

HNE Area Intensive Care

Practice Guideline

Guideline approved for : JHH ICU only

Continuous Renal Replacement Therapy in Intensive Care(CRRT)

CRRT is a form of Renal replacement therapy which is used predominantly in intensive care units. CRRT provides continuous dialysis and /or filtration at much lower fluxes than standard intermittent haemodialysis , hence in general permitting dialysis to be better tolerated in critically ill patients. CRRT enables the control of solutes, fluid balance and hence early nutritional support in critically ill patients in Intensive Care.

[Decision tree CRRT/CRRT with Heparin / CRRT with Citrate / Prescription and Audit chart](#)

1. REFERRALS TO NEPHROLOGY

All decisions regarding the institution of CRRT should be made by the first-on ICU specialist in consultation with the on-call renal physician and the in-charge ICU nurse

2. Indications for CRRT

Acute Renal Failure (ie creatinine >200, U/O <0.5ml/kg/hr) where:-

i) The patient is immobile, ie on continuous respiratory support.

There is no evidence for this, but it is logical to avoid CRRT where the patient could otherwise be fully mobile.

AND

ii) There is a risk of multi-organ failure.

The "risk of multi-organ failure" could be defined as any need for other organ support - eg FIO₂ 0.5, jaundice, DIC, need for inotropes etc.

In the absence of the above, CRRT should also be considered:

iii) Where control of fluid balance is critical.

Intermittent dialysis may be impractical if removal of more than 2 litres/day is needed, eg TPN, congestive cardiac failure.

iv) Where urea/creatinine control is poor.

There is evidence that CRRT achieves urea levels <20. If intermittent dialysis fails to produce levels <35 between dialyses, CRRT should be considered

v) Where continuous removal of a specific toxin is required

The only current example of this is lithium intoxication.

vi) In life-threatening septic shock ie patients requiring significant inotropic support after fluid resuscitation.

Acute renal failure may not be established, but is inevitable in this setting. There may be some benefit in commencing CRRT pre-emptively as a means of controlling the septic state by reduction of inflammatory

mediator levels.

3. INTERMITTENT HAEMODIALYSIS should be considered where:-

The indications for CRRT no longer exists but renal failure persists. Biocompatible membranes are available for ICU patients recovering from septic shock

Maintenance haemodialysis is required to support the chronic renal failure patient admitted to ICU with an intercurrent critical illness.

If systemic anticoagulation with heparin and regional anticoagulation with citrate contraindicated

4. ACCESS

4.1 Venous access for CRRT should be via:-

i) either femoral vein (20cm catheters)

14 FG Arrow

ii) right internal jugular (15cm catheter)

14 FG Arrow

iii) left internal jugular

in descending order of preference.

NOTE: The subclavian should not be used in patients that may require chronic dialysis (NB only after discussion with Nephrologist , may cause venous stenosis which can effect successful av fistula formation for long term dialysis)

It is ESSENTIAL that the new catheter has adequate flow before suturing in position. A well positioned vascath should allow the aspiration of 20ml of blood in no more than 6 seconds(use a 20ml syringe and return the blood). Assess for each lumen.

PRESCRIPTION

Orders must include Mode of therapy, blood flow rate, replacement fluid type and rate, dialysis fluid type and rate, net fluid management.

Heparin orders must be written in the prn medication chart.

Where electrolytes are added to the Gambro fluid, they must be recorded on the prn medication chart eg. Lactate free fluid or KCL.

When replacement fluid is greater than 2.5l/Hr , phosphate levels should be monitored and replaced with KH_2PO_4

Replacement/dialysate rates and treatment aims are to be written in the comments section on patient flow chart and also on the prescription chart.

SPECIAL CONSIDERATIONS

1. Do not replace a clotted filter AT ANY TIME unless authorised by the first on specialist (i.e. contact intensivist even if after hours)
2. Filters should be changed 72 hours or if 780 litres of blood flow through filter has occurred. Discuss with First on staff specialist prior to continuing CRRT.

3. Nutritional Therapy:

The filter can not discriminate between uraemic toxins and nutrients, so the losses of nutrients such as amino acids, water soluble vitamins and minerals can be significant.

Therefore, it is important to ensure that protein intake is between 1.5-2.5g/kg/day.

