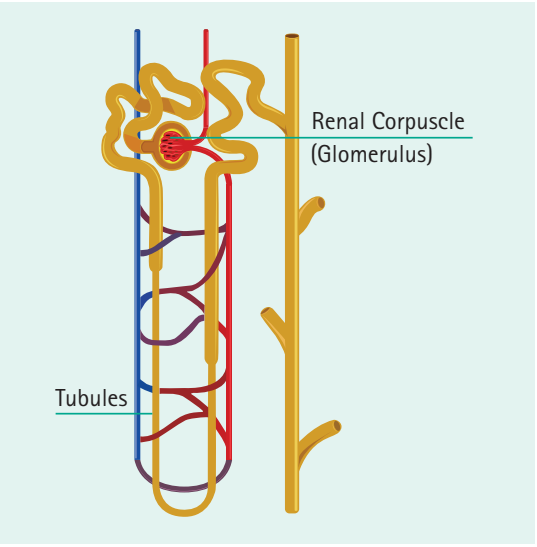
Anatomy of the Kidney[2]

The Glomerulus

When blood enters the nephron, it is transported to the glomerulus which is a network of capillaries, each with a semi-permeable membrane, allowing select molecules to exit the blood and enter the 'filtrate'. The ability of molecules to cross the semi-permeable membrane is determined by their size, charge and structure.The high osmotic/hydrostatic pressure in the glomerulus creates ideal conditions for movement. This process is known as ultrafiltration.

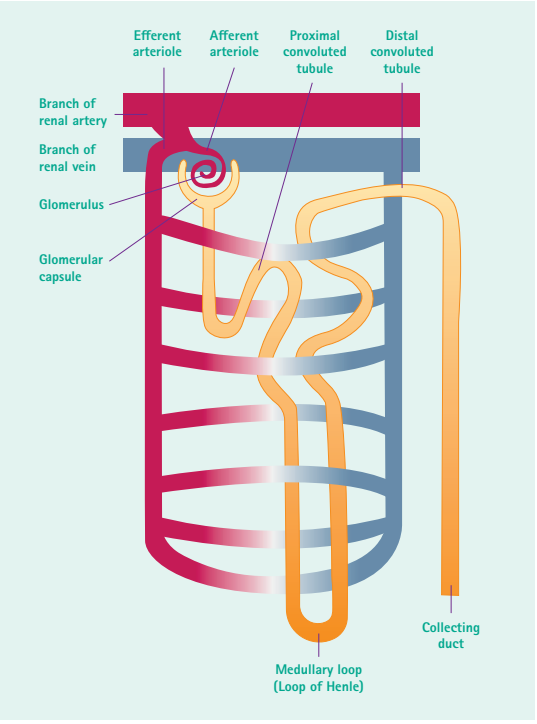
Removal of molecules from the blood via this selective process is key in maintaining fluid balance, electrolyte status and excreting waste products. The membrane is impermeable to molecules larger than 70,000 Da. A huge volume of plasma water is removed by the glomerulus on a daily basis (approximately 180 litres per day), this is concentrated throughout the structures in the kidneys and therefore only approximately 2 litres of urine is actually lost.

The plasma water which is removed via the semipermeable membrane is known as the glomerular filtrate, and the glomerular filtrate rate is the amount of plasma water removed via the glomerulus per minute, in a healthy adult the GFR is around 95 - 125 ml/min.



Vasculature[21]

Blood leaves the heart, enters the abdominal aorta, and enters the kidney through the renal artery. The renal artery divides into seven branches of arterioles until it becomes the afferent arteriole. The afferent arteriole carries blood to the glomerulus, where it is filtered. It then leaves the glomerulus through the efferent arteriole and is returned to the venous system. This system branches into many larger vessels until it becomes the renal vein. Blood leaves the kidney via the renal vein and is returned to the heart via the inferior vena cava.



Tubular System

Of the 180 litres of filtrate that is produced throughout the day via ultrafiltration, 2/3 of it is reabsorbed within the proximal convoluted tubule. Substances destined for removal, which were not removed during the initial filtration process, are then secreted in the distal convoluted tubule (this is known as tubular secretion), these include molecules such as phosphate, glucose, bicarbonate, potassium, sulphate, amino acids, and some smaller proteins. Both reabsorption and secretion continue to occur down the length of the tubules until finally the filtrate reaches the collecting duct by which time, 99% has been reabsorbed into the blood stream. The urine then passes through the renal pelvis, into the ureters and is stored in the bladder for excretion.

Renal Pathology

The kidney has advanced compensatory mechanisms which allow it to continue to function effectively, even when a large number of nephrons are damaged. This compensation continues until around 50% of all nephrons are damaged, for this reason, many people can go on to live heathy lives with only one kidney. After around 90% loss of kidney function, renal replacement therapy is likely to be indicated.

Acute Kidney Injury

Acute Kidney Injury (AKI)

There are numerous definitions of acute kidney injury but possibly the most respected definition is by Nissenson[3], who states that acute kidney injury is an abrupt decline in glomerular filtration rate resulting from ischemic or toxic injury to the kidney. It often results in sudden loss of kidney function. Approximately 30% of all intensive care patients develop an acute kidney injury and the most common cause is hypo-perfusion. Hypo-perfusion is categorised as a prerenal acute kidney injury, these account for around 60% of all kidney injuries. AKI results in the patient retaining various uremic substances such as urea and creatinine, it may also be accompanied with a deranged fluid balance, a deranged electrolyte and/or acid base balance.

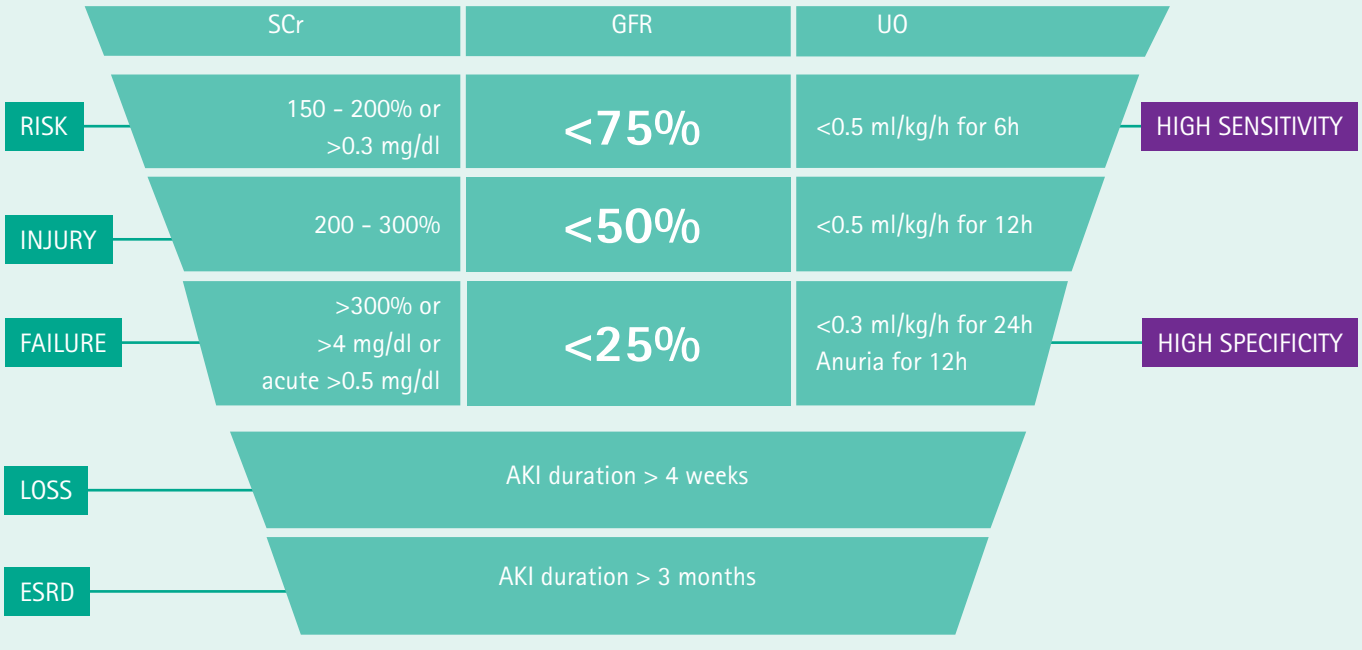
KDIGO have published guidelines for the classification of AKI based on serum creatinine levels and urine output. It is widely agreed that uraemia is a unreliable indicator of renal function as is GFR. RIFLE have also produced AKI classifications. Most patients experiencing AKI in the intensive care unit present with multi organ failure and therefore the percentage survival rate is approximately 50%. AKI is most commonly reversible when treated and the majority of those who survive will go on not to require long term dialysis[4].

