Editorials

Why do we continually ask "Do we need Intensivists"?

I know of no other discipline that consistently reviews its practice with and without specialists.[1-10] Cardiologists, nephrologists, thoracic physicians, etc, go about their business with the understanding that theirs is a specialty that requires an appropriately trained and experienced individual before an excellence in care (and we assume a reduction in patient morbidity and mortality) can be provided. Yet intensivists continually provide evidence that their specialty is relevant and that training is important if a reduction in morbidity and mortality for critically ill patients, and decreased cost in running the intensive care unit are to be achieved.[2-10] In one recent multi-centre study, daily rounds by an intensivist were associated with a three fold reduction in in-hospital mortality in abdominal aortic surgery patients,[11] implying that a hospital could be negligent if it allowed major vascular surgery to be performed without providing postoperative care by an intensivist in an intensive care unit.

Early in the genesis of the specialty of critical care medicine in the United States of America it was believed that "no one person can become competent in all aspects of critical care".[12] However, this is probably true of any specialty. Yet this common wisdom along with an editorial "concern" with the concept that "critical care service should be provided by full time physicians who assume primary responsibility for patient care",[13] implied that an 'open' format intensive care unit (i.e. where the admitting clinician dictated management) was the preferred model. In the United Kingdom, an Inter-Faculty Collegiate Liaison Group on Intensive Therapy took a stronger stand by stating that it "does not recommend the emergence of the 'Intensivist' as a separate specialist, but considers that consultants with a special interest in intensive therapy would also pursue a clinical career in their parent specialty (anaesthesia, medicine or surgery)".[14]

From the beginning in Australia and New Zealand a 'closed' format intensive care unit (i.e. where the resident intensivist dictated management) became the predominant working model. Specialists from either anaesthesia or internal medicine (or both) functioned as the 'intensivist'.[15] From 1980, Intensive Care Medicine became a sectional specialty of the principal specialties Anaesthetics and Internal Medicine, and both the Royal Australasian College of Physicians and the Faculty of Anaesthetists, Royal Australasian College of Surgeons developed their own training schemes. Currently, the administrative issues of training are changing and it appears that a single Australasian body for certification in Intensive Care Medicine will soon become a reality.[16]

While intensive care units do not have a standard administrative structure (due to regional differences in hospital services),[17] the issue concerning the need for an intensivist has now became clear. Intensive care units that have a 'closed' format have a lower morbidity and mortality when compared with intensive care units that have an 'open' format.[4-11] The data are compelling; hospitals that manage critically ill patients need intensivists if they are in the business of reducing morbidity, mortality and costs.

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How should we measure the emotional well-being of paediatric intensive care patients?

Paediatric intensive care units struggle with managing the emotional well-being of their patients. The spectrum of diseases, ages and severity of illness in these units almost mandate individually tailored approaches, which at times are demonstrably unsuccessful despite thoughtful protocols.

Adult patients' experiences in intensive care have been well studied.[1] This is not so for children. The effects of pain on recovery from illness and the impact of long term sequelae have been described in children,[2] but not for paediatric intensive care patients. Thus, the impacts of paediatric intensive care management on recovery from illness and the effects on long term psychological sequelae have yet to be determined. In a milestone pilot study published in this journal,[3] Taylor *et al* have commenced the exploration of these issues by investigating the incidence of children recalling general and specific events during their intensive care admissions. Of particular note is their patients' ability to have extensive recall of painful events, with implied effects on short and long term recovery, and psychological well-being.

The study has limitations which are acknowledged by the authors. However, the importance of the study and the implications on the need for further enquiry means it deserves careful consideration. Hopefully it will create constructive debate. How can children's experiences be more carefully analysed? How can the many variables such as age, severity of illness and socioeconomic background best be considered? This path of research has considerable potential for improving clinical practice of paediatric intensive care patients.