Magnesium, potassium and phosphate can all be depleted, so they need to be carefully monitored and replaced.

Enteral formulas designed specifically for renal failure are unwarranted because fluid, protein and electrolyte restrictions are unnecessary during CRRT.

CAUTION:

Care needs to be taken once CRRT ceases, as nutrition therapy being provided is likely to be inappropriate if CRRT no longer running. If no dietetic advice has been provided in this situation, the safest formula would be Nepro, which is low in electrolytes and fluid, until a nutritional assessment can be conducted.

**Created: 9/98 by Dr Rowley Reviewed: 3/2007 by Dr Havill
Guideline to be reviewed before : 11/2009**

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CRRT Decision Tree

Identify the need for CRRT not IHD

Access/site

High flow Vascath - Site choice:

1. Femoral 20cm
2. Internal Jugular 15cm

Adequate flow from the access lumen by using a 20ml syringe must be established before suturing in place.

Can the patient have Heparin?

YES

([hyperlink to anti-coagulation guideline](#))

NO

Does the patient have adequate/sufficient Liver function?
Determined by LFT's (esp. bilirubin & PT)

NO

YES

MODE:
CVVHDF
Dose: 25mls/kg ultrafiltrate divided evenly into between the PBP and post filter dose
In addition 1 litre Dialysate
Blood Pump speed 200mls/min

If No
Then Use
Heparin Free
or alternate
anti coagulant

Citrate CVVH(F)
Citrate 1.6litres PBP, 1 litre pre filter and N/Saline 250mls/hr post Filter
Blood Pump speed 180mls/min
[Hyperlink to Citrate](#)

Choice of fluid

Use **Hemosol** if:
i) High or rising Lactate.
ii) Worsening pH or acidosis
iii) Liver failure
Otherwise use
Haemofiltration Fluid No.1

Mode setup parameters
See Help library Practice and Procedures

NOTE: The **K⁺** required in the fluid when a Patient has a normal **K⁺** level is 4mmols/litre.

Haemofiltration Fluid No.1 = 5mmol - **ADD** 15mmols

Hemosol = Nil - **ADD** 20mmols

Citrate = Nil - **ADD** 20mmols

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CRRT with heparin systemic anticoagulation

5. ANTICOAGULATION PROTOCOL

5.1 Baseline laboratory and Haemachron APPT's prior to commencement of CRRT (via venepuncture or a non-heparin containing line).

5.2 Systemic bolus dose of heparin 15-20 minutes prior to CRRT of 40-60 u/kg (actual weight max 100kg). Prescribed by 1st on ICU specialist or delegate (specialist or Fellow) unless contraindicated

5.3 Maintenance heparin infusion at 10% of loading dose ie. 4-6 u/kg /hr

Standard Starting Concentration: 5000units heparin diluted to 40mls in 50ml syringe(250units/ml).

e.g 60kg patient

Systemic bolus 2,400 to 3,600 units heparin

Maintenance Heparin starting range 6U/kg/hr running at 3ml/hr . Hence Heparin required in 20ml syringe is 1,500 units.

Aim to achieve APPT (hemachron) of 160-180 seconds after the first 4 hours ,unless otherwise specified by first on Intensivist or Fellow

5.4 Systemic bolus dose prior to recommencement should be calculated on the baseline APPT and interval since cessation of treatment. Where the interval is short, the new loading dose should be 50% of the original eg. routine change of circuit.

5.5 Monitoring APPTs should be drawn from arterial line .

5.6 Laboratory APPT and hemachron samples must be taken from the same site as above to ensure accurate comparison

5.7 Heparin should NOT be withheld despite the presence of severe thrombocytopenia.

5.8 If the Hemachron APTT reading is significantly sub therapeutic i.e. remaining at or below 150 secs , then a heparin bolus of half the original bolus dose should be prescribed and given. The infusion rate is then increased appropriately to facilitate return to optimum APPT range. RE-CHECK Hemachron in 1 (one) hour to see if responding.

