Editorials

Predicting the need for renal replacement therapy and diuretic therapy in ICU: dopamine revisited?

Predicting who is going to need renal replacement therapy (RRT) in the intensive care unit (ICU) might be useful. It might allow earlier intervention with continuous haemofiltration, which might be beneficial to the patient. It might also allow earlier planning for the allocation of nurses and other resources to a particular patient in a busy ICU, where demand often exceeds supply. It might even prevent clinicians from implementing unnecessary, futile and perhaps injurious escalations in therapy (e.g. low-dose dopamine, mannitol boluses, further fluid loading, introduction of additional vasoactive drugs) to rescue kidneys that are beyond rescuing.

If all of these suppositions were true, then finding a way to predict who is going to need RRT is indeed a worthwhile goal. Dr. Ho and colleagues no doubt share the view that early prediction of who is going to need RRT is clinically useful and have sought to find a way to achieve it by retrospectively studying a cohort of patients with acute renal impairment in their ICU.[1] The authors present the results of their investigation in this issue of *Critical Care and Resuscitation*. Using logistic regression analysis they find that lack of response to frusemide challenge and need for vasopressor support with noradrenaline are the most powerful predictors of an ICU patient requiring subsequent RRT. They also find that the area under the ROC curve is 0.88 for the ratio of post frusemide to pre-frusemide hourly urinary output (averaged over 8 hours) in predicting whether a patient will or will not require RRT.

What are we to make of such observations? Well, first of all the authors themselves acknowledge several shortcomings of their investigation: the retrospective nature of the data, the lack of standardised criteria for the initiation of RRT, the lack of blinding to urinary output by the clinicians that were making the decisions to start RRT and the variable dose of frusemide used in the study patients. These are important shortcomings. To these, one must add the small size of the cohort, the fact that the study involved a single unit, thus making its generalisability to other settings unclear, the lack of reproducible criteria for what might constitute "adequate" fluid resuscitation, lack of information on the adequacy of cardiac output and its assessment, the pursuit of a mean blood pressure of 75 mmHg without apparent adjustments for previous hypertension or suspected vascular disease and so on. More importantly, some intensivists initiate RRT in response to marked oliguria to prevent, rather than simply treat fluid overload. Thus, saying that diuretic unresponsive oliguria in a very sick patient predicts the need for RRT might simply represent a tautology. Finally, the unit in question might also practice a style of management for these patients that differs form the approach in other units. For example, in the study by Ho *et al*, RRT was started in patients with a mean creatinine of 430 μmol/L compared to a mean creatinine of 311 μmol/L in Victorian ICU's,[2] a mean creatinine of 351 μmol/L in Australian ICU's[3] and a mean creatinine of between 309 and 327 in the 3 groups from a recent multicentre randomised controlled trial of RRT dose.[4] In the editorialist's ICU, it would be unusual to wait 8 hours (which the investigators must have at least waited for in order to report their frusemide responsiveness data) before implementing RRT in a critically ill, oliguric, vasopressor-dependent patient.

Beyond these issues, however, looms large the much more contentious issue of whether using diuretics in critically ill patients with acute renal failure might not, in fact, increase mortality.[5] In a recent study of patients with acute renal failure, diuretic use was reported to be associated with a covariate and propensity score adjusted odds ratio of death of 1.68. When tested for lack of renal recovery, the odds ratio was 1.79 and when the two were combined, the odds ratio was 1.77. In response to such data, a moratorium was called on the use of diuretics in such patients[6] until more indirect evidence accrues that they might at least be safe or until a prospective randomised controlled trial shows that they are.

The similarities with the PAC catheter are striking,[7] as perhaps are those with the albumin controversy.[8] Even more those with low-dose dopamine.[9] Although such work highlights the serious limitations of multivariate logistic regression analysis or propensity analysis (you can only enter the variables you collect which represent but a small fraction of the variables that matter), it also highlights the lack of randomised controlled trials to test whether diuretics are at least safe if not efficacious. In the area of acute renal failure management, the need for such studies is striking. Dr. Ho's paper reminds us once again of how little we know about what we do and why we do it. The feeling of *déjà vu* must be strong for a group like the ANZICS Clinical Trial Group that tackled and challenged the efficacy of low-dose dopamine in patients with renal dysfunction. Is the time ripe for another randomised controlled trial?

