Worcestershire Acute Hospitals NHS Trust

Guideline for Renal Replacement Therapy within Worcestershire Critical Care Units

Key Document code:	WAHT-KD-022
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Approved by:	*Intensive Care Forum*
Date of Approval:	20th October 2021
Date of review:	20th October 2024

Key Amendments

Date	Amendment	Approved by
8th October 2019	Document extended with no changes as part of Disease Management section in critical care	Dr Nick Cowley/ Dr Andy Burtenshaw
12th October 2021	1. Incorporation of Regional Citrate Anticoagulation guidelines into Renal Replacement Therapy guidelines 2. ST150 filtration sets as standard set choice 3. Removal of haemolactol as a RRT fluid option 4. Creation of quick reference guides (Appendix J & K) 5. Revised to match software found on new Prismax machines	Dr Andy Burtenshaw / Dr Janos Mayer/ ICF

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INTRODUCTION

Acute Kidney Injury (AKI) may be broadly described as a rapid loss (days to weeks) of the functional ability of the kidneys to excrete the waste products of metabolism and to maintain fluid, electrolyte and acid-base homeostasis.

The incidence of AKI is 10–25% of critical care patients, with Renal Replacement Therapy (RRT) being required for approximately 3-5% of admissions.[1]

Patients in whom AKI is diagnosed, particularly those requiring RRT, have an increased mortality. In a large cohort study evaluating the RIFLE criteria for AKI (not limited to the critical care population) the mortality rates for Class R, Class I and Class F AKI were 8.8%, 11.4% and 26.3% respectively compared to 5.5% for patients without AKI.2

Table 1: Risk, Injury, Failure, Loss, and End-stage kidney injury (RIFLE) classification:

Class		Glomerular filtration rate criteria	Urine output criteria
R	Risk	Serum creatinine × 1.5	< 0.5 ml/kg/hour × 6 hours
I	Injury	Serum creatinine × 2	< 0.5 ml/kg/hour × 12 hours
F	Failure	Serum creatinine × 3, or serum creatinine ≥ 300 μmol/L with an acute rise > 40 μmol/L	< 0.3 ml/kg/hour × 24 hours, or anuria × 12 hours
L	Loss	Persistent acute renal failure = complete loss of kidney function > 4 weeks	
E	End-stage kidney disease	End-stage kidney disease > 3 months	

This document is a guide to effective provision of CRRT (Continuous Renal Replacement therapy) within the WAHT critical care directorate using the Baxter Prismax system for those patients who are considered likely to benefit. This includes indications for RRT. However, suitability of individual patients for advanced organ support, including RRT, is beyond the scope of this guideline.

Much of the information contained within the guideline is supplemented by more detailed explanation within the appendices.

INDICATIONS FOR RRT

The most common indications for commencing RRT are:

- Metabolic acidosis (Clinician directed threshold for therapy)
- Hyperkalaemia [K^+] > 6.5 mmol.L^{-1} (unresponsive to medical management or in the context of established AKI)
- Severe fluid overload adversely affecting organ function and unresponsive to medical management (e.g. pulmonary oedema)
- Symptomatic uraemia (e.g. vomiting, seizures, pericarditis)
- Anuria / oliguria anticipated to progress to one of the above four scenarios in spite of medical management
- As a potential adjunct in severe sepsis/Septic shock
- Removal of toxins following overdose

[1] Standards and Recommendations for the Provision of Renal Replacement Therapy on Intensive Care Units in the United Kingdom. *Intensive Care Society.* January 2009

CHOICE OF ANTICOAGULATION

The choice of anticoagulation will determine the setup for RRT and is therefore the primary decision that must be made once the decision to initiate RRT has been made. In the majority of cases, RCA will be the anticoagulation mode of choice.

RRT may be conducted using any of the following options for minimising the risk of thrombus formation within the extracorporeal circuit:

DEFAULT	
Regional Citrate Anticoagulation (RCA)	See Section A
ALTERNATIVE ANTICOAGULATION OPTIONS	
Heparin Epoprostenol Anticoagulant free	See Section B

Regional Citrate Anticoagulation (RCA) is the mode of choice.

Indications for heparinisation include:

1. Patients already on unfractionated heparin for other indications (e.g. concurrent management of a mitral valve replacement or heparinisation to facilitate $ECCO_2R$)
2. Patients in whom Citrate is contra-indicated (see section A)_and in whom heparin anticoagulation is considered safe.

Epoprostenol or anticoagulant free RRT is only indicated in the context of patients in whom both citrate and heparin anticoagulation are contra-indicated.

VASCULAR ACCESS

The first choice for vascath length should be:

Internal Jugular 15cm
Subclavian 20 cm
Femoral 20 cm (or 25 cm in selected cases where the benefit of access to the inferior vena cava rather than femoral / iliac vein is anticipated to overcome additional resistance of a longer line)

As a general guide, the largest gauge cannula possible should be inserted to permit high blood flow rates. High blood flow rates help reduce filter clotting.

All vascular devices must be inserted using the guidance encompassed in the 'Matching Michigan' initiative.

Triple lumen Vascaths are the access of choice for Regional Citrate Anticoagulation as they provide a lumen through which to return calcium chloride, which can otherwise be administered via a separate CVC.

SECTION A – RRT SETUP USING REGIONAL CITRATE ANTICOAGULATION

OVERVIEW OF RCA

The use of extra-corporeal circuits, such as those used for renal replacement therapy, frequently require the use of anticoagulation to prevent clotting in the circuit and thus prolong filter life and reduce the amount of patient blood lost due to discarded circuits.

Historically heparin has been the drug of choice for anticoagulation in these patients which, although relatively simple to administer and titrate, has a number of drawbacks. These include the need for systemic anticoagulation (i.e. anticoagulation of the patient as well as the circuit) and the associated risk of bleeding. Systemic anticoagulation may be contraindicated in those who have recently undergone surgical intervention, or have other haemorrhage risks such as those who have had a recent intracerebral bleed. Heparin is ineffective in patients with anti-Thrombin III deficiency and it can be difficult to monitor efficacy in patients with coagulopathy. Furthermore, heparin induced thrombocytopenia is not infrequently suspected in the critical care population and this can preclude the use of heparin whilst awaiting confirmatory tests.

Citrate has been show to prolong circuit life and reduce patient blood transfusion requirements relative to heparinisation.

Citrate is used as regional anticoagulation which means that the anticoagulation effect can be reversed before the blood is returned to the patient. This means it can be used in situations where heparin may previously have been contraindicated.

