

Editorials

Albumin use declining in UK intensive care

In one sense, it is reassuring to observe that intensivists in the UK do have time to read the literature and that many have changed their clinical practice on finding evidence that is contrary to their training and experience. However, in another sense, it is disturbing to observe the ease with which clinical practice can be altered by high profile publications even when their conclusions are clearly not supported by careful scrutiny. Major journal reviewers and editors should reflect upon their powerful ethical obligations to ensure that their product reflects the truth.

In this issue of the Journal, an interesting survey of 292 UK intensive care units is reported.¹ Since the Cochrane Injuries Group Reviewers published a meta-analysis concluding that intravenous albumin resuscitation increases patient mortality by 6%,² albumin issued for use in the UK has fallen by 40%.³ Forty two percent of the 80 intensive care units in the UK that now do not use albumin solutions at all, commenced this practice following the Cochrane British Medical Journal publication. Fifty per cent of 131 units reported that their clinical use of albumin solutions had changed since the controversial paper was published. Reassuringly perhaps, 130 of the 261 responding units reported that their clinical practice had not changed. Finally, and in keeping with just about every other publication on this topic to follow the Cochrane Injuries Group Reviewers paper, the authors conclude that a well-conducted large scale randomised trial of sufficient statistical power is needed now to put the controversy to rest.

Arguably, one of the few positive benefits of the Cochrane albumin analysis,² was that it provided the Australian and New Zealand Intensive Care Society Clinical Trials Group (ANZICS-CTG) with the justification needed to mount and fund one such large randomised trial - the "saline or albumin fluid evaluation (SAFE)" study. This randomised, blinded study of 7,000 intensive care unit patients will commence in 19 Australian and New Zealand intensive care units in June and July, 2001. Clearly there will be difficulties, and design can never be perfect. But this study will be the best that 2 years of discussion and negotiation around Australasia could achieve, and the conclusions will be unquestionably more reliable than any preceding effort. Those who become preoccupied with blinding issues should remember that the primary

goal is to ensure that allocation concealment is absolute. Anything further gained would be a bonus, which should enhance the trial's reliability, and would move it even further beyond all previous efforts on this subject.

It is hoped that in 2 years time this randomised trial will report that baseline equivalence was impeccable (owing to true randomisation, allocation concealment, and absence of bias) and that both groups have equivalent indices of preload and resuscitation adequacy. For this the authors will be largely dependent upon central venous pressures. Hopefully, the study does not conclude that under- or over- resuscitation with one fluid is worse than adequate resuscitation with another.

The trial organisers would urge Australia's many bright minds to relax, to resuscitate their patients to their usual sensible physiological goals, and not to be frustrated by the idiosyncrasies of the new masked bottles, the green pump sets and the trial obligations. ANZICS-CTG and Australian and New Zealand intensive care both have much to gain from ensuring this huge venture is a practical success.

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REFERENCES

1. Brown JM, McCormick BA, Vella K, Rowan K. Use of albumin in intensive care units in the United Kingdom. *Crit Care Resus* 2001;3:22-29
2. Cochrane Injuries group Albumin reviewers. Human albumin administration in critically ill patients: a systematic review of randomised controlled trials. *Brit Med J* 1998;317:235-240
3. Roberts I, Edwards P, McLelland B. Use of albumin in UK fell substantially when systematic review was published. *Brit Med J* 1999;318:1643

Has renal-dose dopamine finally been relegated to join the long list of medical myths?

"All who drink of this remedy will recover...except those whom it does not help, who will die. Therefore, it is obvious that this remedy fails only in incurable cases"

Galen

Dopamine may now belong to the folklore of critical care medicine, which is probably the fault of the Medline-fed beast called evidence-based medicine that is gobbling up so many of the comforting certainties of "the faith". However, is such loss of status warranted? Or is its use just going through another stage in the passing parade of medical fashions?