THE KDIGO AKI STAGING GUIDANCE[5]

Stage	Serum Creatinine (SCr) Criteria	Urine Output Criteria
1	1.5 to 1.9 times baseline OR ≥ 26 µmol/l (≥ 3.0 mg/dl) increase	< 0.5 ml/kg/hr for 6 - 12 hours
2	2 to 2.9 times baseline	< 0.5 ml/kg/hr for ≥ 12 hours
3	Serum Creatinine increase 3.0 times baseline OR increase in serum creatinine ≥ 352 µmol/l (≥ 4.0 mg/dl) OR Initiation for RRT OR in patients <18 years decrease in eGFR to < 35 ml/min per 1.73 m^2	<0.3 ml/kg/hr for ≥ 24 hours OR Anuria for ≥ 12 hours

Must have met initial criteria for definition of AKI.

ADQI RIFLE STAGING FOR AKI[6]



Acute Kidney Injury

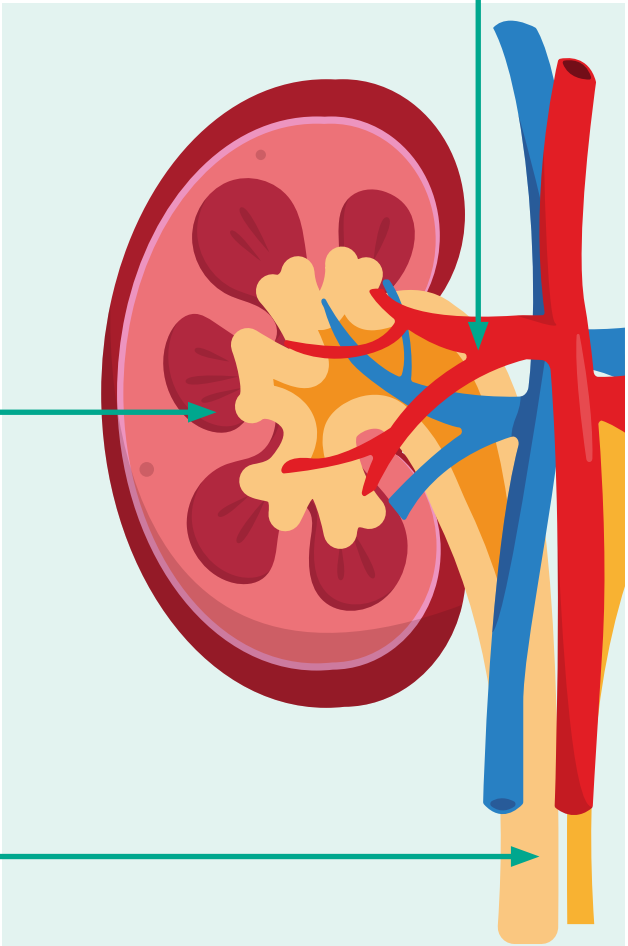
Types of AKI[7,22]

AKI can be classified as pre-renal, renal (intrinsic) or post-renal. Pre-renal and post-renal AKIs have better outcomes (the exceptions are hepotorenal and cardiorenal syndrome).

PRE-RENAL

Pre-renal AKI accounts for around 60% of cases and refers to acute kidney injury caused by problems 'before' the kidney, usually being caused by reduced blood flow to the kidneys themselves.

There is no structural damage to the kidneys, however inadequate blood flow results in reduced glomerular filtration leading to AKI. Examples of pre-renal AKI include hypovolemic causes such as bleeding, cardiogenic causes such as heart failure and obstructive causes such as pericardial tamponade. If pre-renal failure is not reversed, it can lead to renal AKI.



RENAL

Renal AKI accounts for around 35% of cases. This usually occurs as a result of functional disturbances and direct injury to the kidney itself.

Renal AKI classification can be divided into acute tubular necrosis such as pancreatitis, burns etc. cortical necrosis such as DIC, infective endocarditis etc. and acute interstitial nephritis such as drug toxicity. Primarily, renal AKIs can be attributed to sepsis aetiology.

POST-RENAL

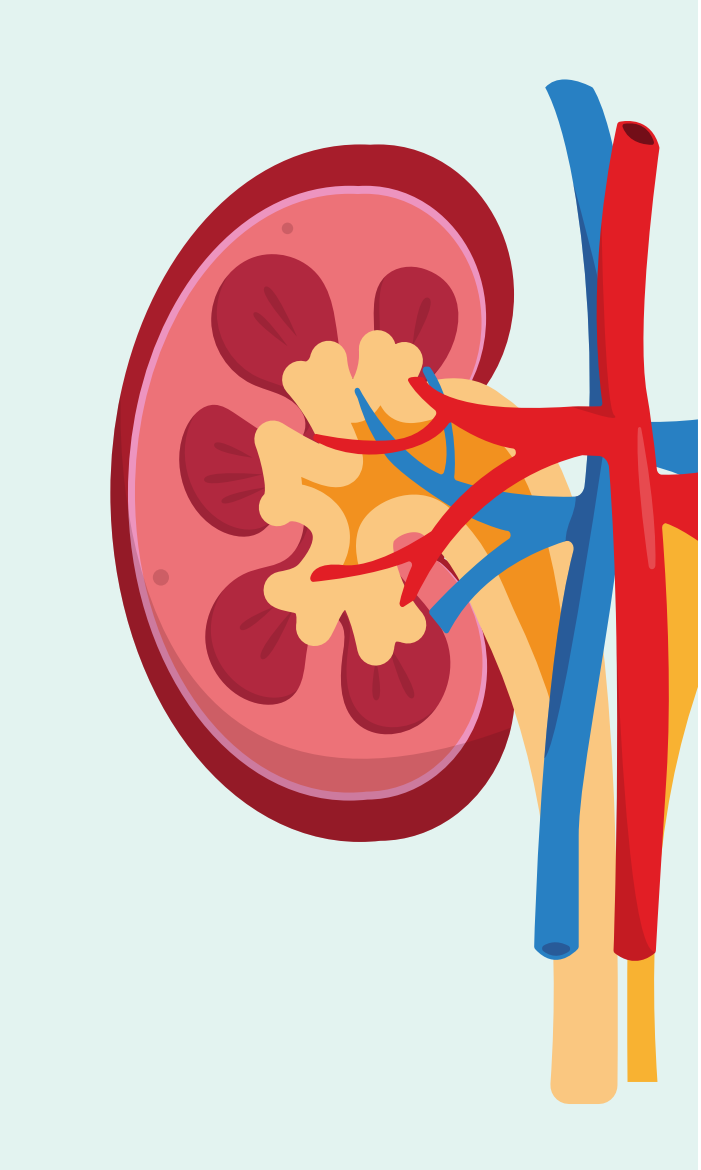
Post-Renal AKI accounts for only around 5% of cases and occurs due to a disruption in the urinary tract or due to problems occurring after the kidneys themselves.

