Optimal mechanical ventilation for ARDS

The notion that ventilator-induced lung injury (VILI) contributes to pulmonary dysfunction, systemic organ dysfunction and outcome has been well established in laboratory studies.[1] However, optimal mechanical ventilation in patients with the acute respiratory distress syndrome (ARDS) remains contentious. The most prominent clinical data have been published by the ARDS Network, particularly their study examining low vs high tidal volume ventilation (6 vs 12 mL/kg predicted body weight).[2] Low tidal volume ventilation resulted in a reduction in mortality from 39.8% to 31%, despite a worsening of oxygenation in the first three days of the study. Although it has been argued that the low tidal volume protocol used in this study should be the current optimal ventilatory strategy in ARDS, there are many unresolved issues. In this issue of the Journal, Roberts[3] contends that high frequency oscillation (HFO) is the logical extension of tidal volume limitation. Many of us will find this provocative; because we have none or limited experience with HFO, because clinical data with HFO in ARDS are interesting but not definitive, and because of the practical and theoretical issues raised.

Acute lung injury (ALI) and ARDS are more common in Australian ICU's than most of us recognize. In a recent survey conducted in South Australia, Western Australia and Tasmania the incidence of ALI and ARDS were 34 and 28 cases per 100,000 population (aged greater than 15 years) per annum, respectively.[4] Extrapolating these data to the total number of ICU admissions, approximately 1 in 9 patients in an Australian ICU develop ALI, and an average ICU that admits 1000 patients per year will manage over 100 patients with ALI. As about 30% of patients 'at-risk' will develop ALI, there are a vast number of patients who may benefit from protective ventilatory strategies.

These data may seem excessive, however, it is important to recognize that the criteria used to diagnose ALI and ARDS were the 1994 American-European Consensus Conference criteria;[5] which are the same criteria used by most studies in this area including the ARDS Network. Indeed, the benefit demonstrated with low tidal volume ventilation by the ARDS Network was found in a cohort of ALI patients remarkably similar to ALI patients in Australia.[4] Perhaps we tend to focus on patients with severe ARDS, many of whom already suffer from VILI, when intuitively the greatest benefit with protective ventilatory strategies will be pre-emptive or early in the course of the disease. Consequently, simple measures such as the detection of 'at-risk' patients, increased recognition of ALI and ARDS, and easily applied ventilatory strategies will have the greatest clinical impact.

The greatest clinical experience with HFO has been with preterm infants. However, these data remain inconclusive, and Bryan, who has pioneered much of the laboratory work on HFO, concludes that there appears to be little advantage with HFO compared with well managed conventional ventilation.[6] This view is supported by recent studies[7,8] and a Cochrane review[9] which concluded that despite more rapid improvement in oxygenation, there was no improvement in mortality with HFO and reduced chronic lung disease was only found when early recruitment was combined with an open lung strategy. A similar result was reported by Derdak and coworkers[10] in the first randomized trial using HFO for ARDS. They found an early improvement in oxygenation at the cost of a higher mean airway pressure. There was no difference in the oxygenation index and mortality was no different in an underpowered study. ARDS trials examining modes of mechanical ventilation are extremely difficult to perform, and Derdak and coworkers must be congratulated for making an important contribution. However, their cohort had relatively severe ARDS as the inclusion criteria also demanded that the patients were receiving at least 10 cmH_2O PEEP, and, as found by the ARDS Network low tidal volume study, surrogate markers such as oxygenation may not predict outcome.

As HFO matches overdistension, a major mechanism of VILI, why have the trials with very low tidal volume ventilation been inconclusive? Bryan postulates that this is due to the implementation of HFO rather than the technique itself,6 and clinical trials must be adequately powered to outcome rather than a physiological endpoint. Pillow and associates,[11] who have examined differences between airway and alveolar pressure with HFO raise a further important issue. During HFO, mean airway pressure is significantly higher than during conventional ventilation. Alveolar pressure and its oscillations are far less as mean airway pressure is dissipated by the lung. In the normal lung this leads to low alveolar pressures and reduced VILI. This is also the case in homogeneous lung disease such as found in preterm infants. However, in heterogeneous lung disease such as ALI, ARDS and bronchopulmonary dysplasia, regional differences in compliance lead to a much greater transmission of proximal airway pressure to areas with short time constants. The aim with the protective ventilatory strategies is to recruit and not overdistend these areas of low compliance. Paradox-

ically, HFO may be repeatedly and preferentially delivering high pressure pulses to these regions thereby worsening VILI. These data not only help understand the pros and cons of ventilatory modes such as HFO, they signal the importance of techniques to investigate ventilation and lung heterogeneity as a means to finding the optimal parameters for mechanical ventilation in ARDS.