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CITRATE REGIONAL ANTICOAGULATION FOR CRRT

(adapted from The Austin Hospital protocol)

Citrate regional anticoagulation operates on the principle of citrate administered pre filter chelates the free calcium in the blood, hence inhibiting the coagulation system within the dialysis circuit. The anticoagulation effect is reversed immediately the blood is returned to the systemic circulation of the patient provided that the systemic free calcium is at normal levels. The normal free calcium is maintained by calcium being replaced (that which has been lost in the ultrafiltrate) into the systemic circulation via a separate central venous line. Thus , the circuit is anticoagulated but the patient is not. It is important that the citrate is then metabolised by the liver to release the chelated calcium and produce bicarbonate that is lost in the ultrafiltrate.

[Additional Background information](#)

[Ca infusion algorithm](#) / [Monitoring](#) / [Citrate toxicity assessment](#)

[Calcium gluconate modification](#)

Indications

Whenever systemic anticoagulation is inadvisable or contraindicated

- Surgery within 48hrs
- Active bleeding
- Platelet count less than 80,000
- Heparin induced thrombocytopenia(HITTS)
- Head injuries

Contraindications

- Patients with acute liver failure
- Patients with acute on chronic liver failure
- Suspected ischaemic hepatitis(or shock states in which there may be decreased hepatic blood flow and citrate clearance.)
- Patients requiring high volume haemofiltration(>2L/hr)

Citrate Circuit Setup

Vascath access:as per CRRT clinical guidelines

Central line access:for calcium and magnesium infusions

Arterial line: for monitoring systemic ionised calcium and ABG's

ICU blood gas analyser: the ionised calcium functions must be operational

CRRT mode:CVVH only

Prisma circuit setup: Same as standard setup .

Circuit priming:5,000 units heparin, unless HITTS

Blood flow rate:180ml/min

Replacement fluid :Citrate replacement fluid.

Replacement fluid rate:2.6L/hour

Calcium Chloride infusion: Prepare 25mmol of calcium chloride(35ml) and dilute to 50ml with 5% dextrose(concentration 0.5mmol/ml). Infuse through central line. Commence at 4mmol/hr(8ml/hr), starting 15 minutes before starting CVVH. Thereafter adjust infusion according to sliding scale

Magnesium infusion:Dilute 25mmol of MgCl(25 ml 5mmol/5ml MgCl)to 50ml of 5% dextrose. Commence infusion at 3ml/hr through central line. * **MgCl is found with the CaCl in the hypertonic fluid cupboard.**

To complete setup: Set appropriate fluid removal rate and set heparin infusion rate to 0ml/hr(note a heparin infusion not made up)

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Citrate Regional Anticoagulation Background Information

(extracted from Austin Hospital protocol)

Citrate regional anticoagulation has been used for intermittent dialysis in patients at high risk of bleeding for close to 20 years. In a randomised controlled trial, it has been shown to decrease the risk of bleeding in high-risk haemodialysis patients. Since its description for continuous veno-venous haemodiafiltration 12 years ago, it has also been used extensively for the anticoagulation of continuous haemofiltration circuits worldwide. This type of anticoagulation relies on the principle that a citrate containing fluid is administered into the circuit pre-filter. The citrate chelates calcium and stops/attenuates the coagulation process in the circuit. Calcium is then replaced separately so that the patient's serum ionised calcium is maintained at normal levels. Thus, the circuit is anti-coagulated but the patient is not. In general, all reports seem to show that circuits last longer and the risk of bleeding is minimized.

Protocol

Principles of action Citrate causes anticoagulation by chelation of calcium. Chelation is a process by which an ion is attached to a neighbouring atom by at least 2 bonds. This process creates a strong bond that markedly decreases the ionised calcium available to the coagulation process. Because ionised calcium is a major co-factor in several of the coagulation cascade steps, its chelation decreases blood coagulability within the circuit. In this way, the CVVH circuit is effectively anti-coagulated. However, when the blood returning from the circuit (180 ml/min) returns to the patient circulation (5,000 ml/min), it mixes with normal blood and the calcium concentration is restored to normal. Citrate is taken to the liver and metabolized to yield CO₂/Bicarbonate (1 mmol of citrate = 3 mmol of bicarbonate) and calcium is released back to the body. However, some calcium is lost in the ultrafiltrate and needs to be replaced by a separate infusion to maintain overall calcium balance. With this method, the circuit is anti-coagulated, but the patient is not. Thus filter life is usually excellent but the patient's risk of bleeding is markedly minimized. Hence replacement fluid is different to usual CRRT replacement solutions because it cannot contain calcium or magnesium.