Intravenous dopamine and the things we know

The use of dopamine overtime has been sustained by several physiological observations.¹ First, when given intravenously at 4 - 8 µg/kg/min, dopamine increases heart rate, cardiac output and, sometimes, blood pressure in normal mammals and also in patients with various types of shock. This effect has been attributed to stimulation of β-adrenergic receptors. When given intravenously at higher doses (from 15 µg to 40 - 50 µg/kg/min) dopamine typically increases blood pressure in normal mammals as well as in some patients with shock, an effect that has been attributed to stimulation of α-adrenergic receptors. In the gray area between 8 and 15 µg/kg/min a variable and unpredictable combination of so-called β and α effects is often seen. More controversially, at lower doses (1 - 2 µg/kg/min) when given intravenously to volunteers or normal mammals, dopamine exerts a diuretic and natriuretic effect, which is associated with decreased proximal tubular sodium reabsorption and increased renal blood flow in the absence of detectable systemic haemodynamic effects.^{2,3} These effects have been attributed to stimulation of renal dopaminergic receptors.

On the basis of such physiological observations in normal volunteers, low-dose dopamine is often administered to patients considered at risk of renal failure to augment renal blood flow and preserve glomerular filtration rate.⁴ As renal ischaemia is believed to be the leading cause of acute renal failure,⁵ low-dose dopamine would be expected to increase renal blood flow and help preserve renal cellular oxygenation, glomerular filtration rate and urine output. These beneficial effects would then prevent acute renal failure (ARF).

However, dopamine is also a proximal tubular diuretic and it increases the presentation and reabsorption of chloride by the ascending limb of the loop of Henle. This may inadvertently increase medullary oxygen consumption and exacerbate medullary ischaemia.⁵

Several animal studies have evaluated the effect of dopamine as a renal protective drug.⁴ However, in none, was dopamine the sole nephroprotective agent and any beneficial effects reported were associated with doses higher than 3 µg/kg/min (i.e. with a β-adrenergic effect increasing the cardiac output, heart rate and blood pressure). Such changes in systemic haemodynamics can be achieved with other β-adrenergic agents (e.g.

dobutamine or dopexamine), are not unique to dopamine and are likely to affect renal blood flow indirectly.

Low-dose dopamine has also been investigated in the prevention of renal failure in high-risk clinical settings.⁶⁻⁹ However, all of these clinical studies were of insufficient statistical power, only a few were randomised, double-blinded and placebo-controlled in design and "low-dose" dopamine was usually administered at 3 µg/kg/min. Any beneficial effect that may occur under such circumstances would then not be unique to dopamine, but would most likely reflect the impact of an increased cardiac output on renal blood flow. Indeed a randomised, controlled, double-blind crossover comparison of the effect on glomerular filtration rate (GFR) of low-dose dobutamine and low-dose dopamine showed that dobutamine at 200 µg/min improved GFR, while dopamine did not.¹⁰

The controversy

The considerable doubts concerning the efficacy of low-dose dopamine as a renal protective agent have been counterbalanced by the perception that dopamine is extremely safe. However, dopamine decreases serum prolactin concentrations thus inducing a transient decrease in T-cell function,¹¹ decreases growth hormone secretion and thyrotropin release.¹² Low-dose dopamine may also induce mucosal hypoxia in haemorrhagic shock,¹³ depress the respiratory drive and cause arrhythmias. These effects are unpredictable because dopamine clearance and metabolism are altered in acutely ill patients, with extreme variability in plasma dopamine concentrations.¹⁴ In the light of the above observations, it is not surprising that the administration of low-dose dopamine has become the subject of much controversy.¹⁵⁻¹⁶

In response to the "low-dose dopamine controversy", the Australian and New Zealand Intensive Care Society - Clinical Trials Group (ANZICS-CTG) recently conducted a multicenter, randomised, double-blind, placebo-controlled trial of its use in patients at risk of acute renal failure (ARF).¹⁷ The study found that patients randomised to dopamine reached a peak serum creatinine concentration equal to that of patients randomised to placebo. It also found no differences in other clinical markers of renal function such as plasma urea, urine output, need for renal replacement therapy and number of patients who reached a serum creatinine greater than 300 µmol/L. Taken in their aggregate, these findings support the view that low-dose dopamine does not confer a clinically significant degree of renal protection in critically ill patients at risk of renal failure.