This diagnosis can be subdivided into intra-ureteral, extra-ureteral, bladder obstruction and urethral obstruction. Examples include stricture, cancer or thrombi.

Complications of Acute Kidney Injury (AKI)[8]

UREMIA

- Nausea
- Vomiting
- Drowsiness
- Bleeding
- Uremic flap
- Coma
- Uremic encephalopathy
- Pericardial rub



VOLUME OVERLOAD

- Salt and water retention effecting respiratory and cardiac function
- Peripheral oedema affecting wound healing

ELECTROLYTE DISTURBANCES

- Hyponatraemia
- Hyperkalaemia
- Hyperphosphatemia

ACIDOSIS

- Retention of organic production of anions
- Reduced production of bicarbonate

ANEMIA/BLEEDING

- Due to decreased erythropoiesis and RBC fragility
- Uremia is also associated with platelet dysfunction
- Increased risk of GI bleeding

DRUG ACCUMULATION

- Side effects of accumulation i.e. respiratory failure

IMMUNOSUPPRESSION

- Humoral and cellular immunity is impaired

METABOLIC CONSEQUENCES

- Hyperglycemia occurs due to peripheral insulin resistance and increased hepatic gluconeogenesis
- Protein catabolism is also activated

Continuous Renal Replacement Therapy

Medical reversal of AKI should be attempted prior to initiation of CRRT[9].

- Stabilise haemodynamics for optimal renal blood supply
- Correct fluids, electrolytes & acid/base
- Stop nephrotoxic medications
- Catecholamines
- Loop diuretics
- Crystalloids
- Potassium binders
- Sodium bicarbonate

CHECK ALL KNOWN NEPHROTOXIC DRUGS[10]:

- Antimicrobial Agents: Penicillin, Amoxicillin, Ampicillin, Ciprofloxacim, Cephalosporin, Sulfonamide, Rifampicin, Co-trimoxazole, Gentamicin, Aminoglycoside, Amphotericin B, Vancomycin, Aciclovir
- Iodated Contrast Agents
- NSAID's/Salicylates: Ibuprofen, Naproxen, Indomethacin, Piroxicam
- Antiulcer Agents: Cimetidine, Omeprazole
- Others: ACE-I, ARB, Cyclosporine, NSAR, Phenytoin, Thiacide Diuretics, Furosemide, Allopurinol, Mesalazine

Research has been done into the right time for commencing CRRT, with pros and cons seen for both early and delayed commencement. There is no clear, definitive answer[11].

Early initiation can lead to prolonged anticoagulation, hypotension, and infection in critically ill patients.

Conversely, late intervention can be associated with an increased risk of life-threatening complications such as uremic syndrome (a build-up of urea can lead to encephalopathy and pericarditis), hyperkalaemia, pulmonary oedema etc[12].

Physicians are required to make an individual decision for the patient, and it is not a 'one size fits all' approach[13].

UREMIC SYNDROME
- Disorder of consciousness (Encephalopathy)
- Pericardite

HYPERKALAEMIA
- Vital risk due to cardiac arrest

METABOLIC ACIDOSIS

Continuous Renal Replacement Therapy

Dialysis

Intermittent haemodialysis (IHD) is an alternative therapy to CRRT, it is the most common treatment choice for those patients with chronic renal failure. IHD is a more rapid treatment, designed to remove fluid and waste over approximately four hours, it is usually given two - three days /week. IHD is often unsuitable in the critically ill patient due to the speed at which fluid and solutes are removed as it can cause further haemodynamic instability.

CRRT is primarily used over IHD in ICU because solute and fluid removal is slower and more gentle on account of the continuous nature of the therapy. This means that unstable patients can tolerate CRRT better than IHD. CRRT can be administered 24 hours a day for as long as necessary and the prescription and therapy type can be altered at any time in accordance with patient status[14].

Characteristics of IHD vs CRRT[15]:

IHD - HD, HF, HDF, SLEDD	CRRT - SCUF, CVVH, CVVHD, CVVHDF
Intermittent and fast	Continuous and slow
Can be used every couple of days and lasts around four hours	Can be used continuously for hours/days/weeks
High clearance of small molecules	Low clearance of small molecules
Used in chronic renal patients	Used to treat acute kidney injury
Poorly tolerated in critically ill patients	Well tolerated in critically ill patients
Access via fistula or alternative	Access via venous catheter or alternative
Low heparinisation	Continuous heparinisation
Good patient mobilisation	Poor patient mobilisation
Accumulation of fluids and toxins between treatments	
Disequilibrium syndrome (cerebral odema)	

Complications associated with continuous and intermittent dialysis[15]:

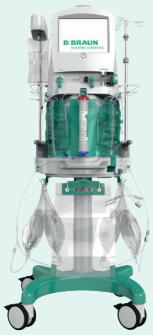
IHD	CRRT
Hypotension	Filter clotting
Arrhythmia	Fluid balance error
Hemolysis	Hypothermia
Muscle cramps	Vascular access problems
Air embolism	Coagulation problems
Disequilibrium syndrome	Hypotension
Pulmonary odema	Infection sepsis
Hyperkalaemia	Air embolism
Hypertension	Disconnection

Continuous Renal Replacement Therapy

Indications and goals of CRRT

CRRT allows for solute and/or fluid removal from a patient in a slow and continuous manner. Most commonly CRRT will be commenced for those with fluid overload, acidosis, uraemia, and hyperkalaemia. There are also some non-renal indications for initiating CRRT such as sepsis, pancreatitis, hyperthermia, rhabdomyolysis etc.

GOALS OF CRRT[21]



- Removal of solutes
- Removal of fluid
- Correction of acid/base balance
- Correction of electrolytes
- Removal of septic mediators
- Removal of undesirable drugs
- Hemodynamic stabilisation

Molecular Transport in CRRT

During dialysis, fluid and solutes are filtered from the blood via four different processes. Fluid is removed by the process of ultrafiltration and solutes are removed through diffusion, convection and/or adsorption. Both ultrafiltration and adsorption are present in all modalities of CRRT whereas diffusion and convection can be applied individually or in combination as with CVVHDF.