The use of citrate as an anticoagulant also has some disadvantages. It is a more complex system to set up and monitor, and requires the use of different dialysate and replacement fluids from those used with heparin, epoprostenol or when providing anticoagulant free RRT (See section B). It also has the potential to cause metabolic disturbances and hypocalcaemia although these can be monitored and managed.

SUMMARY CHARACTERISTICS OF RCA

Ionised calcium is the active form of calcium in the blood and is a key requirement of the clotting process.

Citrate binds to ionised calcium to prevent clotting.

Using the pre-blood pump of the PrisMax™ allows citrate to be added to the patient's blood as soon as it enters the extracorporeal line.

The citrate chelates the calcium (and other divalent cations, principally Mg^{2+}) to result in a calcium-citrate (or other cation-citrate) complex.

Adjustment of the citrate infusion is made to maintain a very low ionised calcium level in the blood entering the filter. This is measured as a post-filter ionised calcium level as this is much easier in practice than sampling pre-filter.

The calcium-citrate complexes are principally removed in the dialysis fluid and the calcium is replaced via a systemic calcium infusion to maintain normal ionised calcium levels in the patient.

The calcium replacement is given via a central line or a dedicated lumen of a triple lumen vascath and not via the dialysis line to prevent recirculation and thus the need for higher doses of citrate.

The small amount of citrate and calcium-citrate complex that returns to the patient is metabolised by the liver and is converted to bicarbonate.

If more citrate returns to the patient than the liver can metabolise (this is fairly rare) then it will build up in the circulation, causing chelation of the systemic calcium and a metabolic acidosis. This can be monitored and managed.

Maintenance of the systemic ionised calcium between 1.0 and 1.3 is paramount to prevent hypocalcaemia.

Hypocalcaemia can lead to cardiac arrhythmias, hypotension, and cardiac arrest.

Monitoring for citrate toxicity (using the total calcium:ionised calcium ratio) and maintenance of normal electrolyte levels (ionised calcium, magnesium) are fundamental to the successful and safe use of citrate anticoagulation.

BACKGROUND INFORMATION - CITRATE

Citrate refers to the chemical compound citric acid and its salts. These citrate molecules chelate (bind to) calcium and render it inactive in the blood.

Calcium is a fundamental component of the human clotting process and inactivation by the addition of citrate results in profound anticoagulation.

Citrate is added to the patient's blood before it passes through the extra-corporeal circuit thus preventing clotting.

To avoid full anticoagulation of the patient additional calcium must be administered to reverse the systemic anticoagulation effect.

Some of the citrate molecules with their bound calcium are filtered out during renal replacement therapy; the rest is returned to the patient where it is metabolised by the liver to produce bicarbonate and release its calcium back into the blood. Consequently, severe liver failure is one of the contraindications to use of citrate.

Dialysate and pre-dilution fluids should be calcium free so that they do not reverse the effects of the citrate before the blood has returned to the patient.

Citrate can also bind magnesium, so serum magnesium levels must be monitored. Magnesium is also a component of both the dialysis and replacement fluids.

BACKGROUND INFORMATION - CALCIUM

Knowledge of calcium physiology is important to understanding the monitoring and replacement of body calcium levels.

As well as its involvement in the clotting system calcium is also a vital component of many other physiological systems. These include cardiac function, blood pressure regulation, nervous system control and bone and muscle function. Maintenance of a normal blood calcium level when using citrate is paramount to preserve the activity of these vital body systems.

The majority of calcium in the human body is found in bone. Calcium can be absorbed and resorbed from bone under hormonal control; however, it is only the calcium in the blood which we can measure using blood tests and which we monitor during citrate anticoagulation.

Calcium in the blood is found as 'free' ionised calcium, calcium bound to blood proteins (predominantly albumin), and calcium bound to other complexes (including citrate) in the blood. These three fractions make up the total calcium as measured on a laboratory blood test.

Although there is equilibrium between these three fractions it is the ionised calcium which takes part in clotting and which the citrate will bind to.

Ionised calcium is measured on a near patient testing blood gas analyser.

CITRATE TOXICITY

Patients with liver failure may be unable to metabolise the citrate-calcium complexes resulting in a rise in the plasma citrate level.

As citrate is an anion (negatively charged), accumulation can lead to a raised anion gap metabolic acidosis.

A rise in the total calcium in the absence of a rise in the ionized calcium as an indication that the calcium-citrate complex is accumulating.

Measuring the total calcium to ionised calcium ratio can help identify accumulation of citrate.

A total calcium:ionised calcium ratio of > 2.5 suggests citrate toxicity.

Because citrate returning to the patient is metabolised to bicarbonate by the liver, some patients may develop a metabolic alkalosis as each citrate molecule produces three bicarbonate molecules.

Because the citrate infusion fluid contains a moderately high dose of sodium, hypernatremia may develop.

Using dialysate fluid (i.e. CVVHD/CVVHDF) can reduce the risk of hypernatremia and metabolic alkalosis.

CALCIUM REPLACEMENT

Calcium replacement is administered via a central venous line or a dedicated line of the triple lumen vascath. It should not be delivered through the return port of the renal replacement therapy catheter as this can lead to recirculation of the calcium and a need for higher citrate infusion rates.

Calcium should always be replaced via a central vein and not peripherally as it is a potent cause of extravasation injury.

Normal systemic ionised calcium as measured on a blood gas analyser should be between 1.0 and 1.3mmol/L

The ionised calcium within the renal replacement circuit should be kept <0.35mmol/L to ensure adequate anticoagulation.

INDICATIONS FOR RCA

Regional citrate will be the default anticoagulation for patients requiring renal replacement therapy within the intensive care, unless there is a contraindication or senior medical staff have specified otherwise.

CONTRAINDICATIONS

Contraindications include:

1. Severe liver failure – patients with severe liver failure may be unable to metabolise citrate and thus have a high risk of developing citrate toxicity.
2. Citrate intolerance or allergy.
3. Patients with an ionised calcium <0.8 – this must be corrected to >1.0 prior to initiation of citrate

PREPARATION

EQUIPMENT

Table 1 lists the equipment required in order to set up the PrisMax™ for regional citrate anticoagulation:

CRRT Prescription	2 x 1L 0.9% sodium chloride
1 x Prismaflex™ ST150 filter	2 x 21g needles for sampling
1 x 5L Prism0cal	1 x 50ml BD syringe
1 x 5L Phoxilium	3 x 10ml ampoules of Calcium Chloride 10%
1 x 5L bag of Prismocitrate 18/0	1 x Prismaflex™ circuit

SET-UP

Prepare the calcium replacement by drawing up 30mls of 10% Calcium Chloride (30mmol) into a 50ml syringe and make up to 50mls using 0.9% sodium chloride giving a final concentration of 0.6mmol/ml.