Professor A. D. Bersten
Flinders Medical Centre
Bedford Park
SOUTH AUSTRALIA 5042

REFERENCES

1. Dreyfuss D, Saumon G. Ventilator-induced lung injury: Lessons from experimental studies. Am J Respir Crit Care Med 1998;157:294-323.
2. The Acute Respiratory Distress Syndrome Network. Ventilation with lower tidal volumes as compared with traditional tidal volumes for acute lung injury and the acute respiratory distress syndrome. N Engl J Med 2000; 342:1301-1308.
3. Roberts RG. High frequency oscillatory ventilation tidal volumes: how low can we go? Critical Care and Resuscitation 2003;5:63-65.
4. Bersten AD, Edibam C, Hunt T, Moran J, and the ANZICS Clinical Trials Group. Incidence and mortality of acute lung injury and the acute respiratory distress syndrome in three Australian States. Am J Respir Crit Care Med 2002;165:443-448.
5. Bernard GR, Artigas A, Brigham KL, et al. The American-European Consensus Conference on ARDS: definitions, mechanisms, relevant outcomes, and clinical trial coordination. Am J Respir Crit Care Med 1994;149:818-824.
6. Bryan CA. The oscillations of HFO. Am J Respir Crit Care Med 2001;163:816-817.
7. Johnson AH, Peacock JL, Greenough A, et al. High-frequency oscillatory ventilation for the prevention of chronic lung disease of prematurity. N Engl J Med 2002;347:633-642.
8. Courtney SE, Durand DJ, Asselin JM, et al. High-frequency oscillatory ventilation versus conventional mechanical ventilation for very-low-birth-weight infants. N Engl J Med 2002;347:643-652.
9. Henderson-Smart DJ, Bhuta T, Cools F, Offringa M. Elective high frequency oscillatory ventilation versus conventional ventilation for acute pulmonary dysfunction in preterm infants. Cochrane Database Syst Rev 2000:(2):CD000104.
10. Derdak S, Mehta S, Stewart TE, et al. High-frequency oscillatory ventilation for acute respiratory distress syndrome in adults: A randomized controlled trial. Am J Respir Crit Care Med 2002;166:801-808.
11. Pillow JJ, Sly PD, Hantos Z, Bates JHT. Dependence of intrapulmonary pressure amplitudes on respiratory mechanics during high-frequency ventilation inn preterm lambs. Pediatr Res 2002;52:538-544.

Plan A, Plan B and Plan C: management of the difficult airway in the critically ill

One of the most frightening and challenging aspects of critical care medicine is the difficult airway. The actual incidence of either a difficult intubation or a "can't intubate, can't ventilate" scenario is uncommon, but is still likely to touch all practitioners at some stage during their careers. The complications of such a scenario range from teeth damage and airway trauma, through unnecessary tracheostomy to brain injury, cardiac arrest and death.

Many publications have addressed parts of this problem, including an update of the American society of anaesthesiologists' (ASA) "Practice guidelines for management of the difficult airway" which has recently become available.[1,2] Unfortunately, patients that are critically ill do not usually avail themselves of some of the potential assessment phases that an elective surgical patient may offer an anaesthetist. The verbal or documented history may be absent, the obtundation or severity of illness of the patient may preclude optimal airway assessment and their cardiorespiratory reserves may preclude standard approaches.

In this issue of the Journal, Lim and Hunt-Smith summarise the previously published literature surrounding difficult airway management and attempt to highlight those issues particularly relevant to critical care practitioners.[3] Factors predisposing to a difficult laryngoscopy, such as obesity, limited neck mobility and limited mouth opening can be assessed quickly in an emergent scenario, but others such as the Mallampati test or additional investigations may be impractical or dangerous. As rightly pointed out by the authors, no single test or combination provides adequate sensitivity (i.e. picking the problem ones). This points to the first conclusion, that despite the information available, all critically ill patients should be assumed to be difficult intubations.

