Acute renal failure (ARF) is a sudden decrease in glomerular filtration rate (GFR) where GFR represents the amount of blood that is filtered by the kidneys. The kidneys are responsible for numerous physiologic processes including excretion of toxic metabolites and maintaining appropriate fluid balance in the body. Thus, ARF can result in a sudden, potentially life-threatening, dysregulation of both of these processes. Inappropriate or inadequate monitoring and therapy for patients with ARF can be lethal.

One of the most important functions of the kidney is strictly regulating serum potassium concentration. Kidney failure causing inefficient potassium excretion can lead to hyperkalemia. Nerve conduction in the body depends on a consistent gradient between the levels of potassium inside and outside of the cells. This gradient is particularly important in cardiac muscle. As levels of potassium outside of the cell rise, the gradient is diminished and cardiac muscle becomes more excitable. The result is cardiac arrhythmias that can be life-threatening. Animals showing evidence of hyperkalemia on their ECG tracing should receive emergency treatment. This includes the administration of calcium gluconate which temporarily helps to stabilize myocardial excitability. The ECG must be monitored continuously. Administration of 50% dextrose promotes endogenous insulin release which, in turn, facilitates movement of serum potassium into cells. Regular insulin can also be administered intravenously to augment this effect. Dextrose should always be administered with insulin to prevent hypoglycaemia. Regular (every 1 - 4 hours) monitoring of serum potassium is essential in patients with ARF that present with hyperkalemia.

The degree to which kidneys retain the ability to produce urine varies greatly in patients with ARF. While most patients lose the ability to concentrate their urine and become polyuric (urine production > 2 mL/kg/hr), others with more severe renal injury may be oliguric (urine production < 0.5 mL/kg/hr) or anuric (no urine production). It is essential to monitor the urine production of a patient with ARF every 1 to 4 hours and allow it to guide fluid therapy. This is best accomplished via placement of an indwelling urinary catheter. In those patients in whom urinary catheterization is not possible, every effort must be made to quantify their urine production and other losses (eg. vomiting and diarrhea). Monitoring the body weight of cats and small dogs is also helpful.

Fluid therapy must be carefully calculated to correct their fluid deficit (hypovolemia and/or dehydration) in addition to keeping up with their ongoing urinary and other losses. Conversely, animals that are oliguric (with a low specific gravity), must have their fluids titrated very carefully in order to avoid fluid overload and pulmonary edema. These patients benefit from central venous (jugular) catheterization and monitoring of central venous pressure. Monitoring central venous pressure can help to predict impending volume overload. The fluid therapy of oliguric patients that are adequately hydrated is best managed using ‘ins and outs’. That is, the fluid rate is matched to the level of urinary production (and other losses) in order to keep the overall fluid volume of the body constant.

Patients with ARF that are anuric have a guarded to grave prognosis. Such patients must be managed very carefully and must have a urine collection system in place. It is very important to rule out a bladder rupture in such patients. If the kidneys are definitely the culprit, carefully controlled methods of inducing urine production should be attempted. If administration of one or two aggressive fluid boluses followed by a one or two aggressive boluses of furosemide fail to result in urine production, peritoneal or hemodialysis is the treatement option.