Set up the PrisMax™ machine using the “New Patient” setting.

Select CCVHDF protocol.

Choose “Citrate-Calcium via Prismaflex™ syringe pump” as the anticoagulation method.

Ensure that the correct fluid bags are chosen and placed on the correct scales. This should be checked by another member of staff also trained in providing citrate anticoagulation.

- *Prismocitrate 18/0* on the **white** scales connected to the **pre-blood pump**.
- *Prism0cal B22* on the **green** scales connected to the **dialysate circuit**.
- *Phoxilium* on the **purple** scales connected to the **replacement fluid circuit**.

Connect the calcium replacement syringe previously prepared to the Prismax™ syringe pump.

Prime the circuit using 0.9% sodium chloride and heparin.

5000units Heparin should be added to the first 1000ml bag of 0.9% sodium chloride, subsequent bags do not need the addition of heparin. This surface treats the circuit and uses the second litre of saline to clear any free heparin.

The filters circuits have a coating of polyethylimine which has a positive charge and therefore heparin sticks to the filter. Priming with heparin coats the filter so that any systemic heparin does not bind to the filter and thus it is easier to achieve the target APTT.

BLOOD FLOW RATE

During setup the system will ask you to set the "blood flow rate", "dialysate" and "replacement" rates. This should be done based on the patient's weight using the parameters demonstrated in Table 2. Use actual body weight for most patients. For patients with a BMI exceeding 35 use adjusted body weight and review the effect of the therapy, adjusting the subsequent weight settings upon which CRRT is based according to the treatment effect.

Weight (kg)	Blood Flow (ml/min)	Dialysate (ml/hr)	Replacement post-filter (ml/hr)	Treatment dose obtained (ml/kg/hr)
<= 50	100	1000	200	37
51 -60	110	1100	400	37
61- 70	120	1200	500	35
71 - 80	130	1300	500	33
81 - 90	140	1400	500	31
91 - 100	150	1500	600	31
101 - 110	160	1600	700	30
111 - 120	170	1700	800	30
>= 121	180	1800	1000	30

CALCIUM REPLACEMENT

When prompted the starting citrate dose should be set to 3mmol/L

The initial calcium compensation should be set based on Table 3 and is dictated by the patient's initial ionised calcium from a blood gas sample.

Patients with an ionised calcium below 1.0mmol/L should receive 10mls calcium chloride 10% over 30mins via a central vein before initiation of citrate therapy.

Table 3:

Patient ionised calcium (mmol/L)	**Starting calcium compensation (%)**
Less than 1.00	Give calcium replacement and start at 110%
1.00 – 1.11	110%
1.12 – 1.30	100%
Greater than 1.30	90%

MONITORING

Monitoring will consist of three elements:

1. Patient total and ionised calcium levels
2. Filter ionised calcium level
3. Liver function, magnesium and phosphate levels

The frequency of testing is shown in Table 4.

Steady state sampling can commence once there have been no changes required to citrate or calcium doses for two consecutive hours.

The total calcium is not corrected for albumin when used to calculate the total calcium:ionised calcium ratio.
Table4:

Sample	Initially	Steady State
Filter ionised calcium	Hourly for 2 consecutive hours until stable	6 hourly
Patient ionised calcium	Hourly for 2 consecutive hours until stable	6 hourly
Total calcium (uncorrected)	6 hours after starting	Daily
Calcium ratio Total calcium/patient systemic ionised calcium Target ratio < 2.5	6 hours after starting	Daily and twice daily in significant liver dysfunction. Within one hour if changes made.
Magnesium	-	Daily
Phosphate	-	Daily
Liver function tests	-	Daily

In fulminant liver failure or severe shock states, the total and ionized calcium ratio should be monitored more frequently
.

ADJUSTMENTS

Based on the sampling from the patient use Table 5 to make adjustments to the dose of the citrate and calcium

		Filter ionised calcium		
		<0.25	0.25-0.5	>0.5
Patient ionised calcium	<1.0	Citrate dose decreased by 0.5mmol/L	Calcium compensation increased by 10%	Citrate dose increased by 0.5mmol/L **and** Calcium compensation increased by 10%
	1.0-1.3	Citrate dose decreased by 0.5mmol/L	No change required	Citrate dose increased by 0.5mmol/L

	>1.3	Calcium compensation decreased by 10% **and** citrate dose decreased by 0.5mmol/L	Calcium compensation decreased by 10%	Calcium compensation decreased by 10%

RESTARTING PATIENTS ON CITRATE REGIONAL ANTICOAGULATION AFTER A BREAK IN RRT DELIVERY

Restart using the citrate dose and calcium replacement rate from when treatment last finished rather than restarting with default settings and re-establishing a steady state.

TOTAL CALCIUM:IONISED CALCIUM RATIO AND MANAGEMENT OF CITRATE TOXICITY

Daily checks of the total calcium:ionised calcium ratio must be performed – this is to monitor for citrate toxicity. This will require a laboratory calcium, and a blood gas calcium to be taken at the same time.

To calculate the "Total calcium : Ionised calcium ratio", simply divide the total calcium level (uncorrected for albumin) by the patient's ionised calcium from a concurrent arterial blood gas sample.

The ratio should be <2.5. If this is the case then repeat the calculation again in 24 hours.

If the ratio is >2.5 this can be a sign of citrate toxicity and expert help should be sought from a consultant or senior registrar.

Actions:

1) Exclude low Patient Ionised Calcium as the cause of an elevated ratio

If at any stage the patient's systemic ionised calcium is <0.7mmol/L administer 10mls calcium chloride 10% to prevent physiological instability. This should be done as a priority and should be given via a central line as a slow bolus over 10 minutes. A drop in the patient's heart rate or blood pressure should always prompt assessment for hypocalcaemia and emergency calcium replacement if low.

2) Consider Citrate Toxicity

Possible signs of citrate toxicity include:

1. A total calcium:ionised calcium ratio >2.5,
2. worsening metabolic acidosis without alternative explanation (pH <7.35 or BE <-4) or
3. rising anion gap.