The author's also discuss many of the myriad of airway techniques that are available to all of the practitioners that are responsible for airway management.[3, 4] Again the author's rightly point out that the time for trying out a new technique is not the airway emergency. They suggest that practitioners should become familiar with more than one technique, though the difficult intubation trolley should not have "too much equipment" which could result in "confusing the practitioner with excessive choices". It would not be in the patient's best interests if yet another intubation or ventilation technique were tried, if it was to significantly delay

definitive management or was associated with more morbidity than a surgical airway technique. It is very reasonable to expect all practitioners to have at least one additional technique for the ventilation of a patient where the initial attempt at intubation has failed and mask ventilation is inadequate. The laryngeal mask airway (LMA) is the most widely used technique, and as described is easy to learn and is usually effective. Practitioners should also have at least one technique with which they are comfortable, and prepared to use, to acquire a transtracheal airway (e.g. cricothyrotomy). This leads to the second conclusion, that adequate planning must be made in advance for the potential failure at each step in the algorithm.

In addition to the above, the author's have provided us with a simplified failed intubation algorithm. They rightly point out that the previous (and current) ASA difficult airway algorithms incorporate such alternatives as awaken the patient or use regional nerve blockade.[2] Four issues that are touched upon require further expansion.

Firstly, critically ill patients not only have decreased cardiorespiratory reserves, but now are often on large amounts of respiratory support via a face mask (very high percentage of inspired oxygen, high levels of positive-end-expiratory pressure, and/or high levels of inspiratory pressure support). Emergency intubations in these scenarios should be prevented at all costs, and semi-elective intubation should be considered when expertise is present. Attempts at intubation (or re-intubation) may be able to be postponed until the experienced back-up help is available.

Secondly, the techniques of awake intubation need to be described in more detail. Awake intubation can refer to a blind nasal technique, a fibreoptic technique (usually nasal) and a direct laryngoscopy technique (with local anaesthetic spray and/or minimal sedation). These techniques offer the potential advantage of protecting the airway until it is secured (if it was being protected previously), and the latter technique, of course, is almost always used in cardiac arrests. Awake direct laryngoscopy offers an excellent opportunity to intubate without sedation or paralysis and is a technique that should be learned by all critical care practitioners responsible for airway management. It may not, however, be an appropriate technique for the non-arresting patient with upper airway obstruction, where a surgical airway may be required.

Thirdly, there is no reason that the initial attempt at intubation should not be made incorporating most of the "best attempt laryngoscopy" techniques. Proper planning for the first attempt should allow optimal head positioning, a laryngoscope blade and handle chosen to suit the patient, a stylet to preform the endotracheal tube (a size small enough to be easily passed, e.g. 7.5 in an adult male), and a tracheal manipulation manoeuvre (e.g. backwards, upwards, rightwards pressure).

Fourthly, despite their potential limitations, end-tidal carbon dioxide monitors should always be available, and used to confirm final tracheal placement of the airway device.[5]

The authors should be congratulated on their work, but perhaps the take home message could be re-emphasised: all critically ill patients should be assumed to be difficult intubations, and prevention and preparation are paramount.

Dr. P. Morley
Royal Melbourne Hospital
Parkville
VICTORIA 3050

REFERENCES

1. Caplan RA, Benumof JL, Berry FA, et al. Practice guidelines for the management of the difficult airway. A report by the American society of anesthesiologists task force on management of the difficult airway. Anesthesiology 1993;78:597-602.
2. Caplan RA, Benumof JL, Berry FA, et al. Practice guidelines for management of the difficult airway. http://www.asahq.org/publicationsAndServices/Difficult%20Airway.pdf.
3. Lim MST, Hunt-Smith JJ. Difficult airway management in the intensive care unit: practical guidelines. Critical Care and Resuscitation 2003;5:43-52.
4. Lim MST, Hunt-Smith JJ. Difficult airway management in the intensive care unit: alternative techniques. Critical Care and Resuscitation 2003;5:53-62.
5. Cummins RO, Hazinski MF. Guidelines based on the principle 'First, do no harm'. New guidelines on tracheal tube confirmation and prevention of dislodgment. Resuscitation 2000;46:443-447.