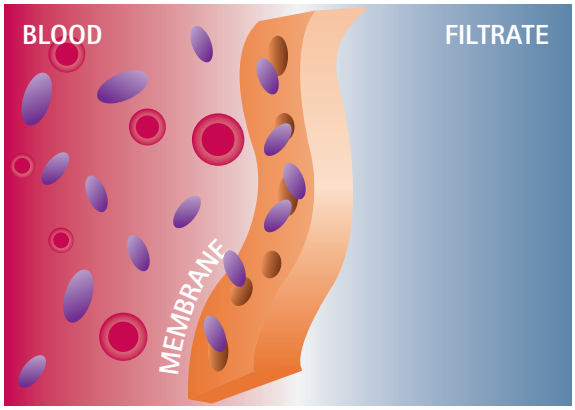
	ULTRAFILTRATION	ADSORPTION	CONVECTION	DIFFUSION
Haemofiltration (CVVH)	✓	✓	✓	
Haemodialysis (CVVHD)	✓	✓		✓
Haemodiafiltration (CVVHDF)	✓	✓	✓	✓

Continuous Renal Replacement Therapy

Adsorption

Definition: Adsorption is the adherence of larger molecules such as proteins to the filter structure (sometimes referred to as protein cake). Molecular structure, charge and other characteristics determine the degree of membrane-molecule interaction.

When there is a large degree of adsorption, the filter will begin to clog. After a time, this clogging will lead to clotting.

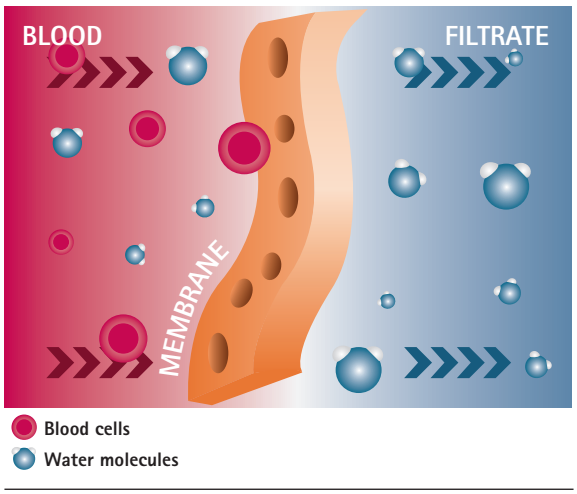


Ultrafiltration

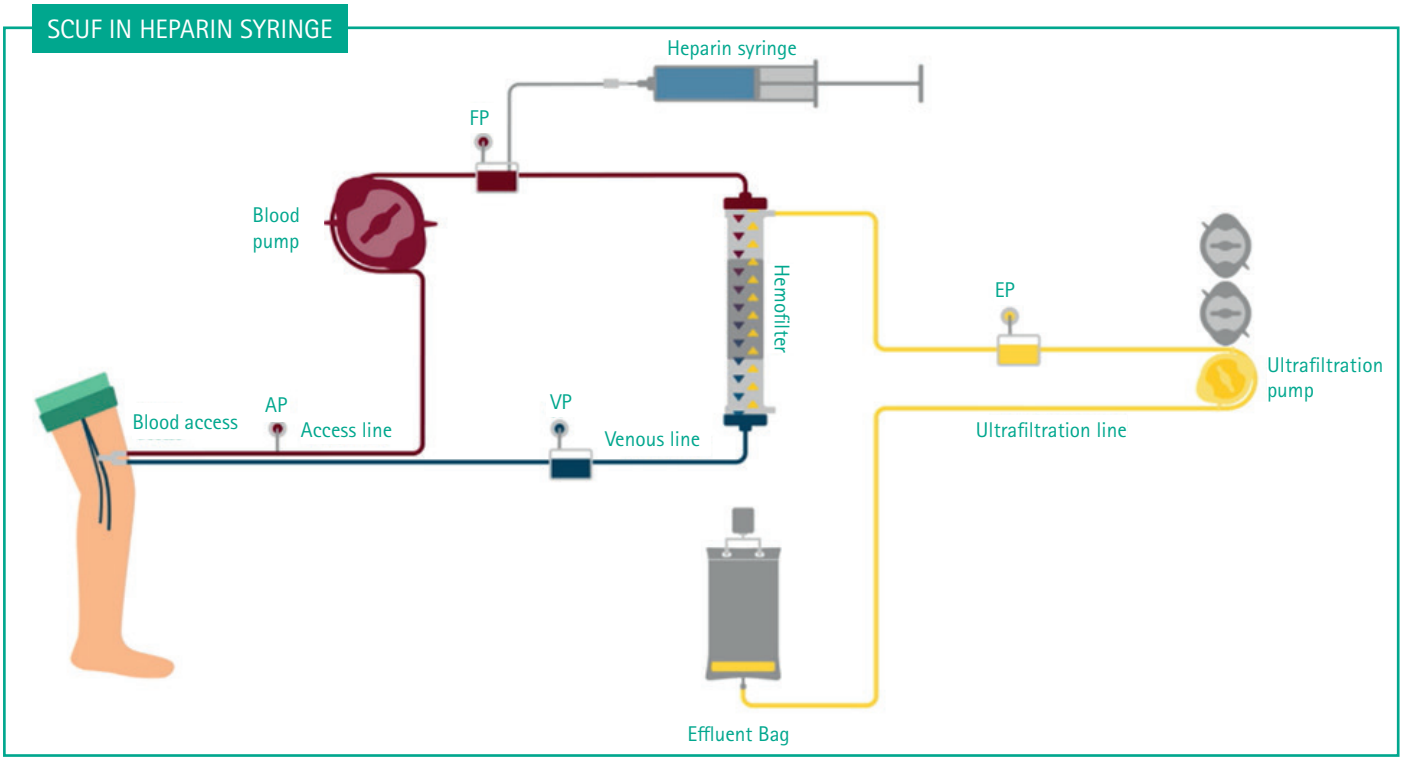
Definition: Ultrafiltration is the one-way movement of water from an area of high pressure to an area of lesser pressure via a semipermeable membrane. It is free of solvents.

The rate of ultrafiltration is affected by intrinsic values such as transmembrane pressures and permeability of the membrane. It is via ultrafiltration that ultrafiltrate is removed AKA 'net fluid removal'.

Therapy application: Slow continuous ultrafiltration (SCUF) is a therapy used to achieve fluid removal only, without any solvent removal. This therapy can be administered to patients experiencing fluid overload without concerns over electrolyte or acid base imbalance. There is no need for dialysate or substitution fluid as neither convective, nor diffusive transport is taking place and there is no requirement for volume replacement.