Professor R. Bellomo
Austin Hospital
Heidelberg
VICTORIA 3084

REFERENCES

1. Ho KM, Walters S, Faulke D, Liang J. Clinical predictors of acute renal replacement therapy in critically ill patients with acute renal impairment. Critical Care and Resuscitation 2003;5:97-102.
2. Cole L, Bellomo R, Silvester W, Reeves JH. A prospective multicenter study of the epidemiology, management and outcome of severe acute renal failure in a "closed" ICU system. Am J Respir Crit Care Med 2000;162:191-196.
3. Silvester W, Bellomo R, Cole L. Epidemiology, management and outcome of severe acute renal failure of critical illness in Australia. Crit Care Med 2001;29:1910-1915.
4. Ronco C, Bellomo R, Homel P, et al. Effects of different doses in continuous veno-venous haemofiltration on outcomes of acute renal failure: a prospective randomised trial. Lancet 2000;355:26-30.
5. Mehta RL, Pascual MT, Soroko S, Chertow GM. Diuretics, mortality, and nonrecovery of renal function in acute renal failure. JAMA 2002;288:2547-2553.
6. Lameire N, Vanholder R, Van Biesen W. Loop diuretics for patients with acute renal failure. JAMA 2002;288:2599-2601.
7. Connors AF Jr, Speroff T, Dawson NV, et al. The effectiveness of right heart catheterization in the initial care of critically ill patients. JAMA 1996;276:889-897.
8. Cochrane Injuries Group Albumin Reviewers. Human albumin administration in critically ill patients: systematic review of randomized controlled trials. BMJ 1998;317:235-240.
9. Australian and New Zealand Intensive Care Society (ANZICS) Clinical Trials Group. Low-dose dopamine in patients with early renal dysfunction: a placebo-controlled randomized trial. Lancet 2000;356:2139-2143.

Insertion of neonatal intercostal catheters

Resuscitation and support of neonatal patients is fraught with problems, relating in particular to small patient size and weight (at times less than 500 g). Techniques and devices taken for granted in paediatric and adult critically ill patients must be adapted or reinvented for the neonatal population. Methods for endotracheal tube intubation, arterial line insertion, central venous cannulation and chest tube insertion must be meticulous in equipment design, approach and vigilance in order to monitor and avoid complications. For example, the research, development and manufacture of endotracheal tubes, specifically for small patients, have aided the careful placement and management of artificial airways. On the other hand, pulmonary artery and intracranial pressure monitoring are extremely difficult technically and not often attempted in this age group.

At times, and often of necessity, devices used for older patients are reduced in size without modification to suit neonatal patients. As an example, Davies and Dunster[1] describe an extraordinary result in their study of tip placement on chest X-ray for trocar type chest catheters, where 13 of 24 intercostal catheters (54%) crossed the midline with potential for serious complication. They attribute the problem to distance markings on the side of the catheters being zeroed from the last side hole, some two extra centimetres from the catheter tip, resulting in the catheter tips being placed two centimetres further in than thought. The tolerance afforded in older patients is not acceptable for neonates, and these findings are important when considering pleural drainage in the neonate. The presumed counter argument in support of the Argyle® thoracic catheter design is that the distance markings help ensure the side holes are within the chest cavity and thus maintain a seal. The marking system is consistently 2 cm from the proximal side hole across all sizes, including adult catheters.

Different types of intercostal catheters have been studied for use in neonatal patients. Pigtail type catheters have advocacy and have been variously described as better from insertion, drainage (effusion, chylothorax) and complication rate points of view,[2] to poor from the point of view of blood and air drainage.[3] Trocar type catheters are less likely to block but add to the dead space ventilation in small patients. Whatever the catheter type, they are best placed in the sixth intercostal space, at the anterior axillary line for both air and fluid drainage to avoid subclavian vein perforation from a high anterior approach.[4] From a practical perspective, length of insertion will depend on what is to be drained and the site of pathology within the chest that needs to be drained, aiming to especially avoid mediastinal structures by not crossing the midline. Serious complications of insertion in neonates include haemorrhage, pneumothorax, phrenic nerve injury, and penetration of the mediastinum, pericardium, oesophagus and viscera.