Definition of potentially suitable patients

All patients receiving hemofiltration, except for:

1. Patients with acute liver failure
2. Patients with acute on chronic liver failure
3. Suspected ischemic hepatitis (or shock states in which there may be decreased hepatic blood flow and citrate clearance).

(Bicarbonate replacement fluid should be used in these circumstances)

Contraindications and Explanations

Citrate is metabolised in the liver and it accumulates in blood during liver failure. It should not be used in patients with known liver disease.

Citrate cannot be used with high blood flows (>200 ml/min) and high ultrafiltration rates (high-volume haemofiltration) because too much citrate would have to be given to anti-coagulate the greater amount of blood and the liver would be unable to metabolise it.

If citrate accumulates, it can cause any combination of these 3 complications:

1. Metabolic acidosis
2. Hypocalcemia
3. Systemic hypocoagulability

Therefore monitoring of acid-base status and ionised calcium levels represent important aspect of safe citrate-CVVH management (see below).

Composition of citrate replacement fluid:

Citrate 12mmol/L, Sodium 140 mmol/L, Chloride 99 mmol/L, Potassium 1 mmol/L all in a 5-litre bag.

This composition means that, if hyperkalemia is not a problem, potassium chloride should be added as is already routine at 15 mmol per bag, which would change the potassium concentration to 4 mmol/L.

At a standard operating situation in the adult of 2 L/hr of UF with a blood flow of 150 ml/min, the patient would receive 28 mmol/hr of citrate and achieve an intra-circuit citrate concentration of 3.1 mmol/L, values very similar to the higher ranges reported in the literature (3-7). It should deliver circuit ionized Ca levels <0.3 mmol/L.

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Monitoring in Citrate Regional anticoagulation for CRRT

Monitoring	Special consideration
6/24 ABG's for (record on flow chart Ionised calcium as ICa)	ionized calcium(maintain 1.1 to 1.3 mmol/L)
Daily total calcium	base excess(beware if BE falls by > 5)*
Daily Mg	calculate total/ionised Ca ratio(Normal 1.9-2.1:1)*
Daily UEC(Na,Cl,PO4,U,Crt)	anion gap(beware if increased)*
Daily APTT , PT	
Daily at 1200 post filter ionized Ca(record on patient flow chart as PFCa in ABG section)	

Replacement

Ca infusion([Calcium infusion algorithm](#))

Mg infusion at 3mmol/hr (3ml/hr of 0.5mmol/ml MgCl solution)

KCL added to citrate replacement fluid(15mmol per bag) if hyperkalaemia is not an issue

* Increased anion gap, decreased base excess and/or rising total:ionized Ca ratio may indicate accumulation of citrate and the need to stop citrate CVVH

Special Issues

Persistent metabolic acidosis: Bicarbonate is continually lost in the UF and citrate is converted to bicarbonate by the liver (i.e. replacing the lost bicarbonate in the UF),at times this may not be enough. If necessary additional bicarbonate can be infused but must be a separate line to calcium infusion. Citrate toxicity must be excluded first. (discuss with intensivist)

Metabolic alkalosis: This may occur if high amounts of citrate are delivered to the patient. If requires treatment (pH>7.5)suggested management is : Obtain 2M hydrochloric acid from pharmacy(5ml ampoules of 2M HCl). Draw up 50ml of 2M HCL and dilute in 500ml saline, usual infusion rate 100ml /hr for 5 hours
~~(Discuss with intensivist)~~

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Citrate toxicity Assessment

Citrate Toxicity: This is a result of accumulation of citrate in the body, resulting in profound and refractory hypocalcaemia precipitating fatal arrhythmias.

Be aware of at risk patients.

- 1/ Acute or acute on chronic liver disease
- 2/ Citrate may accumulate in some patients with early ischaemic hepatitis (before an enzyme increase).
- 3/ In states of decreased hepatic blood flow (cardiogenic shock or severe septic shock)
- 4/ In some patients with lactic acidosis there may be a state of decreased hepatic flow. In some of these patients it is possible but relatively unlikely that citrate will accumulate.