Intravenous magnesium sulphate in acute coronary syndromes: are there any indications?

Intravenous magnesium sulphate has been recommended for numerous non magnesium deficient disorders including atrial and ventricular tachyarrhythmias, acute coronary syndromes, tetanus, phaeochromocytoma, seizures, stroke, cerebral vasospasm, spinal cord injury, pre-eclampsia, eclampsia and asthma.[1]

However, it has only been rigorously investigated in a few of these disorders. For example, in pregnant patients with hypertension and hyperreflexia who are

admitted to hospital for delivery, a loading dose of 4 g (16 mmol) of intravenous magnesium sulphate over 5 min, followed by 1 g/hr (i.e. 4 mmol/hr) and continued for 24 hr after delivery, halves the risk of eclampsia[2] and is more effective than phenytoin in preventing eclampsia.[3] This dose of magnesium sulphate is also more effective than diazepam or phenytoin in controlling eclamptic seizures.[4] Also a meta-analysis,[5] and a recent multicentre randomised controlled trial[6] confirmed that 8 mmol of intravenous magnesium sulphate improved pulmonary function in patients with severe acute asthma. However, in patients with acute coronary syndromes the early enthusiasm for intravenous magnesium sulphate has recently been tempered.

Early studies in normomagnesaemic patients with acute myocardial infarction,[7] including those treated with thrombolytic and aspirin therapy,[8] reported a reduction in the mortality by 24%, and a reduced incidence of left ventricular failure by 25% with intravenous magnesium sulphate (8 mmol in 5 min followed by 30 - 80 mmol over 24 - 48 hr). In a follow-up study (at an average of 2.7 years later), mortality from ischaemic heart disease was reduced by 21% and all-cause mortality by 16%.[9] A meta analysis of magnesium in acute myocardial infarction, reported a 49% reduction in ventricular tachycardia and ventricular fibrillation and a 54% reduction in mortality.[10]

The beneficial effects of magnesium were thought to be due to coronary vasodilation (improving myocardial oxygenation), peripheral vasodilation (reducing myocardial oxygen demand), inhibition of platelet aggregation (antithrombotic effect), myocardial protection against reperfusion injury (reducing excessive intracellular calcium and attenuating free radical generation[11]) and protection against catecholamine-induced myocardial necrosis; all of which could reduce myocardial infarct size and cardiac failure.[12,13] The timing of intravenous magnesium administration in acute myocardial infarction was said to be critical,[14] as magnesium given at a mean of 3 hr after the onset of chest pain appeared to be beneficial whereas magnesium given at a mean of 8 hr was not,[15] indicating that it was required before coronary reperfusion to protect ischaemic myocardium and modulate reperfusion injury when coronary reperfusion occurred.[16]

However, in the fourth International Study of Infarct Survival (ISIS-4), no significant advantage was recorded with intravenous magnesium sulphate (8 mmol in 15 min followed by 72 mmol in 24 hr) in any patient group (e.g. with or without thrombolytic therapy, and in those in whom it was infused before thrombolytic reperfusion had occurred).[17] Yet those who promoted the beneficial effects of magnesium believed that the optimal 24 hr dose of magnesium ranged between 50 - 65 mmol and that doses > 75 mmol (ISIS-4 used 80 mmol/24 hr) may increase mortality (due to an increase in bradyarrhythmias and heart failure),[18] and stated that the use of magnesium in myocardial infarction warranted reexamination.[14,16,19]

A recent prospective, randomised, double-blind, multicentre, magnesium in coronaries (MAGIC) trial in 6213 patients with acute ST elevated myocardial infarction, found that magnesium sulphate (8 mmol i.v. over 15 min followed by 68 mmol over 24 hr, i.e. a total of 76 mmol) administered within 6 hours of the onset of symptoms had no effect on 30 day mortality.[20] While this is 1 mmol greater than the proposed upper limit of 75 mmol and only 4 mmol less than the ISIS-4 trial, the conclusion that "currently there is no evidence to recommend the routine use of magnesium sulphate in patients with acute myocardial infarction",[21] appears to be inescapable. Nevertheless, intravenous magnesium sulphate was not associated with any harm and the statement that "it can continue to be administered for repletion of documented electrolyte deficits and life threatening ventricular arrhythmias such as torsades de pointes", indicated that the MAGIC trial investigators believed that it may still be a useful agent in certain conditions associated with acute coronary syndromes.[20]