The future

Is the ANZICS study going to sound the death knell for so-called renal dose dopamine? I doubt it. Some

clinicians will argue that if dopamine is administered at slightly higher doses (3 to 5 $\mu\text{g}/\text{kg}/\text{min}$), safety is preserved and its benefits will become apparent. At such doses, dopamine has predictable systemic β -adrenergic effects, which increase heart rate, cardiac output and, in some cases, blood pressure.¹⁵ Such effects must be good for kidneys and patient. It is true that increasing blood pressure and cardiac output are likely to increase renal blood flow. On the other hand, these effects are not unique to dopamine and can be achieved with other β -adrenergic agents. However, most Australian hospitals accept the administration of dopamine at < 5 - 6 $\mu\text{g}/\text{kg}/\text{min}$ in the general wards but not that of dobutamine, adrenaline or noradrenaline, and dopexamine is not available in this country. Under such logistic circumstances, clinicians can only use the drug that is available.

Should all patients who require dopamine in the wards because they are hypotensive enough or oliguric enough, or both, be nursed in a monitored environment (e.g. intensive care, coronary care or high dependency unit)? My view is that they should. But that's another issue.

Will the findings of the ANZICS trial and the associated scepticism about renal-dose dopamine spread to the other uses of dopamine? Unlikely. Will the β -adrenergic dose of dopamine be progressively replaced by phosphodiesterase inhibitors, dobutamine or low dose adrenaline in cardiac patients? Maybe. Does anyone still use α -dose dopamine out there? It certainly looks like that if one reads the North American literature, where dopamine continues to be recommended as the agent of first choice for septic shock,¹⁸ despite its inferior physiological performance in this setting in a well conducted phase II trial comparing it with noradrenaline.¹⁹

The problem with dopamine is that there has never been (until now) a phase III study of its clinical use and that all the phase I to II equivalent studies conducted in intensive care units so far often show it to be no better, or occasionally worse, than other agents in achieving "desirable" physiological outcomes. But then the relationship of such physiological outcomes to the "big five" (i.e. duration of ventilation, need for renal replacement therapy, length of stay in intensive care, length of stay in hospital and mortality) remains unclear. Furthermore, all the 'would be' substitutes for dopamine are guilty of the same sin, i.e. no randomised controlled trials of suitable power and outcome. Finally, no case has yet been made that getting a cardiac output of X and a blood pressure of Y with drug A is different from achieving the same goals with drug B. In fact, we don't even know what the haemodynamic goals should be! In the absence of such information, practice will continue to be mostly based on heuristic bias, habit,

mentorship, unit folklore and so on. Is this a bad thing? We do not know, because there are no randomised controlled trials comparing protocol-based vasoactive drug therapy versus judgement-based vasoactive drug therapy.

So, wither dopamine now? I expect that dopamine will remain more commonly prescribed in smaller intensive care units and by older practitioners but still widely used. In fact, now that I think about it, I did prescribe it once, last year.

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REFERENCES

1. Girbes ARJ, Smit AJ. Use of dopamine in the ICU. Hope, hype, belief and facts. *Clin Exper Hypertension* 1997;19:191-199.
2. McDonald R, Goldberg L, McNay J, Tuttle E. Effects of dopamine in man: augmentation of sodium excretion, glomerular filtration rate and renal plasma flow. *J Clin Invest* 1964;43:1116-1124.
3. Hollenberg NK, Adams DF, Mendall P, et al. Renal vascular responses to dopamine: hemodynamic and angiographic observations in normal man. *Clin Sci* 1973;45:733-742.
4. Denton MD, Chertow GM, Brady HR. "Renal-dose" dopamine for the treatment of acute renal failure: scientific rationale, experimental studies and clinical trials. *Kidney Int* 1996;49:4-14.
5. Brezis M, Rosen S, Silva P, Epstein FH. Renal ischemia: a new perspective. *Kidney Int* 1984; 26: 375-383.
6. Baldwin L, Henderson A, Hickman P. Effect of postoperative low-dose dopamine on renal function after elective major vascular surgery. *Ann Intern Med* 1994;120:744-747.
7. Myles PS, Buckland MR, Schenk NJ, et al. Effect of "renal-dose" dopamine on renal function following cardiac surgery. *Anaesth Intens Care* 1993;21:56-61.
8. Swygert TH, Roberts LC, Valek TR, et al. Effect of intraoperative low-dose dopamine on renal function in liver transplant patients. *Anesthesiology* 1991;75:571-576.
9. Weisberg LS, Kurnik PB, Kurnik BR. Dopamine and renal blood flow in radiocontrast-induced nephropathy in humans. *Renal Failure* 1993;15:61-68.
10. Duke GJ, Briedis JH, Weaver RA. Renal support in critically ill patients: low-dose dopamine or low-dose dobutamine? *Crit Care Med* 1994;22: 1919-1925.
11. Devins SS, Miller A, Herndon BL, O'Toole L, Reisz G. effects of dopamine on T-lymphocyte proliferative responses and serum prolactin concentrations in critically ill patients. *Crit Care Med* 1992;20:1644-1649.
12. Van den Berghe G, de Zeager F. Anterior pituitary function during critical illness and dopamine treatment. *Crit Care Med* 1996;24:1580-1590.
13. Segal JM, Phang PT, Walley KR. Low-dose dopamine