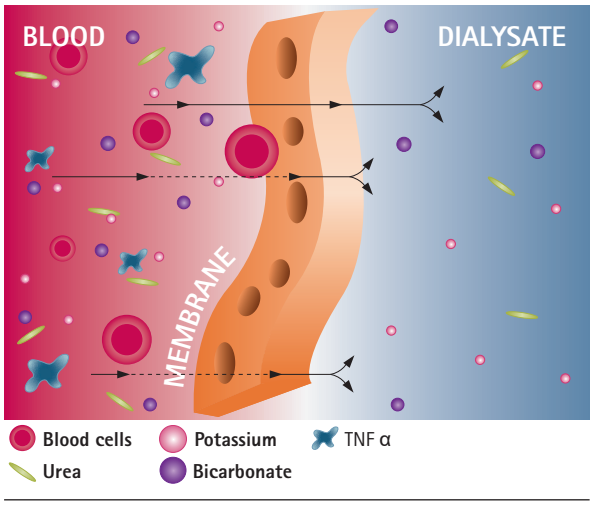
Instead, pressure is applied to the blood as it travels down the filter, creating a pressure gradient in order to facilitate ultrafiltration. The ultrafiltrate leaves the blood side compartment and enters the fluid side compartment, this waste fluid is then termed 'effluent'.



Continuous Renal Replacement Therapy[21]

Diffusion

Definition: Diffusion is the random movement of molecules across a semi permeable membrane from an area of high concentration to an area of low concentration until an equilibrium is reached. This process is slower than movement via convection. You can think of diffusion as a similar process to making tea, whereby you leave the teabag in the water and after a time, the concentration of the tea inside the bag and the concentration of tea outside the bag will be the same. You don't even need to stir it!



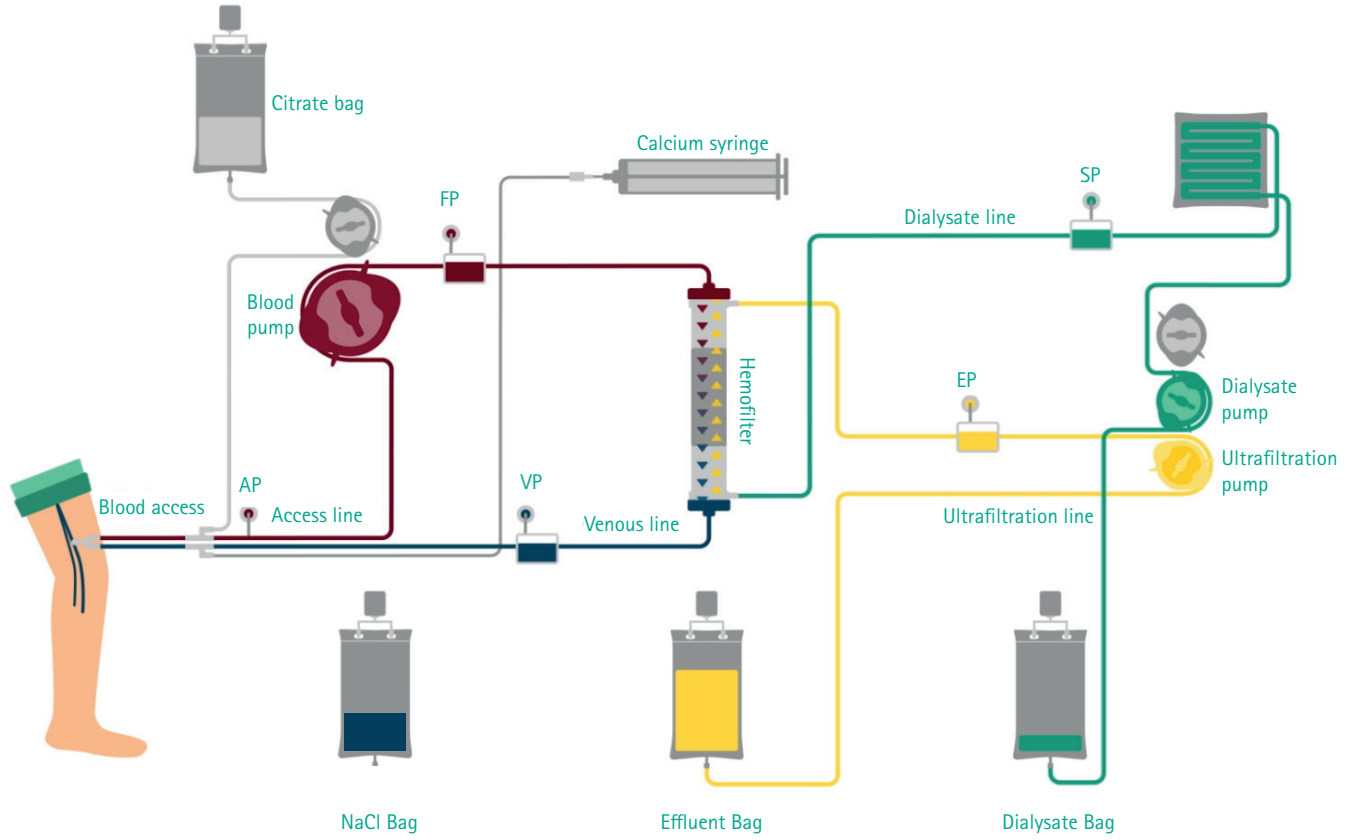
Therapy application: In diffusive therapies, blood flows down the middle of the filter and the dialysate surrounds it flowing counter current to the blood. The benefit of the blood compartment and fluid compartment flowing counter current is that an equal concentration gradient is observed throughout the full length of the haemofilter. The dialysate fluid used in diffusion contains near normal levels of electrolytes, encouraging the excess solutes to diffuse across from the blood to the dialysate side. Remember, blood is not mixed with substitution fluid.

Factors governing diffusive solute transport:

SOLUTE FACTORS	FLOW RATES	MEMBRANE FACTORS	OTHERS
Size	Blood flow/solute delivery	Type	Duration of dialysis
Charge	Dialysate flow/solute removal	Porosity	
Protein binding	Concentration gradient	Surface area	

Continuous Renal Replacement Therapy[21]

CVVHD IN CITRATE ANTICOAGULATION



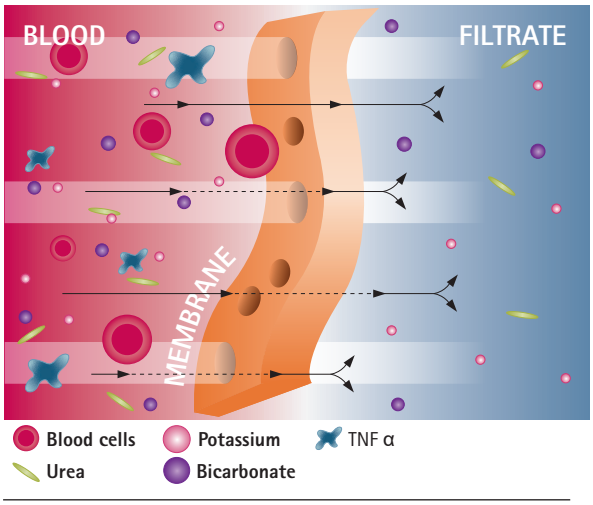
This is an example of CVVHD in citrate anticoagulation where you can see the dialysate fluid enters the haemofilter at the bottom and flows counter current to the blood. There is no substitution fluid involved. Citrate anticoagulation is infused into the arterial line and calcium substitution if infused into the venous line.