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

REFERENCES

1. Connolly E, Worthley LIG. Intravenous magnesium. Critical Care and Resuscitation 1999;1:162-172.
2. The Magpie Trial Collaborative Group. Do women with pre-eclampsia, and their babies, benefit from magnesium sulphate? The Magpie Trial: a randomised placebo-controlled trial. Lancet 2002;359:1877-1890.
3. Lucas MJ, Leveno KJ, Cunningham FG. A comparison of magnesium sulfate with phenytoin for the prevention of eclampsia. N Engl J Med 1995;333:201-205.
4. The Eclampsia Trial Collaborative Group. Which anticonvulsant for women with eclampsia? Evidence from the collaborative trial. Lancet 1995;345:1455-1463.
5. Rowe BH, Bretzlaff JA, Bourdon C, Bota GW, Camargo CA Jr. Magnesium sulfate treatment for acute asthmatic exacerbations treated in the emergency department. Evidence-based Medicine 1999;4:138.
6. Silverman RA, Osborn H, Runge J, et al. IV magnesium sulfate in the treatment of acute severe asthma: a multicenter randomized controlled trial. Chest 2002;122:489-497.
7. Teo KK, Yusuf S, Collins R, Held PH, Peto R. Effects of intravenous magnesium in suspected acute myocardial infarction: overview of randomised trials. Br Med J 1991;303:1499-1503.
8. Woods KL, Fletcher S, Roffe C, Haider Y. Intravenous magnesium sulphate in suspected acute myocardial

infarction: results of the second Leicester Intravenous Magnesium Intervention Trial (LIMIT-2). Lancet 1992;339:1553-1558.
9. Woods KL, Fletcher S. Long-term outcome after intravenous magnesium sulphate in suspected acute myocardial infarction: the second Leicester Intravenous Magnesium Intervention Trial (LIMIT-2). Lancet 1994;343:816-819.
10. Horner SM. Efficacy of intravenous magnesium in acute myocardial infarction in reducing arrhythmias and mortality. Meta-analysis of magnesium in acute myocardial infarction. Circulation 1992;86:774-779.
11. Garcia LA, Dejong SC, Martin SM, Smith RS, Buettner GR, Kerber RE. Magnesium reduces free radicals in an in vivo coronary occlusion reperfusion model. J Am Coll Cardiol 1998;32:536-539.
12. Editorial. Magnesium for acute myocardial infarction. Lancet 1991;338:667-668.
13. Hearse DJ, Stewart DA, Braimbridge MHV. Myocardial protection during ischemic cardiac arrest. J Thorac Cardiovasc Surg 1978;75:877-885.
14. Antman EM. Magnesium in acute MI. Timing is critical. Circulation 1995;92:2367-2372.
15. Casscells W. Magnesium and myocardial infarction. Lancet 1994;343:807-809.
16. Baxter GF, Sumeray MS, Walker JM. Infarct size and magnesium: insights into LIMIT-2 and ISIS-4 from experimental studies. Lancet 1996;348:1424-1426.
17. ISIS-4. ISIS-4: a randomised factorial trial assessing early oral captopril, oral mononitrate, and intravenous magnesium sulphate in 58 050 patients with suspected acute myocardial infarction. Lancet 1995;345:669-685.
18. Galløe A, Gradual N. Magnesium and myocardial infarction. Lancet 1994;343:1286-1287.
19. Seelig MS, Elin RJ. Reexamination of magnesium infusions in myocardial infarction. Am J Cardiol 1995;76:172-173.
20. The Magnesium in Coronaries (MAGIC) Trial Investigators. Early administration of intravenous magnesium to high-risk patients with acute myocardial infarction in the magnesium in coronaries (MAGIC) trial: a randomised controlled trial. Lancet 2002;360:1189-1196.
21. Hennekens CH, Albert CM, Godfried SL, Gaziano JM, Buring JE. Adjunctive drug therapy of acute myocardial infarction - evidence from clinical trials. N Engl J Med 1996;335:1660-1667.

