



FOCUS ON: RENAL

Renal replacement therapy in the intensive care unit

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S U M M A R Y

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Renal replacement can be defined as “An extracorporeal system or attachment to supplement, support or replace some or all functions of the kidney”. The kidney is uniquely sensitive, because of its microvasculature and permeability, to injury, either as a result of direct damage by toxins, oxygen free radicals or filtered inflammatory mediators, or microvascular failure in states of shock and sepsis. The resulting renal injury and failure is a particular problem in the Intensive Care setting, leading to a greatly increased mortality.

This article explores the techniques available for renal replacement and support in the intensive care unit, discussing vascular access, choice of technique, choice of membrane, choice of dialysis buffer and strategies for maintaining circuit patency. It examines the techniques in common use in the United Kingdom and the outcome following renal replacement therapy, discussing some of the controversies surrounding renal replacement in terms of timing and dose. It also discusses some future development in technologies for renal replacement.

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1. Introduction

There is no clear single definition of this term in the literature, so for the purposes of this review we use the term to mean, “An extracorporeal system or attachment to supplement, support or replace some or all functions of the kidney”. Renal replacement therapy (RRT), as a treatment for renal insufficiency, was first described in 1861. The first extracorporeal system as such was used in 1913, but it had very poor efficiency and at that time there were no safe anticoagulants. This situation continued until the discovery of heparin in 1942.¹ The technique of dialysis was first described in 1945 by Kolff and colleagues, but did not enter common use until the Korean War.^{2,3} These techniques were all intermittent; continuous renal repair or replacement therapy was first described by Kramer in 1977.¹ Dialysis membranes have also evolved dramatically over the years, with “drum type” membranes being replaced by microfibre microdialysis membranes in the 1960s. Nowadays, hollow fibre membranes are in almost universal usage.

2. Why is acute kidney injury a problem in the intensive care unit?

The kidney is uniquely sensitive, because of its microvasculature and permeability, to injury, either as a result of direct damage by

toxins, oxygen free radicals or filtered inflammatory mediators, or microvascular failure in states of shock and sepsis. While all vascular beds and organ systems can be affected by these processes in critical illness, the kidney, because of its permeability and numerous specialised cell types, is especially vulnerable. Consequently, a significant proportion of the critical care patient population goes on to develop transient or in some cases permanent renal dysfunction. Although acute tubular necrosis is the commonest form of renal injury seen in critical care population, patients may also present with primary intrinsic renal pathologies, or may develop other forms of renal injury as a result of their critical illness.

In an Australian study, over 10 years, 4.6%–6.9% of all patients admitted to intensive care units (ICU) developed acute kidney injury (AKI). The risk was associated with patients at the higher end of the age range, other comorbidity, emergency admission and medical (as opposed to surgical) primary diagnosis. The mortality rate in those with AKI was 42.7%, as compared with 13.4% in those without it. Likewise, those with AKI had an increased length of stay both in the ICU (4.4 days versus 2.6) and hospital stay (14.2 days versus 11.7).⁴ It should be recognized that the decision to commence RRT on the ICU will depend on a number of different factors but as a guide currently accepted indications are summarised in Table 1.

3. Classification of renal replacement therapy

A number of classifications of renal support have been proposed over the years. Broadly speaking, support systems rely on the

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Table 1
Indications for Renal Replacement Therapy.²

Anuria/Oliguria (associated with deteriorating clinical condition) Urine output <200 ml 12 h ⁻¹
Electrolyte/Acid-Base Abnormality pH <7.1 K > 6.5 (refractory to medical therapy) Na <115 or >160 (correct gradually if chronic)
Uraemia & its complications Uraemia >30 mmol l ⁻¹ (associated with deteriorating clinical condition) Pericarditis Encephalopathy Neuropathy/Myopathy
Clinically significant organ oedema (particularly lung)
Hyperthermia
Drug overdose with a dialysable toxin

physical principles of diffusion (eg., dialysis systems) or convection (haemofiltration). Additionally, pure filtration (ultrafiltration systems) may be included. Some dialysis systems represent a hybrid of the above physical principles (for example, haemodiafiltration). The techniques involved in haemofiltration are summarised in Table 2, which also indicates whether the method of vascular access is arterio-venous or veno-venous. Some arterio-venous systems rely on a passive flow of fluid down any pressure gradient; unlike the more common, veno-venous, systems, which rely on the use of roller pumps to propel blood through the circuit and dialysis machine in a way which is not as damaging to red blood cells. Associated flow rates are summarised in Table 3.