It would seem easy to criticise such a high rate of catheter 'over insertion' in this study. But this belies the above comments on the difficulties of caring for such small patients, and the small tolerances of placing and maintaining devices safely in correct position for neonates. Of course the ultimate reassurance of intercostal catheter tip position comes from a follow up

chest X-ray, with re-manipulation of the catheter when necessary.

Dr. N. Matthews
Women's and Children's Hospital
North Adelaide
SOUTH AUSTRALIA 5006

REFERENCES

1. Davies MW, Dunster KR. Insertion distance of neonatal intercostal catheters using a 10 French Argyle® trocar thoracic catheter. Critical Care and Resuscitation 2003;5:103-105.
2. Wood B, Dubik D. A new device for pleural drainage in newborn infants. Paediatrics 1995;5:955-956.
3. Roberts JS, Bratton SL, Brogan TV. Efficacy and complications or percutaneous pigtail catheter thoracostomy in paediatric patients. Chest 1998; 114:1116-1121.
4. Genc A, Ozcan C, Erdener A, Mutaf O. Management of pneumothorax in children. J Cardiovasc Surg 1998;39:849-851.

Ethyl alcohol: not always a benevolent agent

Ethyl alcohol (ethanol) is readily available and usually ingested as a beverage of beer, wine or spirits. It is rapidly absorbed by the stomach and small intestine to reach peak blood levels approximately 30 minutes after ingestion (depending on the presence or absence of other stomach contents), with a volume of distribution equal to the total body water (i.e. 0.6 L/kg).[1] While fatalities with other alcohol poisonings are often due to the effects of toxic metabolites (e.g. formic acid with methanol poisoning,[2] glycolate, glyoxylate and oxalate with ethylene glycol poisoning[3]), in normal adults ethanol is not metabolised to toxic compounds although it can cause death due to respiratory failure particularly if taken by the novice drinker and if more than 300 mL of 100% ethanol is ingested (e.g. at a blood ethanol level of 0.355 g/dL or greater). [4]

However, the effect of ethanol on consciousness is variable, with chronic ingestion causing tolerance to high blood ethanol levels as an adaptive process.[5,6] A number of cases have been recorded where survival has occurred at extremely high blood ethanol concentrations (e.g. greater than 1.00g/dL).[7,8]

In this issue of the journal, Sanap and Chapman report a case that highlights once again the dangers of ethanol,[9] particularly for the non- or infrequent- drinker, where excess ethyl alcohol can lead to life threatening complications. Treatment is largely supportive, although diagnosis and management of other primary or secondary injuries (e.g. intracranial haemorrhage, aspiration pneumonia) are also important. In some cases renal replacement therapy has also been used with prompt restoration of consciousness in patients who have severe ethanol toxicity and persistent elevated blood levels (e.g. greater than 0.5g/dL).[10,11]

Ethanol is a social drug that is associated with acute and chronic morbidity and mortality. Thankfully, acute ethanol poisoning is rare, yet it still requires careful management to avoid tragedy.

Dr. L. I. G. Worthley
Department of Critical Care Medicine
Flinders Medical Centre
SOUTH AUSTRALIA 5042

REFERENCES

1. Jones AW, Jonsson KA, Neri A. Peak blood-ethanol concentration and the time of its occurrence after rapid drinking on an empty stomach. J Forensic Sci 1991;36:376-385.
2. Kruse JA. Methanol poisoning. Intensive Care Med 1992;18:391-397.
3. Brent J, McMartin K, Phillips S, et al, for the Methylpyrazole for Toxic Alcohols Study Group. Fomepizole for the treatment of ethylene glycol poisoning. N Engl J Med 1999;340:832-838.
4. Heatley MK, Crane J. The blood alcohol concentration at post-mortem in 175 fatal cases of alcohol intoxication. Med Sci Law 1990;30:101-105.
5. Davis AR, Lipson AH. Central-nervous-system depression and high blood ethanol levels. Lancet 1986;i:566.
6. Perper JA, Twerski A, Wienand JW. Tolerance at high blood alcohol concentrations: a study of 110 cases and review of the literature. J Forensic Sci 1986;31:212-221.
7. Berild D, Hasselbalch H. Survival after a blood alcohol of 1127 mg/dL. Lancet 1981;ii:363.
8. Johnson RA, Noll EC, Rodney WM. Survival after a serum ethanol concentration of 1½%. Lancet 1982;ii:1394.
9. Sanap M, Chapman MA. Severe ethanol poisoning: a case report and brief review Critical Care and Resuscitation 2003;5:106-108.
10. Atassi WA, Noghnogh AA, Hariman R, et al. Hemodialysis as treatment of severe ethanol poisoning. Int J Artif Organs 1999;22:18-20.
11. Koch-Weser J, Sellers EM, Kalant H. Alcohol intoxication and withdrawal. N Engl J Med 1976;294:757-762.