Gastric emptying and nutrition in the critically ill patient

Enteral nutrition (EN) is considered superior to either intravenous fluids alone or total parenteral nutrition (TPN) for the critically ill. However, the evidence for this is not as compelling as is generally believed.[1] Enteral nutrition is associated with less septic complications in trauma victims,[2] but with the advent of antibiotic/antiseptic impregnated central venous lines reducing the incidence of line sepsis,[3] this benefit may be somewhat less. Also feeding studies must now be interpreted in light of recent work on control of blood sugar in the critically ill.[4] Detrimental effects of TPN may be less if blood sugar is aggressively controlled.

Nevertheless, current consensus opinion supports the use of EN over TPN.[5] It is inexpensive and should be commenced early to reduce septic complications and length of hospital stay.[6] However, EN is associated with a number of problems, the most significant of which is failure to achieve nutritional goals. Recent surveys of enteral feeding have reported that only up to 50% of nutritional goals are achieved.[7,8]

The effect of strict delivery of nutritional goals (usually calculated using formulae that were originally derived from measured energy expenditure) on clinical outcomes has never been assessed in the critically ill. It is as yet unknown whether it is clinically important to meet these goals. Over feeding causes fatty liver and hyperglycaemia in patients given TPN and does not avoid the negative nitrogen balance seen in the critically ill. Increasing caloric intake above resting energy expenditure causes fat deposition and does not prevent loss of muscle mass.[9] However, the clinical effect of underfeeding (which is a current clinical reality) compared with careful adherence to nutritional goals has not been adequately studied. Supplementing EN with TPN in an attempt to achieve nutritional goals improves biochemical markers of nutrition,[10] but studies performed so far are too small to demonstrate an effect on clinically relevant outcomes (e.g. mortality).

One retrospective study in obese patients suggested that limiting calories to < 20 kcal/kg (adjusted weight) per day while maintaining protein intake at 2 gm/kg ideal body weight per day reduced ICU length of stay and use of antibiotics.[11] While interpretation of these data are limited, as it was not a randomised controlled trial, it opens up ethical possibilities for more studies on the effect of limiting caloric and protein delivery to the critically ill.

Failure of EN is usually due to delayed gastric emptying as indicated by large gastric aspirates.[7] Measurement of gastric aspirate volume is a simple clinical measurement of gastric emptying that has a reasonable correlation with scintigraphy in the critically ill, although a significant false negative rate can occur as the gastric tube may block.[12] While scintigraphy is the recognised gold standard measurement of gastric emptying this is logistically difficult in the critical care environment and therefore rarely performed. A recent report of scintigraphic studies in unselected ICU patie-

nts demonstrated a marked delay in gastric emptying in 1 in 5 patients.[12]

Other methods of measurement of gastric emptying which have been used in the critically ill patient include breath testing using C^{13} or C^{14} labeled markers. This is a convenient, simple method of measurement and has been validated in noncritically ill and diabetic patients. This method has been used for measurement of gastric emptying and to assess the effect of prokinetic agents in critically ill patients but is as yet not validated in this group.[13-15] Paracetamol absorption, as used in the study published in this journal, is limited by a variable and unpredictable first pass effect in the critically ill making its interpretation difficult, even in cross over studies. Ultrasonography has also been explored in other patient groups but again has its own peculiar problems in the critically ill patient with fluid accumulation in the peritoneal cavity and difficulties in positioning patients.

There are two commonly used techniques for attempting to improve the success of EN: using prokinetic agents and placement of the feeding tube distal to the pylorus.

Prokinetic agents (e.g. metoclopramide, cisapride and erythromycin) improve gastric emptying and the success of feeding in the critically ill.[16,17] The use of cisapride has been restricted because of its arrhythmic side effects and has never been marketed in an intravenous form for this reason. Erythromycin also has the propensity to cause cardiac arrhythmias particularly if used with other drugs that prolong the QT interval (e.g. fluconazole). Little is known about the optimal dose, dose interval and duration of treatment of erythromycin for this indication. Small doses (e.g. 70 mg i.v.),[15] twice daily[17] and for 3 days[18] are effective and may be associated with fewer cardiac side effects and less unwanted antibiotic effects. Metoclopramide is more effective than cisapride[16] but there are no comparative studies of prokinetic agents involving erythromycin. An effective prokinetic agent without antibiotic or arrythmogenic effects would be welcomed. In this edition of the journal a well executed and well written account of an unfortunately underpowered study examining the effect of neostigmine on gastric emptying in the critically ill is published.[19] The results while promising are inconclusive due to small numbers.