4. Vascular access for renal replacement therapy

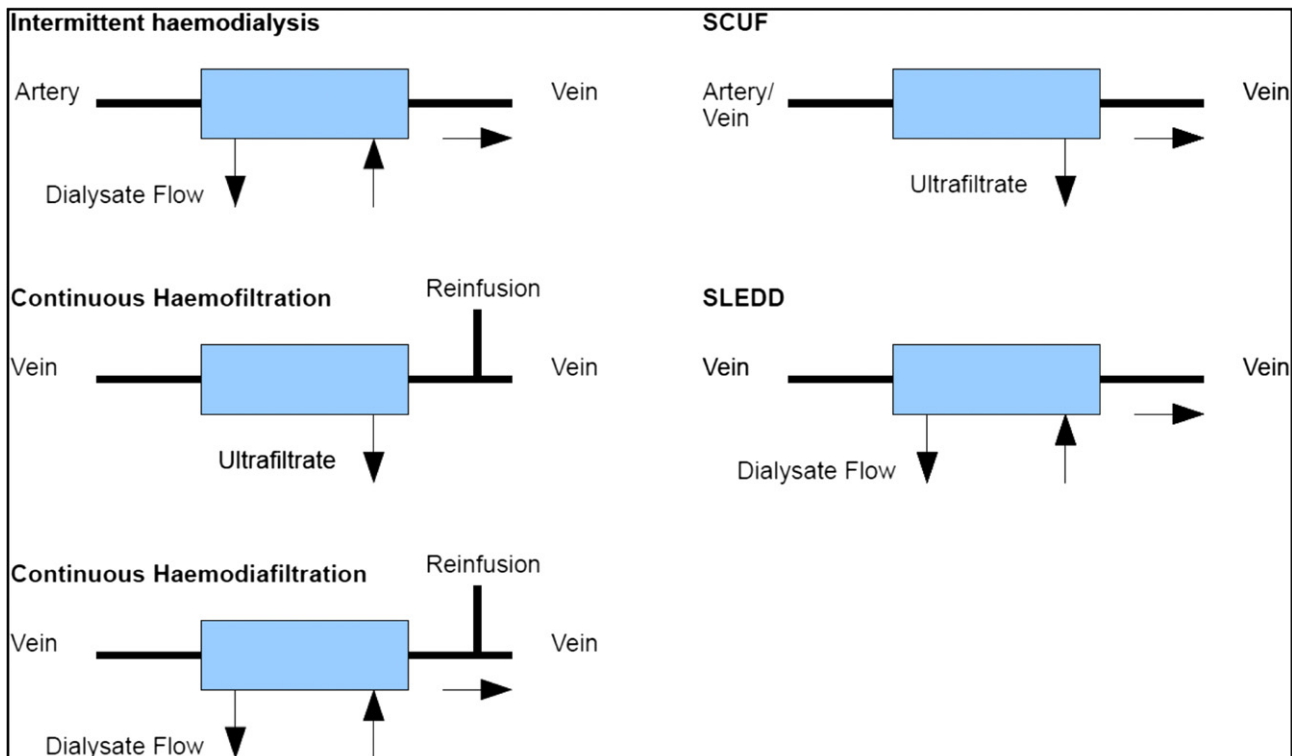
Typically, large-bore dual lumen central venous catheters are used. Higher flow may be achieved by using separate arterial and

venous cannulae, or by using two venous cannulae in separate sites. There is little work on this in humans, although the approach is validated in animal models.⁷ It may be particularly appropriate in paediatric ICUs where blood vessel size limits flow and cannula patency.⁸

In most situations, the internal jugular vein would be the site of access of first choice. Although there is a popular perception that the internal jugular route of access carries a low infection risk compared with the femoral route, there is no evidence to support this in patients with a body mass index below 28.⁹ The infection rates associated with subclavian cannulae is lower, although the subclavian route is generally not used for access for renal replacement therapy because of the large cannula size and the risk of subclavian vein stenosis.¹⁰ In the real world, the site of access is most likely to be dictated by the vascular anatomy of the individual patient, and the duration of time which they have been in the ICU, which other sites have already been used for vascular access (or are currently in use for other cannulae) etc.

There are risks involved in placement of large catheters for RRT. These include the immediate risks involved in cannulation, including vascular injury and pneumothorax (although these can be minimised by the use of ultrasound guidance) and the associated risks of haemothorax and pericardial tamponade. Later risks include formation of arterio-venous fistulae, aneurysms and vascular thrombosis and stenosis. It may be possible to limit the longer term risks by limiting the size of cannula and the duration of its insertion. Catheter length also has a bearing on function. Standard catheters via the femoral route of 24 cm have a lower risk of “catheter dysfunction” as compared with 15 cm catheters. Shorter catheters can give rise to recirculation, haemoconcentration, reduced solute clearance and earlier filter failure. These effects have been well known for many years.

Table 2
Typical techniques involved in renal replacement therapy.²



SCUF = slow continuous ultrafiltration, SLEDD = slow low efficiency daily dialysis.

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