3) Consider causes of citrate build up from administration to breakdown. Potential causes include:

1. Excess citrate administration via RRT
2. Co-administration of exogenous additional citrate such as with blood transfusions
3. Developing or worsening liver dysfunction
4. Worsening hypoperfusion resulting in reduced citrate metabolism by muscles

Citrate toxicity should be managed in a step wise fashion:

1. Reduce citrate administration:
    a. Adjust the filter ionised calcium target to 0.4-0.5 mmol/L (aiming to ride the higher end of the target range) by reducing the citrate dose in 0.2mmol increments.

   b. Check a filter ionised calcium every 30 minute until the new 0.4-0.5 mmol/L target achieved. This may mean that filter life and RRT clearance are reduced.
   c. Remeasure the total calcium:ionised calcium ratio once the filter ionised calcium target of 0.4-0.5 mmol/L is achieved.
2. If the total calcium:ionised calcium ratio remains >2.5 despite the above measures consider any of the following actions:
   a. Double baseline dialysate flow (to increase citrate removal)
   b. Reduce blood pump speed by 10ml/min (this will further reduce the citrate dose administered at the expense of overall filtration dose)
   c. Stop citrate and use alternative anticoagulant or no anticoagulant.

N.B. If calcium replacement exceeds 150% then citrate toxicity should be excluded by checking a total calcium:ionised calcium ratio.

RISK OF VENOUS THROMBOEMBOLISM

Citrate anticoagulation does provide only localized anticoagulation for the filter. Assessment of the risk of venous thromboembolism is required in accordance with the Trust policy.

Dose of enoxaparin for pharmacological thromboprophylaxis is as per the trust thromboprophylaxis guidelines, noting that in most cases this will require dose reduction to 20 mg once daily subcutaneously due to reduced GFR.

Increased dose of thromboprophylaxis in case of COVID-19 and AKI: 40 mg enoxaparin once a day subcutaneously.

TROUBLESHOOTING

ADJUSTING CLEARANCE

To increase the clearance of solute the replacement fluid dose can be increased or settings can be adjusted to the next highest weight category as shown in Table 2. For example, if the patient weighs 84kg, use settings for the 91-100kg category.

To decrease clearance of solute the settings can be adjusted a lower weight category as shown in Table 2. For example, if the patient weighs 84kg, use settings for the 61-70kg category. This can be useful when providing RRT for stable ESRF patients, with a much lower weight selection providing adequate RRT with less resource consumption.

LOW BICARBONATE

If bicarbonate is consistently low the amount of citrate reaching the patient and thus metabolised to bicarbonate can be increased. This can be achieved either through reducing the dialysate dose or increasing the blood flow rate (and hence the citrate dose). Administration of systemic bicarbonate may also be considered.

N.B. Please ensure that the total calcium:ionised calcium ratio is <2.5 to exclude citrate toxicity before increasing the citrate dose received by the patient.

METABOLIC ACIDOSIS

Patients in renal failure commonly have a metabolic acidosis as part of their disease process; it is expected that after initiation of RRT and treatment of the cause of the renal failure that any pre-existing metabolic acidosis should improve.

Worsening metabolic acidosis whist on RRT should raise concern about citrate toxicity development

METABOLIC ALKALOSIS

Development of a metabolic alkalosis occurs if the patient is metabolising excess citrate as one citrate molecule is broken down into 3 bicarbonate molecules.

Management may involve either a reduction in the citrate dose (by reducing the blood pump speed in 10mls/min increments) or increasing clearance by doubling the baseline dialysate flow rate.

SECTION B – RRT SETUP USING SYSTEMIC ANTICOAGULATION OPTIONS

This section describes the setup for renal replacement therapy using either heparin or epoprostenol, or for running RRT with no anticoagulation.

The following section describes the setup and management for CRRT is any of these scenarios.

MACHINE SETUP

PRISMAX MODE CHOICE

The default setting for the PrisMax is CVVHDF, with the default dialysis flow set to zero. (This is essentially a CVVH filtration mode but permits a change to CVVHDF should this be required mid-cycle without the need to re-program.)

FLUID CHOICE

The default solution for RRT use is Prismasol 4. This is a bicarbonate buffered solution which also contains 4 mmol L^{-1} potassium. Lactate buffered solutions are no longer supported. Prismasol 4 is used for pre-blood pump, dialysis and post-blood pump purposes.

SELECTION OF FLUID "EXCHANGE RATE"

	Flow Rate	Comments
Standard	25ml/kg/hr	Appropriate in the majority of cases
High Flow	40 ml/kg/hr	For initial management of severe sepsis. Envisaged to be used for initial 24 hours only. Flows in excess of 40 ml/kg/hr are possible but not currently supported by evidence. They may be used in specific situations at the discretion of the critical care consultant. (For example to

		compensate for excessive filter down time)
Low flow	15 ml/kg/hr	For patients requiring medium to long term RRT once gross metabolic derangement resolved and not in the presence of severe sepsis. Intermittent use of standard regimen is an alternative.

Machine settings:

Standard (25 ml/kg/hr)	Pre-Blood Pump	8	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	17	ml/kg/hr
	Pre-post setting	Post	

High Flow (40 ml/kg/hr)	Pre-Blood Pump	13	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	27	ml/kg/hr
	Pre-post setting	Post	

Low Flow (15 ml/kg/hr)	Pre-Blood Pump	5	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	10	ml/kg/hr
	Pre-post setting	Post	

POTASSIUM REPLACEMENT

No potassium replacement is required since Prismasol 4 contains 4mmol/L potassium.

MACHINE SETUP

1. Select filter set

Standard filter set: **ST150**
Surface area approximately 1.5 m^2

Alternative filter set: **ST100**
Surface area approximately 0.9 m^2
Not routinely used but occasionally only these are available (e.g. during pandemics) They are best suited to smaller patients or patients with stable AKI and minimal metabolic clearance requirements.

Both these sets are Surface Treated to reduce the risk of anaphylactoid reaction with ACE-Inhibitors. They also bond heparin to the filter membrane which reduces the risk of filter clotting, promoting longer filter life or to aid anticoagulant free filtration.

2. After switching machine on enter the **patient ID**, **Weight** and Patient's **haematocrit**.
3. Select the therapy by choosing "**CRRT**", then "**CVVHDF**".
4. Choose the filter set (default is ST150).
5. Choose the anticoagulation option, in this case selecting "**Systemic**" for heparin anticoagulation or "**None**" is anticoagulant free CRRT is required. (N.B. Selected of "Systemic" even if no anticoagulation is to be used with the syringe filled with 0.9% saline allows heparin to be added in easily at a later stage without re-programming and is therefore

preferable. Standard herparin anticoagulation will require a 20 ml syringe containing 1000 units/ml.)

