

# ANAESTHESIA AND CHRONIC RENAL FAILURE

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Chronic Renal Failure (CRF) may be caused by primary renal disease or by systemic diseases which also affect the kidney. A decrease in nephron function occurs and can lead to a typical clinical pattern. CRF only becomes biochemically evident when less than 40% of the nephrons are functioning. Dialysis (either peritoneal or haemodialysis) is generally not required until less than 10% of nephrons are functioning. Patients with CRF are more likely to have associated atheroma formation and hypertension.

## *Preoperative Assessment and Treatment of Medical Problems in Renal Failure*

The following factors should be considered when assessing a patient for anaesthesia prior to either an elective or emergency procedure.

**Fluid balance** In CRF sodium and water excretion is relatively fixed and often reduced. The kidneys can have difficulty handling both large fluid loads and dehydration. The degree of hydration should be assessed in the usual way using skin turgor, examination of the mucous membranes, jugular venous pressure, presence of dependent oedema and presence of pulmonary oedema on auscultation. Invasive measurement of central venous pressure may occasionally be indicated. Many patients on dialysis regimens will know their normal hydrated weight and their fluid allowance per day.

The patient must be normovolaemic prior to surgery. Fluid resuscitation should normally be with normal saline but if there has been blood loss this might also have to be replaced.

**Biochemical balance** Although numerous biochemical abnormalities can exist and the potassium can be low, the most significant biochemical problems related to severe uncorrected renal disease are hyperkalaemia and acidosis.

Hyperkalaemia is defined as a serum potassium of more than 5 mmol/l. ECG changes become apparent at 6-7 mmol/l and immediate treatment is needed if the serum potassium is over 7 mmol/l. ECG changes include tall peaked T waves, shortened QT intervals, widened QRS complexes and loss of P waves. Eventually the QRS complexes merge into the T waves to produce a sine wave pattern. Ventricular fibrillation may occur at serum concentrations over 10 mmol/l.

Methods of treating a high serum potassium in an emergency include:

- a) Administration of 0.5ml/kg of 10% calcium gluconate (max 20 ml). This has an immediate but transient stabilising effect on the myocardial cells.
- b) 50mls of 50% glucose as an intravenous bolus or infusion. Glucose and insulin will produce an immediate migration of potassium into the cells thus reducing the serum level. Blood glucose levels should be closely monitored but unless the patient is diabetic, endogenous insulin will be secreted and maintain normal glycaemia. Alternatively 5-10 units of soluble insulin may be added to the infusion. Apart from the risk of errors which may occur, the patient may also become hypoglycaemic as secretion of endogenous insulin is also stimulated.
- c) Administration of 1-2 mmol/kg sodium bicarbonate intravenously over 5-10 minutes. This provides a large sodium and fluid load which may not be desirable.
- d) Nebulised salbutamol 2.5 - 5mg will assist in moving  $K^+$  into the cells.

Total body potassium levels can then be reduced:

- a) By dialysis.
- b) With calcium resonium (0.5 g/kg) 8 hourly either rectally or orally. This takes approximately 12 hours to produce an effect.
- c) By the introduction of a low potassium diet.

**Acidosis** can best be improved by dialysis. Administration of bicarbonate solution should only be considered when the pH is  $<7.2$ . Side effects of bicarbonate solutions include hypernatraemia and volume overload.

**Cardiovascular status** *Hypertension* may be a primary problem, secondary to chronic salt and water retention or to excess renin production. Blood pressure must be controlled preoperatively. *Ischaemic heart disease* is more common and should be assessed preoperatively. *Pulmonary oedema* may occur with fluid overload or with left ventricular failure. *Pericarditis* can occur in uraemic conditions.

**Respiratory function** *Pulmonary oedema* and *pleural effusions* both cause a decrease in lung compliance, functional residual capacity and increased ventilation/perfusion mismatch. All these increase the likelihood of hypoxia and are best treated by fluid removal with diuretics or dialysis.

**Haematological function** *Chronic anaemia* is common in patients with CRF who are not being treated with erythropoietin and is usually well tolerated. Unless the patient has ischaemic heart disease the haemoglobin level may be maintained at around 7-8 g/dl. Uraemic patients may have a *bleeding tendency* due to a decrease in platelet adhesion and fragility of the vessel walls.

**Gastrointestinal system** Anorexia, nausea, vomiting, bleeding from stress ulceration, diarrhoea and hiccups are all common symptoms. These can exacerbate dehydration. Nutrition is often poor and this can impair wound healing.

**Central nervous system** Uraemia causes malaise, fatigue, decreased mental ability and eventually coma. Severe uraemia or fluid or electrolyte imbalance may cause convulsions.

**Endocrine system** *Hyperparathyroidism* leads to demineralisation of bone making patients more susceptible to fractures. Diabetic control may be difficult because of decreased sensitivity to insulin.

**Multiple medications** Patients may be taking corticosteroids or other immunosuppressants which cannot be stopped. Other treatments may have been prescribed for associated diseases.

**Dialysis regimen** Those patients with end stage renal failure who are maintained on peritoneal dialysis should continue dialysing until they go to theatre. Haemodialysis should be ideally undertaken with minimum heparinisation up to 12 hours prior to elective surgery.

*Pharmacology of Anaesthetic Agents in Renal Failure*

The *excretion* of water soluble drugs and their active metabolites will be impaired. For drugs which are renally

excreted the half life increases slowly with deteriorating renal function until severe nephron loss at which point the half life increases sharply with further reductions in renal function. Dialysis can only usually replace a small part of the excretory capacity of the healthy kidney.

**Induction agents** Their effect is terminated by redistribution. All of these agents are myocardial depressants and should be administered cautiously in patients with heart disease.