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Quantifying and improving functional survival in traumatic brain injury

Despite improvements in pre-hospital care, trauma systems and intensive care, survival from traumatic brain injury remains poor with documented mortality rates ranging between 25 - 40%. There are many reasons for the high mortality including the severity of primary injury and compounding secondary ischaemic-hypoxic insults. Whilst strategies directed at reducing mortality are critical, focussing primarily on injury prevention and defending cerebral perfusion pressure, improving the functional outcomes of survivors is an equally important challenge. The physical, psychological and economic impacts of disabled survivors on the community are substantial.

Three important issues arise with respect to quantifying functional survival from traumatic brain injury. Firstly, there is no ideal assessment tool for quantifying outcome following traumatic brain injury. The Glasgow Outcome Score (GOS), described twenty years ago by Jennet and Bond,[1] was designed to categorise the outcome of all patients following head injury, including both survivors and non-survivors. Although the GOS is the most commonly used system, concerns about the reliability of assignments of outcome have been expressed; specifically relating to the influence of subjectivity, inter-observer discrepancy and poor discrimination between survivors with moderate to high levels of disability.[2,3] Furthermore, there is no

consensus regarding the optimal follow up period. This is an important issue as recovery from head injury is a dynamic process as functional outcomes may be quite different at six months compared with 12 months following injury. The hypothermia study by Marion *et al* demonstrated significantly different outcomes between cohorts of survivors at 6 and 12 months, making deductions about the efficacy of an intervention difficult.[4] The GOS has recently been restructured into an eight point extended scale using a standardised questionnaire, with the aim of reducing inter-observer error.[5,6] Although the extended GOS is currently recommended as the primary method of assessing crude outcome in management trials of severe head injury, it is not the perfect tool.[7]

Secondly, correlation between markers of severity of injury and outcome tools such as GOS remains inconclusive. Previous studies have analysed the relationship between post resuscitation Glasgow Coma Scores, cerebral computed tomography (CT) appearance and GOS and demonstrated that whilst patients with severe injuries have poor GOS grades, specificity and sensitivity are low with respect to assessment of functional outcomes.[8,9] Whilst patients who survive traumatic brain injury may make a reasonable physical recovery in locomotion and basic life skills, neuropsychological sequelae of cognitive and behavioural disorders represent a significant impediment to reassimilation into society. This is clearly beyond the scope of physiological scoring systems such as the Acute Physiology and Chronic Health Evaluation score or the GOS. The need for validated functional assessment tools is highlighted by Furlonger *et al* in the current issue of Critical Care and Resuscitation.[10] In a series of 123 severely head injured patients with a cohort mortality of 20%, functional survival in 68 patients was determined using two neuropsychological assessments 12 months after injury. Behavioural outcomes did not correlate with the degree of cognitive impairment suggesting a wide range of functional disability in these patients. These assessments were subsequently correlated against neurophysiological indicators such as pre-hospital hypoxia, anatomical abnormalities detected by cerebral CT scan, adequacy of cerebral perfusion pressure during intensive care admission and somatosensory evoked potentials. Although patients with poor neurophysiological indicators had poor neuropsychological outcomes, no correlation between these variables and cognitive and behavioural assessments was demonstrated.

Thirdly, the difficulty in quantifying outcomes from traumatic brain injury is a significant factor in the design of interventional trials where improved outcome is the primary endpoint, albeit all-cause mortality or improved functional survival. In an important review of published studies in traumatic brain injury, Dickinson *et al* provide a critical analysis of study design in order to demonstrate the smallest absolute risk reduction using standard power calculations.[11] For example, in order to demonstrate a 5% absolute reduction in mortality (which would be considered a "magic bullet" in neurotrauma), a sample cohort of 10240 patients would be required. By nature, this mandates large multicentred trials. Given the argument that functional survival is a more important endpoint with respect to the community as a whole, the difficulties in accurately quantifying functional outcomes add another perspective to study design.

Clearly, the quest for defining and improving meaningful outcomes following traumatic brain injury remains a significant challenge for clinicians and researchers alike.