Summary of diffusive therapies[21]:

	CONTINUOUS DIFFUSIVE REMOVAL OF WASTE PRODUCTS
Primary Therapeutic Goal:	Solute removal and safe management of fluid volume
Primary Indications:	Uraemia, hyperkalaemia, acid/base of electrolyte imbalance, fluid overload
Principle Used:	Diffusion
Therapy Characteristics:	■ Requires dialysate solution ■ No substitution solution ■ Low blood flows ■ Ideal for citrate anticoagulation ■ Used to achieve solute removal (**small and mid sized** molecules) and fluid balance

Continuous Renal Replacement Therapy[21]

Convection



Definition: During ultrafiltration, some solvents can be dragged across the membrane along with the water. This is known as solvent drag and is the process of convection. Again, this process is driven by pressure gradients, in particular, osmotic and hydrostatic pressure gradients.

Convection is effective at transporting small, medium and some **larger** sized molecules. This is a one-way movement and is fairly rapid by comparison to diffusive methods.

You may choose to use convective therapies over diffusive therapies in patients with rhabdomyolysis, compartment syndrome, overdose, and sepsis (to name only a few), as the molecules required to be removed in these pathologies are larger. A classic membrane has a cut off of 30-45 kDa in convective therapies, albumin is 65-70 kDa and therefore you will not remove this vital protein.

You can think of convection as a similar process to making coffee whereby you add the coffee pod to the machine and force hot water through it. The hot water passes out the other side, but it also drags with it many coffee grains and hence you make your cup of coffee.

Therapy application: In convective processes, blood is mixed with substitution fluid. This can be either before or after the haemofilter, or both before and after the haemofilter (CVVH). It is also possible to use both diffusion and convection in combination, as is the case with CVVHDF. On the OMNI, CVVHDF only allows post-filter substitution. If pre-filter substitution is selected (in CVVH), the substitution is infused into the arterial line, in the degassing chamber, and is then pushed through the narrow capillaries at a high speed which assists in creating a pressure gradient.

As the blood moves through the haemofilter and plasma water is removed, it becomes more and more haemo-concentrated. This puts the blood at risk of clotting. More plasma water is removed via convection than is desirable. For this reason, replacement fluid is required after the haemofilter to ensure an even fluid balance (post-filter substitution fluid).

Most hospitals use a ratio of 1/3 pre-filter, 2/3 post-filter when administering CVVH. This can be manipulated if you are struggling with alarms related to TMP, pre-filter pressure etc.

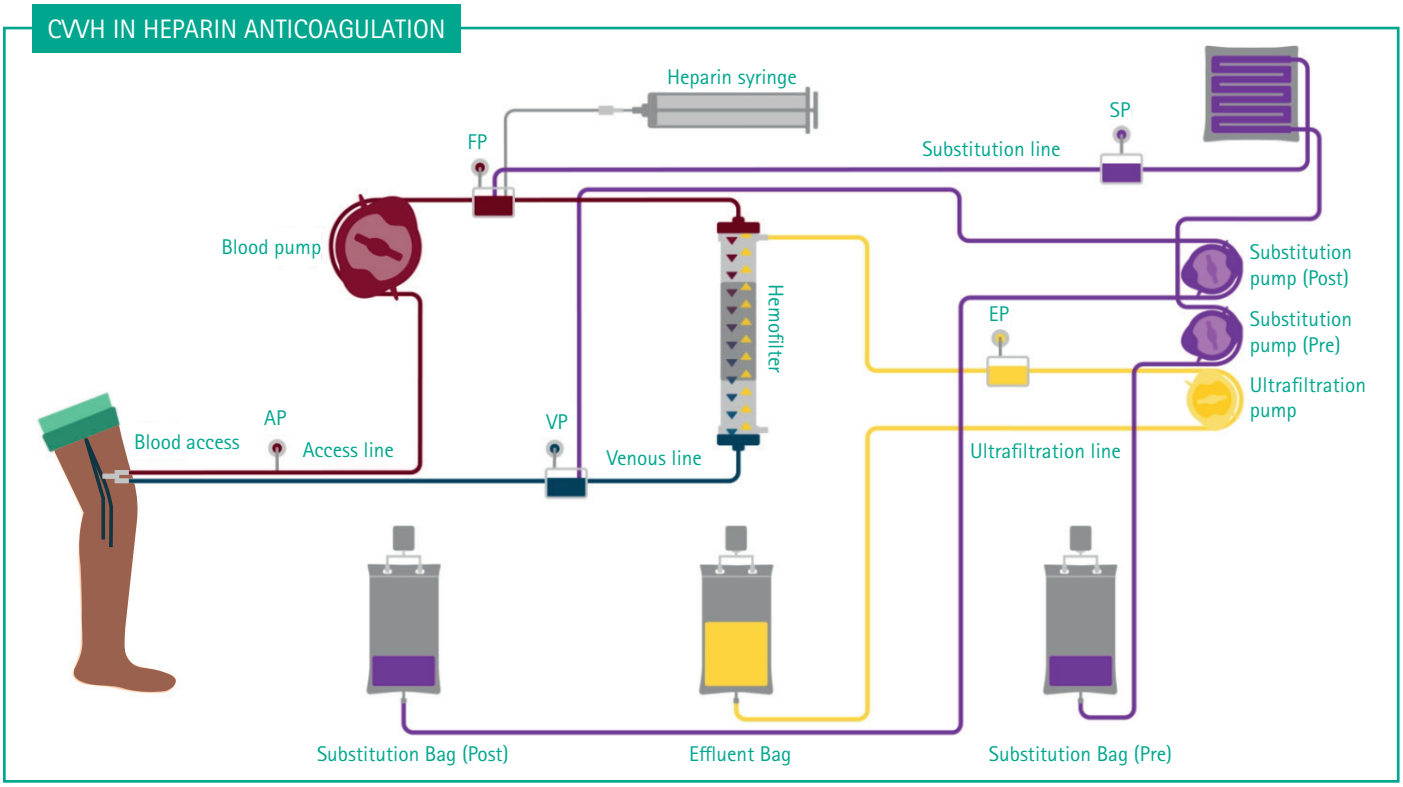
Factors governing convective solute transport:

SIEVING COEFFICIENT OF SOLUTE	TRANSMEMBRANE PRESSURE	MEMBRANE FACTORS	OTHER
Size	Blood flow	Type	Duration of dialysis
Charge	Oncotic pressure	Water permeability	Concentration of solute in blood
Protein binding	Negative hydrostatic pressure	Surface area	

Continuous Renal Replacement Therapy[21]

Selection of pre or post-filter substitution fluid[16]:

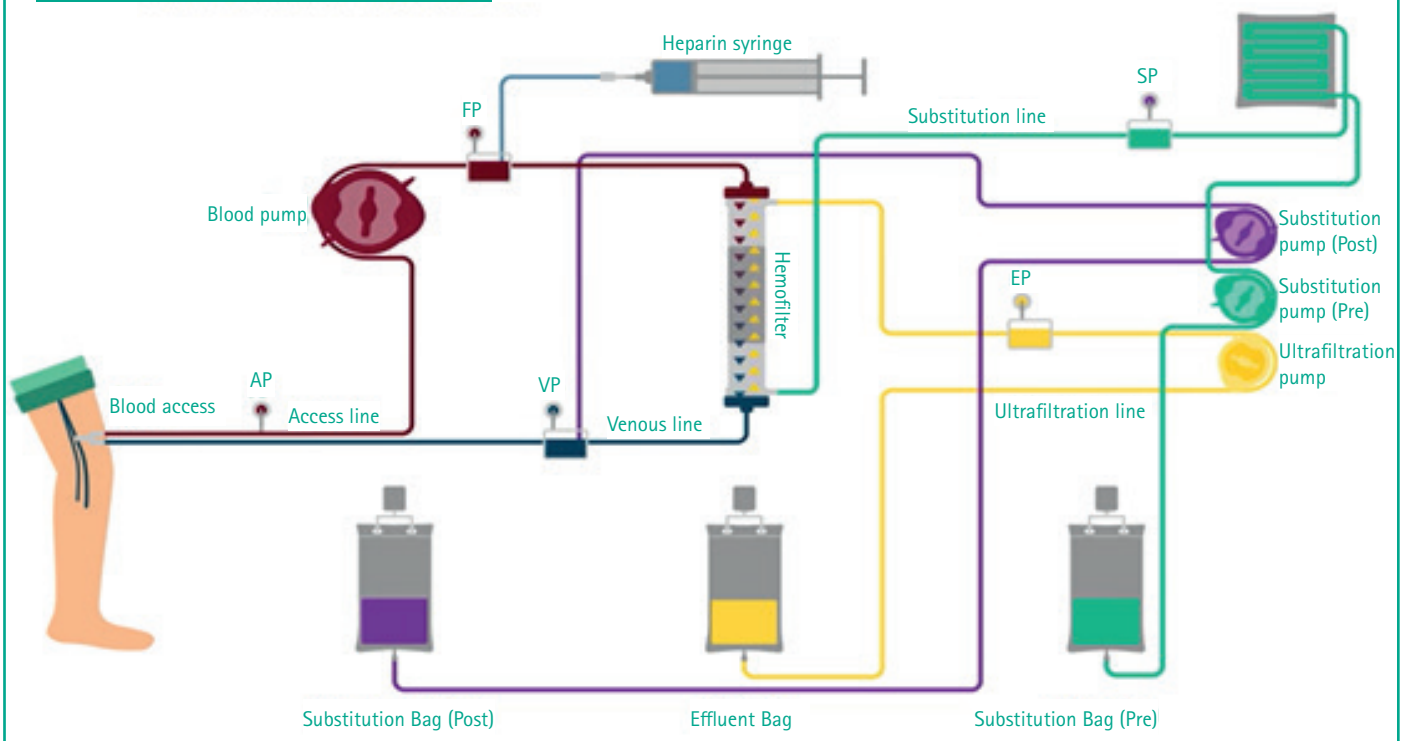
	PROS	CONS
Pre-filter substitution	Less viscous	Greater volume to be filtered
	Reduced risk of clogging leading to clotting	Reduced efficiency
Post-filter substitution	Smaller volume to be filtered	More viscous
	Increased efficiency	Greater rate of clogging leading to clotting



This is an example of CVVH with pre and post-filter substitution fluid and heparin anticoagulation. You can observe the substitution fluids being infused directly into the patients blood via the arterial and venous degassing chambers.

Continuous Renal Replacement Therapy[21]

CVVHDF IN HEPARIN ANTICOAGULATION



In the diagram above, you can see CVVHDF in heparin anticoagulation. On the right scale, dialysate fluid is infused into the bottom of the haemofilter, creating a concentration gradient favourable to diffusive movement.

In addition, pressure is applied to the blood side compartment, facilitating convective movement, for this reason, large volumes of plasma water are removed along with solvents. Post-filter substitution fluid is used to replace the plasma volume lost and is infused into the venous degassing chamber.

It is possible to use CVVHDF in citrate anticoagulation in the latest software versions on the OMNI, in which case, calcium free bicarbonate solution will go on the right-hand scale and calcium containing solution such as Duosol will go on the left-hand scale.

This means that some degree of calcium replacement takes place via the substitution fluid and therefore the dose of calcium delivered by the integrated syringe driver may be slightly lower than would be the case in CVVHD. Always check your protocol.

Summary of convective therapies[21]:

	CONTINUOUS CONVECTIVE REMOVAL OF WASTE PRODUCTS
Primary Therapeutic Goal:	Solute removal and safe management of fluid volume
Primary Indications:	Rhabdomyolysis, compartment syndrome, sepsis, overdose, toxicity
Principle Used:	Convection
Therapy Characteristics:	■ Requires substitution solution with a buffer to drive convection ■ No dialysate solution ■ High blood flows ■ No compatible with citrate anticoagulation ■ Used to achieve solute removal (**small, medium and large sized** molecules) and fluid balance

Filtration ratio[17]

Filtration ratio (FR) becomes relevant when considering convection; it is the volume of plasma water removed from blood during hemofiltration and it is a very good indicator of clogging and latterly, clotting. Ideally, FR should remain below 20 – 25%, if it starts to exceed this, it's possible that the filter will clot, at 40% the haemofilter as well as the patient may start the destabilise.

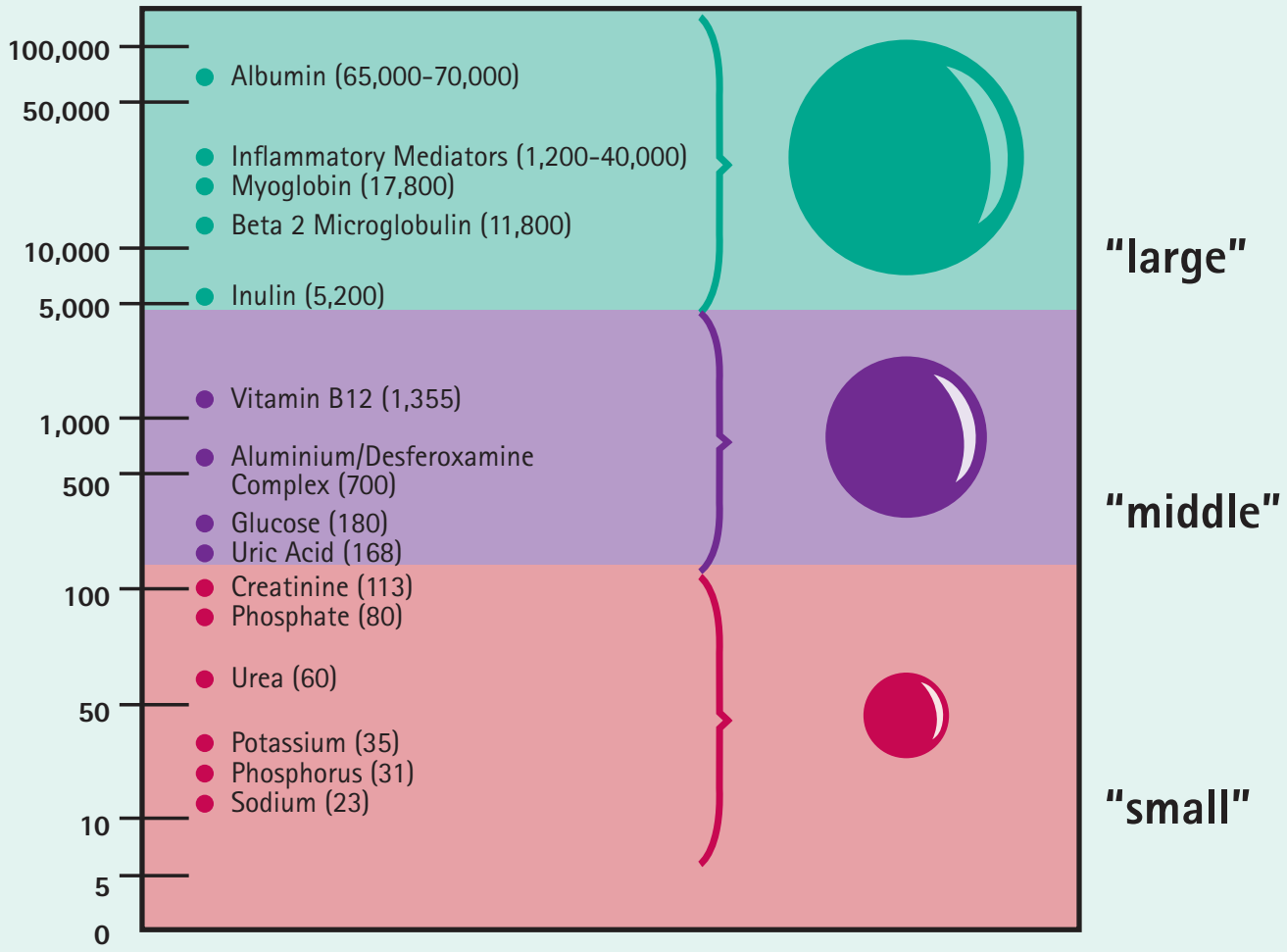
$$FR = \frac{Q_R/Q_D + UFR}{BF \times (1 - HCT)}$$