Cervical spine clearance in unconscious ICU patients - room to improve

Optimal cervical spine clearance for unconscious trauma patients is controversial and accordingly there is marked variability in practice in Australian intensive care units. In this issue of the Journal, Lien *et al*, report that there is no standardised approach to cervical spine clearance in intubated patients in Australia, that only 50% of the Australian trauma centres surveyed had a written protocol and that a standardised approach remains to be defined and tested.[1]

Development of a more standardised approach may be assisted by considering the experience from the Alfred trauma centre in Melbourne where the major trauma population is large (e.g. more than 700 per year) and cervical spine clearance in unconscious patients has been managed according to protocol for over 10 years. During this time the Alfred protocol has had several reviews and modifications.

The protocol was written primarily to enable earlier removal of Philadelphia collars in unconscious patients as it was clear that long periods in the collar provided inadequate immobilisation for a truly unstable cervical spine and was associated with substantial soft tissue and intracranial pressure related complications. The first cervical spine protocol we used for unconscious patients required adequate plain X-rays of the cervical spine followed by early passive flexion-extension X-rays to identify unstable ligamentous injuries. Passive functional X-rays were safe and had a very low but significant pick-up rate for cervical instability.[2] However, functional X-rays were extremely labour intensive for both the intensive care unit (ICU) and radiology staff. Moreover, a practice review found that passive flexion-extension X-rays were insensitive, with 4 of 122 patients having unstable cervical fractures that were not identified at all by the protocol and only recognised during rehabilitation when patients described "new" neurological symptoms. (D. van Gelderen, personal communication).

Accordingly, the Alfred protocol was modified to include both an improved passive flexion-extension technique (under image intensifier) together with complete helical cervical spine computed tomography (CT) and reconstruction in all patients. Cervical spine CT had the advantage of being able to be performed in trauma patients at the same time as the first head CT scan.

After 20 months using both investigations, the outcomes in 277 patients were studied (L. Padayachee, personal communication). The major findings were that routine cervical CT (1 mm cuts from C_0 - C_2 and 3 mm cuts from C_2 - T_2) identified more fractures than previously found and that improved functional X-rays found no new injuries (i.e. those injuries that had not been identified using CT). The lingering concern that an unstable cervical longitudinal ligament injury may only be diagnosed with functional X-rays was not supported by this study. Indeed it appears that fine cut helical CT with reconstruction identified all significant cervical injuries in a very severely injured population.

These findings led to the current Alfred protocol. All unconscious patients have a plain antero-posterior and lateral cervical spine X-ray followed by a CT with reconstruction of the whole cervical spine at the same time as their first CT head scan. When the cervical CT has been reported by a radiologist, neurosurgeon, or trauma consultant the cervical spine is "cleared" and the collar is removed. Routine transportation from the ICU to the radiology department for functional X-rays has been eliminated and Philadelphia collars and their complications are much less commonly found in the ICU. Some patients (e.g. those with clinical signs, or CT abnormalities) require further investigation, usually MRI, but these are a minority.

Publication and critical review of the above studies may be followed by greater standardisation of Australian clinical practice. This is one area where research and practice from American and European Trauma Centres have provided little guidance.

Assoc. Professor D. J. Cooper
Head Trauma Intensive Care
The Alfred Hospital
VICTORIA 3181

REFERENCES

1. Lien D, T. Jacques, Powell K. Cervical spine clearance in Australian intensive care units. Critical Care and Resuscitation 2003;5:91-96.
2. Ajani AE, Cooper DJ, Scheinkestel CD, Laidlaw J, Tuxen DV. Optimal assessment of cervical spine trauma in critically ill patients: a prospective evaluation. Anaesth Intensive Care 1998;26:487-491.