6. Select the "Thermax" heating system.
7. Follow on-screen instructions to connect the Priming bag, Pre-Blood Pump (PBP) bag, Dialysate bag, Replacement bag and Effluent bag.

Priming solutions:

The priming solution should be 1L 0.9% saline containing 5000 units heparin followed by a second bag of plain 0.9% saline. If treatment is planned without anticoagulation, priming with heparin should still take place to permit heparin bonding EXCEPT for patients with Heparin Induced Thrombocytopenia (HIT) for whom 1 L 0.9% saline should be used with either an ST100 or ST150 set.) This reduces extra-corporeal circuit thrombus formation without systemically anticoagulating the patient. It also reduces systemic heparin consumption in the case of the patient who is already systemically anticoagulated with unfractionated heparin for other indications and thus prevents unnecessary variance in APPT ratios.

N.B. For standard CVVH there will be no dialysate flow. A 1 L bag of 0.9% saline can be attached in place of a dialysate bag. (This can be changed later if dialysate is later required as the machine will have been set in CVVHDF mode and simply needs the dialysate fluid attaching and the rate turning up.)

8. Install anticoagulant syringe as per on screen instructions and press "Prime".

N.B. It is necessary to connect the machine to the patient within 30 minutes of priming. This is because ethylene oxide, which is used to sterilise the filter, may leach into the primed solution over time and can result in serious hypotension if distributed systemically. Delays longer than 30 minutes can be managed by re-priming the filter set within 30 minutes of patient connection using 0.9% saline (no further heparin required).

9. Unless specifically indicated by the ITU consultant all patients will require blood warming using the Thermax system.
10. Check patency of vascular access cannula. This can be achieved by aspirating 5ml of blood and injecting onto gauze. If any clots are present repeat this process until no clots are evident. Attempt to aspirate 20 ml blood from each port. If this cannot be achieved in less than 6 seconds then adequate blood flow will not be achieved and the filter set will be at high risk of clotting. (This is equivalent to 200ml/min pump speed) The medical team should appropriately manage the vascular access until such flows can be achieved (on the access line as a minimum) before connection of the filter. Flush lines with 0.9% saline after patency check.
11. Confirm flow settings

Note: The fluid loss/gain limit should read approximately 400 ml (may vary slightly by weight entered) and the access range should be set to "-ve". (+ve refers to situations where high pressure access is encountered such as A-V fistulae (including Novalung circuit) or "piggy-backing" onto CPB or ECMO pumps.)

12. Enter flow settings. *Initial* settings should read:

Blood Flow	80-100	ml/minute
PBP	0	ml/minute
Dialysate	0	ml/minute
Replacement	0	ml/minute
Pre-Post		Post
Patient Fluid removal	0	ml/minute

13. Set Anticoagulation syringe rate as per protocol, or set 0 ml/hr for no anticoagulation.
14. Review prescription and confirm settings

15. Administer bolus dose of heparin to patient as per anticoagulant protocol
16. Connect patient and press “Start”
17. Blood flow rate should be increased slowly to start with, increasing to as high as possible once the blood has visibly circulated entirely through the filter set and back to the patient (dependent upon haemodynamic stability and vascular access) up to a maximum of 450 ml/minute (for ST150 set) or 400 ml/minute (for an ST100). Access pressure should ideally be at least -100mmHg (absolute limit -250 mmHg)
18. In cases of severe cardiovascular instability, very slow initiation of blood flows (e.g 30-50 ml/min) until blood has visibly circulated throughout the entire extracorporeal circuit may limit further hypotension.
19. Once blood flow has been increased to at least 120 ml/minute, adjust PBP, Dialysate, Replacement and patient fluid removal settings according to the prescription.

N.B. Fluid removal should be prescribed as the total amount of fluid the patient is to lose per hour after adjustment for input and output. It will therefore require recalculation and adjustment on an hourly basis.

N.B. A minimum fluid removal rate of 10 ml/hour is advised to allow for minor fluctuations in the scales.

N.B. UFR post % of BFR (Ultrafiltration Rate as % of Blood Flow Rate) should not exceed 25% as this reflects the degree of concentration of blood in the filter and correlates with the likelihood of filter clot formation. If it is greater than 25% either increase the blood flow rate or increase the proportion of fluid replacement given as pre-dilution. (i.e. increase PBP, decrease replacement)

HEPARIN ANTICOAGULATION

A bolus of heparin is required prior to commencement of filtration. A dose of 40 units/kg is usually appropriate but can be reduced according to clinical requirement (e.g No anticoagulation or 10 units/kg in the context of pre-existing coagulopathy or risk of clinically significant haemorrhage, at the clinician’s discretion)

Unfractionated heparin (UFH) infusions should be made up to the trust-wide standardised concentration of 1000 units/ml by diluting 20,000 units into 20ml 0.9% saline.

The UFH infusion should then be commenced at a rate of 10 units/kg/hr and an initial APTT ratio checked four hours after commencing therapy. The infusion rate should then be adjusted to aim for an APTT ratio of 1.5-2.5 which will usually require a UFH infusion rate of between 5-20 units/kg/hr. Audit of filter set duration identified APTT ratio as the only statistically significant determinant of premature loss of filter sets. Therefore, in the absence of a perceived additional risk, maintenance of the APTT range between 2.0 and 2.5 should be preferentially sought.

Weight	Bolus Dose (40 U/Kg)	Initial Rate (10 U/Kg/Hr)	
Kg	Units	Units/hr	ml/hr
50	2000	500	0.5
55	2200	500	0.5
60	2400	600	0.6
65	2600	600	0.6
70	2800	700	0.7
75	3000	700	0.7
80	3200	800	0.8
85	3400	800	0.8
90	3600	900	0.9
95	3800	900	0.9
100	4000	1000	1

(the syringe pump in the Prismax can be set to only the nearest 0.1 ml/hr)

APTT ratio should then be checked at a maximum interval of every 8 hours.

(N.B. A degree of clinical interpretation may sometimes be necessary in adjusting the infusion rate or target range for some patients. Patients may exhibit heparin resistance due to low antithrombin levels and heparin binding to drugs and acute phase proteins and it is clear that APTT ratios do not always accurately reflect the anticoagulant effect of UFH.)