Base Excess: must be monitored as it might indicate accumulation of citrate. A change in base excess towards the negative by $> 5 \text{ meq/L}$ (e.g. from +2 to -3 meq/L) requires medical review.

Calcium infusion: Typical finding of citrate accumulation is an increasing need to administer a greater amount of calcium by infusion to maintain a normal Ca_i and a **high total calcium to ionised calcium ratio**. If the base excess is changing measurement of total calcium (via HAPS) should be performed. The normal total/ionised calcium ratio is 1.9 to 2.1:1, if greater than this suspect citrate toxicity

Increased anion gap: Also a typical finding

The triad of metabolic acidosis, increased anion gap and high total to ionised calcium ratio should always lead to cessation of citrate-CVVH and change to bicarbonate fluid

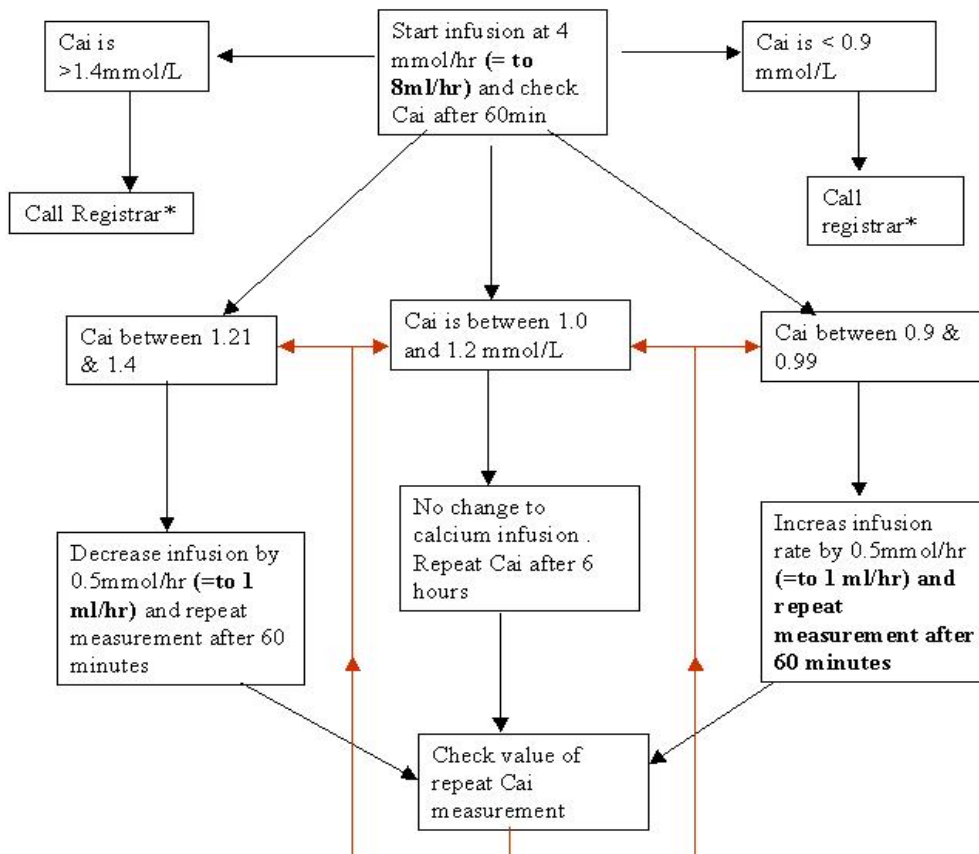
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Calcium Chloride Algorithm for CRRT with Citrate

CALCIUM INFUSION ALGORITHM



Registrar advice: If the Cai is <0.9 mmol/L (ionized hypocalcemia) a slow bolus of calcium chloride or gluconate is indicated (10 mmol over 10 minutes).

*Registrar advice: If the Cai is >1.4 mmol/L (ionized hypercalcemia) the calcium infusion should be decreased by 1 mmol/hr and a measurement repeated after 60 minutes. If still high, the infusion should be stopped for 30 minutes.