An alternative to prokinetic agents is placement of the feeding tube distal to the pylorus. While the stomach in the critically ill is often inert, the pylorus may be overactive and both these factors may contribute to the observed decrease in gastric emptying.[20] Duodenal and small bowel activity continue, although pressure waves and response to feeding are abnormal.[21] Feeding distal to the pylorus may be associated with a reduction in gastrooesophageal regurgitation, an increase in nutrient delivery, a shorter time to achieve desired target nutrition and a lower rate of ventilator-associated pneumonia.[22,23] However, one study comparing erythromycin with postpyloric tube placement found that nutritional success was equivalent for the two techniques.[24]

Postpyloric tube placement is considered a suitable option when gastroparesis resistant to prokinetic agents is preventing adequate gastric feeding. The main difficulty is placement of the tube and usually requires endoscopy with a Seldinger technique. However, various other techniques have been tried including blind placement which has a reported success rate as high as 92%,[25] (although this has not been subsequently replicated and is probably operator dependent). Placement using prokinetic agents,[26,27] flouroscopy,[28] positioning of the patient[27] and gastric air insufflation[29] have also been described. A promising technique (soon to be available on the market) involves using a specially designed tube or insertion wire with a coil at the tip, which allows electromagnetic waves to be monitored by a device placed on the abdominal wall. With this device the passage of the tube can be observed as it passes through the pylorus. This technique has a 90% success rate with a median time of insertion of 14 minutes.[30]

There remain a number of unanswered questions about the place of nutritional care in ICU practice. The pathophysiology of the gut during critical illness is still poorly understood. Furthermore, while it intuitively makes sense to deliver calories to keep up with energy expenditure, and protein to keep up with urinary nitrogen loss (and efforts with prokinetic agents and small bowel feeding are aimed at achieving this), outcome studies on caloric and protein delivery are needed to put these efforts into perspective.

Dr. M. J. Chapman
Department of Anaesthesia and Intensive Care,
Royal Adelaide Hospital, Adelaide
SOUTH AUSTRALIA 5000

REFERENCES

1. Heyland DK. Nutritional support in the critically ill patients. A critical review of the evidence. Critical Care Clinics 1998;14:423-440.
2. Moore F, Feliciano D, Andrassy R, et al. Early enteral feeding, compared with parenteral, reduces postoperative septic complications. The results of a meta-analysis. Ann Surg 1992;216:172-183.
3. Darouiche RO, Raad II, Heard SO, et al. A comparison of two antimicrobial-impregnated central venous catheters. Catheter Study Group. N Engl J Med 1999; 340:1-8.
4. van den Berghe G, Wouters P, Weekers F, et al. Intensive insulin therapy in the critically ill patients. N Engl J Med 2001 345:1359-1367.
5. Jolliet P, Pichard C, Biolo G, et al. Enteral nutrition in intensive care patients: a practical approach. Working Group on Nutrition and Metabolism, ESICM. European