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Relative risks: relative benefits

Thrombolytic therapy for pulmonary embolism continues to be a vexing issue. It seems clear that, compared with heparin alone, thrombolytics result in more rapid physiological improvement. Pulmonary artery pressure and right ventricular end-diastolic volume fall and right ventricular ejection fraction and cardiac output improve more rapidly. However, no improvement in mortality has been proven beyond doubt, and there is an attendant increase in bleeding risk, which becomes a real concern when there has been recent surgery.

In this issue of the Journal, Theron and Laidlow[1] describe thrombolysis, using reteplase, in a patient with massive pulmonary embolism on the day following an anterior cruciate ligament repair. Recent surgery was considered only a relative contraindication because the patient was moribund with apparently little to lose. While I suspect most clinicians would have made a similar decision, if the case involved an alternative site of surgery (e.g. intracranial, spinal cord, hepatic resection) and the patient was haemodynamically unstable, but not moribund, the judgement of the risk-benefit ratio with thrombolytic therapy may have been altered. Atkinson and Worthley summarise the current literature.[2] However, precise data addressing the particular problems of management of an acute thromboembolism in the postoperative patient may never exist, and these patients will continue to pose clinical conundrums.

In the unstable or moribund patient, transthoracic echocardiography can be urgently performed during resuscitation, and a dilated right heart helps confirm the clinical diagnosis. Occasionally, clot will be found in the right atrium or ventricle and this may require physical removal since sudden obstruction may occur and these thrombi seem resistant to thrombolysis.

Resuscitation from acute right ventricular failure, reduction in clot burden, and prevention of further embolic episodes should proceed simultaneously. Hypoxaemia, a potent pulmonary vasoconstrictor, may demand intubation and ventilation. However, because pulmonary vasodilators commonly cause systemic vasodilation and a reduction in right ventricular perfusion pressure, they have little role in patients with hypotension due to massive pulmonary embolism. Indeed, while it may seem counterintuitive, vaso-pressors are commonly required in hypotensive patients, and do not appear to have adverse effects on right ventricular loading.[3]

Right ventricular ischaemia due to increased afterload and a decrease in coronary perfusion pressure results in right ventricular failure. Vasopressor-augmented right ventricular coronary perfusion pressure increases coronary blood flow, reduces right ventricular ischaemia and results in improved right ventricular performance.[4] In an experimental model of pulmonary embolism, noradrenaline infusion improved survival when compared with isoprenaline or fluid loading.[5] However, when right ventricular coronary perfusion pressure is adequate, inodilators such as isoprenaline, dobutamine or milrinone may augment flow.

A recent report of a reduction in mortality with thrombolytic therapy should be interpreted cautiously, but not dismissed.[6] This was a non-randomised uncontrolled study using data from a multicentre registry. Patients receiving thrombolytics were younger and had less cardiorespiratory disease. Shocked patients were excluded. However, the numbers were relatively large (169 patients received thrombolytics and 550 received heparin) and, taking into account group differences, thrombolysis was associated with an improved survival (Odds Ratio 0.46 [0.21-1.00]), despite an increase in bleeding, with 11% of patients requiring cessation of thrombolytics or surgical intervention. Consequently, reduction of clot burden with thrombolytic therapy can only be recommended for patients when the significant physiologic disturbance and presumed risk of death outweighs the surmised risk of bleeding.

Surgical or radiological embolectomy seems an attractive option, but is rarely available, and requires simultaneous protection from recurrent pulmonary embolism. A more conservative approach has been recommended for patients with massive pulmonary embolism who can be stabilized. Moser[7] argues that, because recurrent pulmonary embolus is the primary cause of death in these patients, heparin and an inferior vena caval filter make more sense than thrombolysis. If this approach is adopted, a temporary filter can be inserted and removed a week or two later to minimize complications due to venous hypertension. Certainly, this approach appeals to me when the bleeding risk outweighs the benefits of a quick fix with thrombo-lytics.

While awaiting definitive trials, we will have to rely

on our judgement of an individual patient's risk to benefit ratio. However, its also crucial not to forget that this is a potentially preventable problem both inside and outside the intensive care unit.

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