Substitution fluid/dialysate fluid flow

Ultrafiltrate AKA NFR

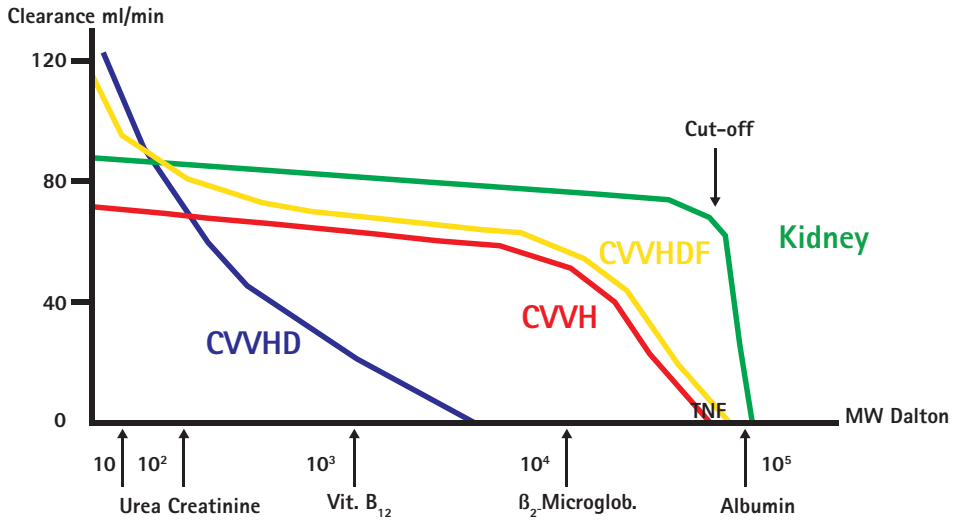
In order to reduce your filtration ratio, you can either reduce the volume of ultrafiltrate (dialysate, pre-filter substitution fluid or NFR) or increase your blood flow. The higher the substitution fluid/dialysate fluid flow rate, the higher clearance that is achieved, however, blood flow is a limiting factor in convection and therefore needs to be comparatively high for effective and sustained use, consequently, good vascular access is key.

MOLECULAR WEIGHTS[21]



Summary of diffusive therapies

MOLECULE CLEARANCE COMPARISON IN THE VARIOUS THERAPIES[18]



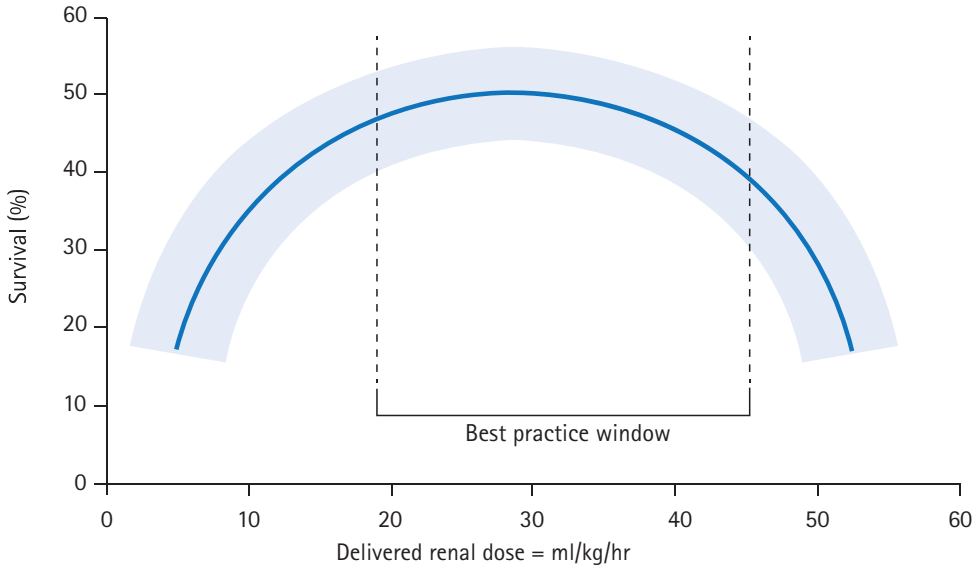
Renal Dose

According to KDIGO, the ideal renal dose is 25-35ml/kg/hr[5,19]

Renal dose = effluent dose e.g. dialysate/pre-filter substitution fluid + NFR (ultrafiltrate)

- Set renal dose - prescribed effluent dose based on flow rates + NFR
- Real renal dose - delivered effluent dose on average over the course of the treatment (will take into account any changes to renal dose and any downtime)
- Target renal dose - effluent dose the OMNI is trying to achieve in order to compensate for any downtime to achieve 98% accuracy of set renal dose

RELATIONSHIP BETWEEN DELIVERED CRRT INTENSITY AND SURVIVAL IN CRITICALLY ILL PATIENTS WITH ACUTE KIDNEY INJURY[20]



Summary

- CRRT is an essential therapy option for those with AKI in ICU
- There is a right time for initiation of CRRT to consider, however this is dependent on a number of factors
- CRRT main goals are removal of waste products, restoration of acid/base balance, and correction of fluid and electrolyte abnormalities
- There are different principles and modes to consider and essential to understand the differences in each
- SCUF for fluid removal
- CVVH for removal of small, mid and large molecules
- CVVHD for removal of small and mid molecules
- CVVHDF for removal of small and mid molecules

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Notes

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