OTHER ANTICOAGULATION STRATEGIES

LOW MOLECULAR WEIGHT HEPARIN (LMWH)

LMWHs are not routinely used in our critical care units for two reasons. Firstly their effect is not readily measured and secondly their action is longer and less easy to reverse. Interest in LMWH for CRRT anticoagulation has been superseded by the widespread adoption of regional citrate anticoagulation.

EPOPROSTENOL

Epoprostenol (a prostacyclin analogue) is a naturally occurring potent inhibitor of platelet aggregation which prevents/reduces clot formation. Its use has also been largely superseded by regional citrate anticoagulation and it should be reserved for use in patients in whom both heparin and regional citrate anticoagulation are contra-indicated and in whom anticoagulant free CRRT has either failed or is clinically ill-advised. This should only be utilised following discussion with the consultant on call.

Epoprostenol is more expensive than heparin and is also a potent vasodilator. This may result in hypotension (half life of hypotensive effect thought to be much shorter than the half life of anticoagulant effect) and has potential for causing hypoxia predominantly through amelioration of hypoxic pulmonary vasoconstriction.
The requirement for epoprostenol in CRRT is therefore very limited.

The trust Epoprostenol monograph should be consulted for dosage guidance.

NO ANTICOAGULATION

With RCA offering an effective means of avoiding systemic anticoagulation it is rarely necessary to run a filter with no anticoagulation at all.

Circumstances in which this might be considered could include:

1. INR ≥ 2.0, APTT ratio ≥ 2.0 or Platelet count < 60 x 10^3/mm^3, together with circumstances in which RCA is inadvisable (e.g. concerns over the potential for citrate toxicity). This may include patients who are already actively anticoagulated for other conditions using unfractionated heparin.
2. High risk of clinically significant haemorrhage or less than 12 hours post-op in patients for whom RCA is also contra-indicated.

Maintenance of a clot free filter in these circumstances may be encouraged by the use of a larger venous access catheter, higher blood flow rates and the use of predominantly, or exclusively, pre-dilutional haemofiltration.

MANAGEMENT OF SODIUM DISORDERS DURING CONTINUOUS HEMOFILTRATION

In rare cases, patients with chronic hyponatraemia or hypernatraemia can develop acute kidney failure which needs renal replacement therapy. Rapid correction of sodium concentration can cause serious and life-changing complications e.g. pontine myelinosis or cerebral oedema. It is important to avoid quick correction of the serum sodium level to avoid complications. Advanced dialysis machines which are used for intermittent dialysis do have their own water supply and the desired sodium level can be dialled on the machine. The Prismax machines on the intensive care units do not have this option and the replacement fluid has a fixed sodium content – 140 mmol/l. This may result in patient sodium level quickly equilibrating towards 140 mmol/l if we use these advanced intensive care dialysis machines. Rapid overcorrection of sodium plasma concentration is to be avoided and modifying the replacement fluid may be required.

Principles:

1. Modification of the replacement fluid is dangerous when using regional citrate anticoagulation. This guidance **MUST NOT use with regional citrate anticoagulation**.
2. It can be used with conventional CVVH or CVVHDF modalities.
3. The ICU pharmacist on duty must be informed before the change but the therapy can be started if the patient is in life-threatening condition needing emergency renal replacement therapy.
4. It is a high-risk procedure and needs to be discussed in the daily safety brief.
5. Clear documentation is needed for the thresholds when urgent medical review is needed
6. Other electrolytes may be affected and frequent monitoring is important. The patient might require additional electrolyte supplementation
7. If the change of the sodium is too much and quick: the sodium content of the replacement fluid might be changed for a lower or higher sodium concentration. Decreasing the filtration rate can also slow down the change of the serum sodium if it is accepted in the clinical situation.
8. Escalation to the consultant in charge is necessary if any question or problem arises during the use of this protocol.

ACUTE KIDNEY INJURY AND HYPERNATRAEMIA (NA+ >155 MMOL/L)

Hypernatraemia is rarely caused by sodium excess. It is mainly due to severe dehydration which is due to decreased water intake or loss of hypotonic fluid. The mainstay of the therapy is to replace the free water. These patients often do no need renal replacement therapy. When this is insufficient and hemofiltration is employed, the sodium content of the replacement fluid will need to be increased by adding 30% sodium chloride in order to reduce the rate of change of the plasma sodium concentration. The aim is to keep the serum sodium reduction to less than 9 mmol/l over the first 24 hours and no more than 12 mmol/l drop over the first 48 hours.

If the serum sodium decreases more than 2 mmol/l in 6hours:

- Decrease the filtration rate if it is clinically possible
- Adjust the replacement fluid sodium content by adding 30% sodium chloride as per the table below

Table 1. Effect of adding different volumes of 30% NaCl to replacement fluid

Volume of 30% NaCl added	Nil	5 ml (=25 mmol Na^+)	10 ml (=50 mmol Na^+)	15 ml (=75 mmol Na^+)	20 ml (=100 mmol Na^+)
Final Na^+ concentration in replacement fluid	140 mmol/l	145 mmol/l	150 mmol/l	155 mmol/l	160 mmol/l

Effect of adding different volumes of 30% NaCl (≈5 mmol/ml) to a 5 l bag of replacement fluid containing a Na^+ concentration of 140 mmol/l.

ACUTE KIDNEY INJURY AND HYPONATRAEMIA (NA+ <125 MMOL/L)

Hyponatraemia is more common than hypernatraemia. Too rapid sodium concentration correction can cause pontine myelinosis and permanent paralysis. If CVVH is needed, the sodium concentration of the replacement fluid should be reduced by adding sterile water in order to reduce the concentration gradient, and thus the rate of change. Follow the hyponatraemia protocol to achieve slow correction. The level of other electrolytes will be affected by dilution so monitoring and replacement of electrolytes is vital to avoid complications.

If the serum sodium increases more than 2 mmol/l in 6hours:

- Decrease the filtration rate if it is clinically possible

- Adjust the replacement fluid sodium content by adding sterile water to achieve a lower sodium concentration in the bag

Table 2. Effect of adding different volumes of water to replacement fluid

Volume of water added (ml)	Final volume of diluted replacement fluid (l)	$[Na^+]$ in diluted replacement fluid (mmol/l)	$[HCO_3^-]$ in diluted replacement fluid (mmol/l)	$[K^+]$ in diluted replacement fluid containing 4 mmol/l
Nil	5	140	35	4.0
150	5.15	136	34	3.9
250	5.25	133	33	3.8
350	5.35	131	33	3.7
500	5.5	127	32	3.6
750	5.75	122	30	3.5
1,000	6.0	117	29	3.3
1,250	6.25	112	28	3.2

Effect of adding different volumes of water to a 5 l bag of replacement fluid with a Na^+ concentration of 140 mmol/l. $[Na^+]$, sodium concentration; $[HCO_3^-]$, bicarbonate concentration; $[K^+]$, potassium concentration.