* Discuss with consultant if any doubt or concern exists

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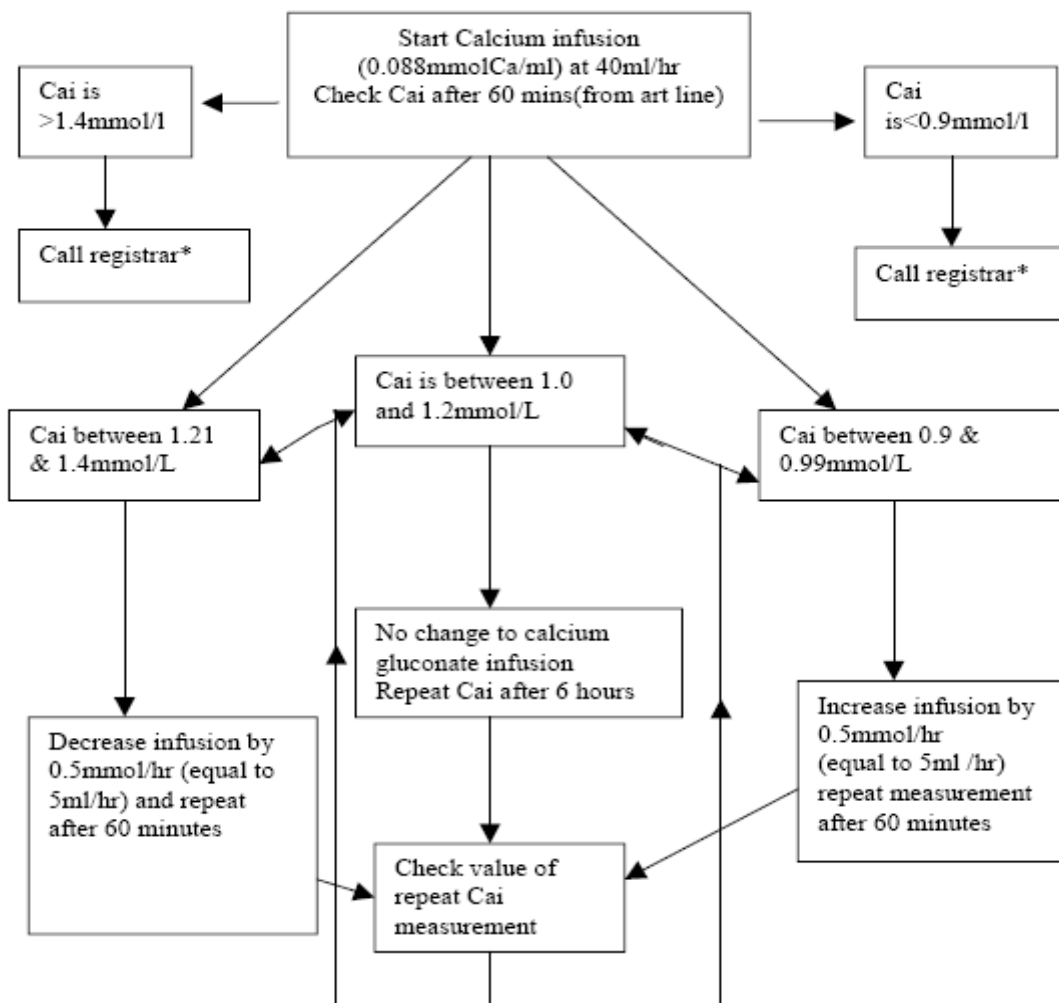
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Calcium Gluconate modification for CRRT

To be used only if calcium chloride is not available

Calcium Gluconate : 200ml calcium gluconate 10% solution in a 500ml bag 5% Dextrose (remove 200ml 5% dextrose before adding calcium) This will give a final concentration of 0.088mmol Ca/ml
Should be infused through central line. It is compatible with MgCl. Refer to algorithm for calcium gluconate

CALCIUM GLUCONATE INFUSION GUIDELINE



Registrar advice: If the Cai is ,0.9mmol/L a slow bolus of calcium gluconate is indicate(10mmol over 10minutes)

- Registrar advice: If the Cai is >1.4mmol/l the calcium infusion should be decreased by 1mmol/hr and REPAEAT MEASUREMENT AFTER 1 HOUR. If still high , the infusion should be stopped for 30 minutes

*Discuss with consultant if doubt persists