**Muscle relaxants** *Suxamethonium* should be avoided if hyperkalaemia is present.

Some non-depolarising muscle relaxants depend on the kidney for elimination. *Atracurium* is the agent of choice as it undergoes spontaneous Hoffman degradation at body temperature.

*Vecuronium* and *mivacurium* are safe to use in renal failure as only small percentages are excreted renally.

*Gallamine* should be avoided and *pancuronium*, *alcuronium*, *pipecuronium*, *curare* and *doxacurium* should be used with caution. Potentiation of neuromuscular blockade may occur in the presence of a metabolic acidosis, hypokalaemia, hypermagnesaemia, or hypocalcaemia and with medications such as aminoglycosides. Monitor neuromuscular blockade whenever possible.

**Opioids** *Morphine* is metabolised in the liver to morphine-6-glucuronide which has about half the sedative effect of morphine with a markedly prolonged half life. Pethidine is partially metabolised to norpethidine which is less analgesic and has excitatory and convulsant properties. Both of these metabolites may accumulate in renal failure after repeated doses or with infusions. Standard intraoperative use will not usually cause problems. When available, morphine is preferable to pethidine.

**Fentanyl** and **alfentanil** can be used as normal.

**Benzodiazepines** can be used in renal failure.

**Inhalational agents** There is decreased elimination of the fluoride ions which are significant metabolites of enflurane, sevoflurane and methoxyflurane which can worsen renal function, so these inhalational agents should be avoided especially if used at low flows.

**Non steroidal anti inflammatory agents (NSAIDS)** should be avoided as all decrease renal blood flow and may precipitate complete renal failure.

### Conduct of Anaesthesia

**Premedication** Oral sedatives such as diazepam or temazepam may be used. H<sub>2</sub> antagonists or non particulate antacids (e.g. sodium citrate) should be given if oesophageal reflux is a problem.

**Anaesthesia** *Venous access* may be difficult. If future haemodialysis is planned it is important to preserve AV fistulas and potential fistula sites. Forearm and antecubital veins should be avoided if possible in these patients.

*Full monitoring* must be established prior to induction of anaesthesia, with special attention being paid to the ECG and blood pressure. The patient should be kept well oxygenated and haemodynamically stable. Hypovolaemia and hypotension worsen renal function therefore blood and other fluid losses should be carefully replaced. If possible the shorter acting sedative agents should be used.

If spinal or epidural anaesthesia is being performed fluid preloading should be kept to a minimum and vasoconstrictors used to maintain the blood pressure. Otherwise postoperative fluid overload may necessitate dialysis.

**Postoperatively** *Postoperative fluid balance* must be meticulous and prompt action taken to limit vomiting and replace any fluids lost. Some patients may require *haemodialysis* for fluid overload postoperatively but this should be delayed if possible as the patient will have to be heparinised. Some patients may become drowsy on relatively low doses of analgesics.

*Oxygen* (2-3 litres/minute nasally or 3-4 litres/minute via face mask) should be administered for 48 hours after major abdominal or thoracic surgery and 24 hours after intermediate surgery.

### PREVENTING ACUTE RENAL FAILURE

Previously healthy patients most at risk of developing acute renal tubular necrosis are those with massive haemorrhage, multiple trauma, sepsis, extensive burns and crush injuries, especially if they already have some degree of renal impairment. Renal failure is diagnosed when urine output is persistently <0.5ml/kg/hour or the serum creatinine rises.

The maintenance of normovolaemia and an adequate renal perfusion pressure are the two most important factors in avoiding acute renal failure. The underlying clinical problem should be controlled and treated as far as possible and adequate hydration guided if necessary by central venous pressure measurement. The urine output should be

measured hourly and should be maintained above 1 ml/kg/hr.

Only after the patient is well resuscitated with fluid should vasoactive drugs be used to maintain an adequate mean arterial blood pressure for the patient (this will depend on the patient's preoperative blood pressure). If the patient becomes oliguric (urine output < 0.5 ml/kg/hr) despite adequate hydration and blood pressure administration of frusemide can be considered, up to 240 mg intravenously over 1 hour. If no diuresis develops further administration of frusemide is useless. *Dopamine and mannitol* both increase urine output but also increase the oxygen demand

of the kidney so frusemide is preferred. Low dose dopamine has not been shown to have any protective effect on the kidney.

*All nephrotoxic drugs* should be avoided if possible. These include *NSAIDs* and *ACE inhibitors*. If *aminoglycosides* are essential their serum levels must be monitored.

*Electrolytes including potassium, sodium and bicarbonate* must be measured at least daily during the perioperative period. Adequate calorie intake is essential and must be established as soon as possible postoperatively.

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**Table 1: Preoperative assessment of the patient in chronic renal failure**

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Fluid balance  
 Biochemistry and acid base status  
 Associated illnesses  
 Associated medications  
 Dialysis regimen

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**Table 2: Common biochemical and haematological abnormalities in chronic renal failure**

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Hyper- (or hypo-)kalaemia  
 Hypo- (or hyper-)natraemia  
 Hyperphosphataemia  
 Hypocalcaemia  
 Metabolic acidosis  
 Normochromic normocytic anaemia

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**Table 3: Treatment of the acutely oliguric patient**

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Control the underlying cause if known  
 Ensure the patient is well hydrated using invasive monitoring if necessary  
 Ensure the blood pressure is normal or above normal for that patient  
 After fluid resuscitation try frusemide 240 mg over 1 hour  
 Avoid all non-essential nephrotoxic drugs  
 Adjust doses of renally excreted drugs  
 Measure sodium, potassium, bicarbonate and urea and/or creatinine twice daily  
 Establish low potassium nutrition as soon as possible

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