References:

1. Ostermann M, *et al*.: Management of sodium disorders during continuous haemofiltration. *Critical Care* 2010, 14:418
2. Mitchell H. Rosner and Michael J. Connor: Management of Severe Hyponatremia with Continuous Renal Replacement Therapies, Clin J Am Soc Nephrol 13: 787–789, 2018
3. François Paquette, Rémi Goupil, François Madore, Stéphan Troyanov and Josée Bouchard: Continuous venovenous hemofiltration using customized replacement fluid for acute kidney injury with severe hypernatremia, Clinical Kidney Journal, 2016, vol. 9, no. 4, 540–542
4. Ostermann at al: Correction of Hyper- and Hyponatraemia during Continuous Renal Replacement Therapy, DOI: 10.1159/000369347

PRESSURE ALARMS

PRESSURE DROP AND TRANSMEMBRANE PRESSURE

Pressure Drop “PD” is the pressure difference in the blood compartment across the membrane. It reflects the ease of passage of blood through the filter and increases significantly in the presence of blood clots. A pressure drop of 150-200 mmHg suggests a clotting filter which will soon require change. This situation is rarely resolved with adjustments to the anticoagulant strategy but may inform subsequent decision making if the filter set does need to be replaced.

Transmembrane pressure “TMP” reflects the pressure across the filter membrane between the blood and filtrate compartments. In contrast to pressure drop, TMP reflects the degree of “clogging” of membrane pores. Once TMP exceeds 300-350 mmHg consideration should be given to changing the filter set, particularly in the context of severe sepsis where removal of inflammatory mediator molecules may have become saturated. This has little to do with anticoagulant efficacy.

Ideally, in the absence of evidence of filter “clotting”, a clogged filter should be washed back before changing the set in order for the patient to avoid unnecessary blood loss.

PRISMAX PRESSURE RANGES

Normal ranges for pressure measurement are shown in the table below. This information is also written on a card on the side of the machine and on the quick reference chart.

	Normal	Limit
Access	-30 to -150	-250
Filter	+80 to +400	+500
Effluent	+50 to -150	
Return	+50 to +250	+350

(All pressures measured in mmHg)

REFERRAL TO NEPHROLOGY

The nephrology team should be notified of all patients with acute kidney injury requiring renal replacement therapy within the critical care unit.

END OF TREATMENT

Please remember to download the treatment data to the memory card at the end of each treatment whether stopped temporarily or permanently.

Unless clotted, the filter set should be washed back using 250 ml 0.9% saline and following the on-screen instructions.

Unless specifically contra-indicated, the vascath should be “hep-locked” with 1000 units/ml heparin according to the lumen volume printed on the vascath in order to limit thrombus formation and blocked lumens during inactivity.

APPENDIX A: LIST OF ABBREVIATIONS

ADQI	Acute Dialysis Quality Initiative
AKI	Acute Kidney Injury
ARF	Acute Renal Failure
CRRT	Continuous Renal Replacement Therapy
CVVH	Continuous Veno-Venous HaemoFiltration
CVVHD	Continuous Veno-Venous HaemoDialysis
CVVHDF	Continuous Veno-Venous HaemoDiaFiltration
HIT	Heparin Induced Thrombocytopenia
HVHF	High Volume HaemoFiltration
LMWH	Low Molecular Weight Heparin
RCA	Regional Citrate Anticoagulation
RIFLE	Risk, Injury, Failure, Loss, End-Stage Kidney Disease
RRT	Renal Replacement Therapy
SCUF	Slow Continuous UltraFiltration
TPE	Total Plasma Exchange
UFH	UnFractionated Heparin

APPENDIX B: DEFINITION OF ACUTE KIDNEY INJURY

In 2006 the Acute Dialysis Quality Initiative published a new definition and classification scheme for renal failure known as RIFLE.[2] RIFLE describes five stages of renal impairment as shown in table 1. The RIFLE classification was introduced in recognition that there was no uniform means of classifying different degrees of renal impairment and that this made comparison and application of research findings to patient populations difficult.

Table 1: Risk, Injury, Failure, Loss, and End-stage kidney injury (RIFLE) classification:

Class	Glomerular filtration rate criteria	Urine output criteria
Risk	Serum creatinine × 1.5	< 0.5 ml/kg/hour × 6 hours
Injury	Serum creatinine × 2	< 0.5 ml/kg/hour × 12 hours
Failure	Serum creatinine × 3, or serum creatinine ≥ 300 μmol/L with an acute rise > 40 μmol/L	< 0.3 ml/kg/hour × 24 hours, or anuria × 12 hours
Loss	Persistent acute renal failure = complete loss of kidney function > 4 weeks	
End-stage kidney disease	End-stage kidney disease > 3 months	

RIFLE class is determined based on the worst of either glomerular filtration criteria or urine output criteria. Glomerular filtration criteria are calculated as an increase of serum creatinine above the baseline serum creatinine level. Acute kidney injury should be both abrupt (within 1–7 days) and sustained (more than 24 hours). When the baseline serum creatinine is not known and patients are without a history of chronic kidney insufficiency, it is recommend to calculate a baseline serum creatinine using the Modification of Diet in Renal Disease equation for assessment of kidney function, assuming a glomerular filtration rate of 75 ml/min/1.73 m^2. When the baseline serum creatinine is elevated, an abrupt rise of at least 40 μmol/L to more than 300 μmol/L is all that is required to achieve class Failure.2

[2] RIFLE criteria for acute kidney injury are associated with hospital

mortality in critically ill patients: a cohort analysis. Hoste EAJ, Clermont G, Kersten A et al. Critical Care 2006, 10:R73

APPENDIX C: INDICATIONS FOR RRT

Indications for commencing RRT include:[3]

- Metabolic acidosis
- Hyperkalaemia [K^+] > 6.5 mmol.L^{-1} (unresponsive to medical management or in the context of established AKI)
- Severe fluid overload adversely affecting organ function and unresponsive to medical management (e.g. pulmonary oedema)
- Symptomatic uraemia (e.g. vomiting, seizures, pericarditis)

Other factors which may provoke consideration of RRT but are not absolute indications include:

- Anuria / oliguria anticipated to progress to established AKI and not responding to medical management (despite not yet reaching above triggers)
- MODS, SIRS or severe sepsis/septic shock
- Severe dysnatraemia ([Na^+] < 115 mmol.L^{-1} or [Na^+] > 160 mmol.L^{-1})
- Drug intoxication amenable to RRT
- Severe hyperthermia refractory to other methods of treatment
- Severe hypothermia refractory to other methods of treatment (limited benefit)

REFERENCES

Standards and Recommendations for the Provision of Renal Replacement Therapy on Intensive Care Units in the United Kingdom. *Intensive Care Society.* January 2009

[1] RIFLE criteria for acute kidney injury are associated with hospital mortality in critically ill patients: a cohort analysis. Hoste EAJ, Clermont G, Kersten A et al. Critical Care 2006, 10:R73

[1] Core Topics in Critical Care Medicine. Cambridge University Press. (April 2010)

[3] Core Topics in Critical Care Medicine. Cambridge University Press. (April 2010)

APPENDIX D: COMPLICATIONS OF RRT

The following are potential complications associated with RRT:

1. Central venous access associated complications:
   - 1.1. Haematoma formation / arterial puncture
   - 1.2. Pneumothorax
   - 1.3. Line infection / sepsis
   - 1.4. Line thrombosis or thromboembolism
   - 1.5. Poor blood flow through cannula (cannula against vessel side wall or kinked)
   - 1.6. Line disconnection
   - 1.7. Air embolism
   - 1.8. Subclavian stenosis associated with subclavian lines
   - 1.9. Lower limb DVT may be associated with femoral venous catheterisation

2. Machine problems:
   - 2.1. Clotting of circuit
   - 2.2. Machine malfunction

3. Clinical problems:
   - 3.1. Allergic reactions
   - 3.2. Hypothermia (discomfort, shivering, coagulopathy & masking of pyrexia)
   - 3.3. Cardiovascular instability (particularly on initiation of therapy)
   - 3.4. Electrolyte imbalance / disequilibrium syndrome / hypophosphataemia.
   - 3.5. Citrate toxicity
   - 3.6. Hypocalcaemia or hypercalcaemia
   - 3.7. Hypomagnesaemia as a result of magnesium chelation
   - 3.8. Metabolic acidosis or alkalosis as a result of citrate toxicity
   - 3.9. Adverse effects of anticoagulation (e.g. gastrointestinal bleeding) including those associated with over or under anticoagulation.
   - 3.10. Unpredictable drug concentrations (e.g. effect upon antibiotic efficacy, effect on vasoactive drug concentrations)
   - 3.11. Nutrient losses
   - 3.12. Metabolic acidosis and lactate rise in situations where lactate cannot be adequately metabolised

APPENDIX I: TEMPERATURE MANAGEMENT

Extracorporeal blood flow exposes the patient's blood to room temperature and results in a cooling effect. An important complication of this is that pyrexia indicative of early sepsis can be easily masked. Indeed, many clinicians would regard normothermia in a patient receiving CRRT as indicative of masked pyrexia.

APPENDIX J: PRISMAX QUICK REFERENCE CHART (FOR SYSTEMIC ANTICOAGULATION)

Standard filter set: **ST150** Surface area of approximately 1.5 m^2

Initial pump settings:

Blood Flow	50-100	ml/minute
PBP	0	ml/minute
Dialysate	0	ml/minute
Replacement	0	ml/minute
Pre-Post		Post
Patient Fluid removal	0	ml/minute

Fluid "Exchange Rate" settings: (once blood flow > 150 ml/minute, preferably higher):

Standard (25 ml/kg/hr)	Pre-Blood Pump	8	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	17	ml/kg/hr
	Pre-post setting	Post	

High Flow (40 ml/kg/hr)	Pre-Blood Pump	13	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	27	ml/kg/hr
	Pre-post setting	Post	

Low Flow (15 ml/kg/hr)	Pre-Blood Pump	5	ml/kg/hr
	Dialysis	0	ml/kg/hr
	Replacement	10	ml/kg/hr
	Pre-post setting	Post	

Initial Heparin Anticoagulation:

Weight	Bolus Dose (40 U/Kg)	Initial Rate (10 U/Kg/Hr)	
Kg	Units	Units/hr	ml/hr
50	2000	500	0.5
55	2200	500	0.5
60	2400	600	0.6
65	2600	600	0.6
70	2800	700	0.7
75	3000	700	0.7
80	3200	800	0.8
85	3400	800	0.8
90	3600	900	0.9
95	3800	900	0.9
100	4000	1000	1

APPENDIX K: PRISMAX QUICK REF CHART (REGIONAL CITRATE ANTICOAGULATION)

Standard filter set: **ST150** Surface area of approximately 1.5 m^2

- *Prismocitrate 18/0* on the **white** scales connected to the **pre-blood pump**.
- *Prism0cal B22* on the **green** scales connected to the **dialysate circuit**.
- *Phoxilium* on the **purple** scales connected to the **replacement fluid circuit**.

Blood Flow Rate:

Weight (kg)	Blood Flow (ml/min)	Dialysate (ml/hr)	Replacement post-filter (ml/hr)	Treatment dose obtained (ml/kg/hr)
<= 50	100	1000	200	37
51 -60	110	1100	400	37
61- 70	120	1200	500	35
71 - 80	130	1300	500	33
81 - 90	140	1400	500	31
91 - 100	150	1500	600	31
101 - 110	160	1600	700	30
111 - 120	170	1700	800	30
>= 121	180	1800	1000	30

Initial Calcium replacement:

Patient ionised calcium (mmol/L)	**Starting calcium compensation (%)**
Less than 1.00	Give calcium replacement and start at 110%
1.00 – 1.11	110%
1.12 – 1.30	100%
Greater than 1.30	90%

		Filter ionised calcium		
		<0.25	0.25-0.5	>0.5
Patient ionised calcium	<1.0	Citrate dose decreased by 0.5mmol/L	Calcium compensation increased by 10%	Citrate dose increased by 0.5mmol/L **and** Calcium compensation increased by 10%
	1.0-1.3	Citrate dose decreased by 0.5mmol/L	No change required	Citrate dose increased by 0.5mmol/L
	>1.3	Calcium compensation decreased by 10% **and** citrate dose decreased by 0.5mmol/L	Calcium compensation decreased by 10%	Calcium compensation decreased by 10%
RECHECK ONE HOUR AFTER ANY CHANGE				