Society of Intensive Care Medicine. Intensive Care Med 1998 24:848-859.
6. Marik PE, Zaloga GP. Early enteral nutrition in acutely ill patients: a systematic review. Crit Care Med 2001 29:2264-2270.
7. de Beaux I, Chapman M, Fraser R, et al. Enteral nutrition in the critically ill: A prospective survey in an Australian intensive care unit. Anaesth Intensive Care 2001;29:619-622.
8. Heyland D, Cook DJ, Winder B, Brylowski L, Van deMark H, Guyatt G. Enteral nutrition in the critically ill patient: a prospective survey. Crit Care Med 1995;23:1055-1060.
9. Hart DW, Wolf SE, Herndon DN, et al. Energy expenditure and caloric balance after burn: increased feeding leads to fat rather than lean mass accretion. Ann Surg 2002;235:152-161.
10. Bauer P, Charpentier C, Bouchet C, Nace L, Raffy F, Gaconnet N. Parenteral with enteral nutrition in the critically ill. Intensive Care Med 2000;26:893-900.
11. Dickerson RN, Boschert KJ, Kudsk KA, Brown RO. Hypocaloric enteral tube feeding in critically ill obese patients. Nutrition 2002;18:241-246.
12. Chapman M, Fraser R, Creed S, et al. Gastric emptying as measured by scintigraphy correlates with gastric aspirates in the critically ill. Abstract In Press Anaesth Intensive Care 2003.
13. Ritz MA, Fraser R, Edwards N, et al. Delayed gastric emptying in ventilated critically ill patients: measurement by ^{13}C-octanoic acid breath test. Crit Care Med 2001;29:1744-1749.
14. Chapman M, Fraser R, De Beaux I, et al. Cefazolin does not accelerate gastric emptying in the critically ill In Press, Intensive Care Med 2003.
15. Ritz M, Fraser R, Chapman M, et al. Two different doses of erythromycin in the treatment of delayed gastric emptying in critically ill patients. Neurogastroenterol Mot 1999;11:205.
16. MacLaren R, Patrick WD, Hall RI, Rocker GM, Whelan GJ, Lima JJ. Comparison of cisapride and metoclopramide for facilitating gastric emptying and improving tolerance to intragastric enteral nutrition in critically ill, mechanically ventilated adults. Clin Ther 2001;23:1855-1866.
17. Chapman MJ, Fraser RJ, Kluger MT, Buist MD, De Nichilo DJ. Erythromycin improves gastric emptying in critically ill patients intolerant of nasogastric feeding. Crit Care Med 2000;28:2334-2337.
18. Bensaid S, Perrin-Gachadoat D, Burdin M, Boiteau R, Tenaillon A. Erythromycin and early enteral nutrition in mechanically ventilated patients. Crit Care Med 2002;30:1237-1241.
19. Lucey MA, Patil V, Girling K, Jacques T, O'Leary M. Does neostigmine increase gastric emptying in the critically ill? - results of a pilot study. Critical Care and Resuscitation 2003;5:14-19.
20. Dive A, Moulart M, Jonard P, Jamart J, Mahieu P. Gastroduodenal motility in mechanically ventilated critically ill patients: a manometric study. Crit Care Med 1994;22:441-447.
21. Toumadre JP, Barclay M, Fraser R, et al. Small intestinal motor patterns in critically ill patients after major abdominal surgery. Am J Gastroenterol 2001;96:2418-2426.
22. Heyland DK, Drover JW, MacDonald S, Novak F, Lam M. Effect of postpyloric feeding on gastroesophageal regurgitation and pulmonary microaspiration: results of a randomized controlled trial. Crit Care Med 2001;29:1495-1501.
23. Heyland DK, Drover JW, Dhaliwal R, Greenwood J. Optimizing the benefits and minimizing the risks of enteral nutrition in the critically ill: role of small bowel feeding. J Parenter Enteral Nutr 2002;26(6 Suppl):S51-55.
24. Boivin MA. Levy H. Gastric feeding with erythromycin is equivalent to transpyloric feeding in the critically ill. Crit Care Med 2001;29:1916-1919.
25. Zaloga GP. Roberts PR. Bedside placement of enteral feeding tubes in the intensive care unit. Crit Care Med 1998;26:987-988.
26. Kalliafas S, Choban PS, Ziegler D, Drago S, Flancbaum L. Erythromycin facilitates postpyloric placement of nasoduodenal feeding tubes in intensive care unit patients: randomized, double-blinded, placebo-controlled trial. J Parenter Enteral Nutr 1996;20:385-388.
27. Komenaka IK, Giffard K, Miller J, Schein M. Erythromycin and position facilitated placement of postpyloric feeding tubes in burned patients. Dig Surg 2000;17:578-580.
28. Huerta G, Puri VK. Nasoenteric feeding tubes in critically ill patients (fluoroscopy versus blind). Nutrition 2000;16:264-267.
29. Salasidis R, Fleiszer T, Johnston R. Air insufflation technique of enteral tube insertion: a randomized, controlled trial. Crit Care Med 1998;26:1036-1039.
30. Young RJ, Chapman MJ, Fraser R, Chorley D, Creed S. A novel technique for nasoduodenal feeding tube placement in critically ill patients. Crit Care 2001;5:S56.