- hastens onset of gut ischemia in a porcine model of hemorrhagic shock. *J Appl Physiol* 1992;73:1159-1164.
14. Juste RN, Moran L, Hooper J, Soni N. Dopamine clearance in critically ill patients. *Intensive Care Med* 1998;24:1217-1220.
 15. Thompson BT, Cockrill BA. Renal-dose dopamine: a Siren song. *Lancet* 1994; 344:7-8.
 16. Kellum JA. The use of diuretics and dopamine in acute renal failure: a systematic review of the evidence. *Crit Care* 1997;1:53-59.
 17. ANZICS Clinical Trials Group. Low-dose dopamine in patients with early renal dysfunction: a placebo-controlled randomised trial. *Lancet* 2000;356:2139-2143.
 18. Astiz ME, Rackow EC. Septic shock. *Lancet* 1998;351:1501-1505.
 19. Martin C, Papazian L, Perrin G, Gisou G. Norepinephrine or dopamine for the treatment of hyperdynamic septic shock. *Chest* 1993;103:1826-1831.

Futility - beyond a definition

The medical concept of futility has been devised over the last decade in an attempt to enable physicians to withhold or withdraw care deemed to be inappropriate, without subjecting such a decision to patient approval.¹ Once such a determination was made the physician could be allowed to withdraw therapy, even over the objections of the patient or family. Thus, a determination of futility could take precedence over patient autonomy and obviate patient or surrogate demands for therapy that was considered inappropriate by the attending clinician.

However, futility has proven difficult to define and consensus on a definition has not been established.² Schneiderman *et al*, distinguished between quantitative and qualitative futility such that, if a medical treatment had been useless in the last 100 cases, the treatment should be considered quantitatively futile, while qualitative futility referred to treatments that maintain permanent unconsciousness or dependence on intensive medical care.¹ Others attempted to determine a threshold of likelihood of survival below which treatment could be considered futile.³⁻⁵ However, consensus was not reached on the threshold, and all attempts at a definition based on a quantitative premise failed because of the inability of physicians to predict survival accurately. Even the most sophisticated prognostic scoring systems such as APACHE III, MPM II, and SAPS II, could not be used in definitions of futility because they are based on population statistics and should not be applied to individual patients.⁶⁻¹⁰

Essentially, all of the definitions of futility that have been proposed can be considered in four conceptual categories:

1. physiologic futility: the therapeutic intervention does not have its intended physiologic effect.
2. imminent demise futility: the patient will die regardless of the therapy.
3. lethal condition futility: the patient has a condition incompatible with long term survival regardless of therapy.
4. qualitative futility: the result will be an unacceptable quality of life.

Halevy and Brody suggested that, to be useful, a definition of futility should be prospective, precise, socially acceptable and must apply to a significant number of patients.¹¹ Using these criteria they examined the futility definitions described in the literature and found that no definition satisfied all of the criteria. They concluded that "any futility policy based on a substantive definition was unworkable".

The Ethics Committee of the Society of Critical Care Medicine suggested that only treatments that have no physiologic benefit should be labeled as futile.¹² Given this definition, futile treatments are rare and the concept of futility is not useful in establishing policies to limit treatment. The Council on Ethical and Judicial Affairs of the American Medical Association also noted great difficulty in defining futility since it was felt to be inherently value-laden.¹³ The problem is that each patient's circumstance is unique, which results in judgements that do not fit into a general definition of futility.

Conflicts between the clinicians and patients or families over decisions to withdraw therapy occur when there is disagreement about whether the desired goal of therapy is appropriate or whether the likelihood of success is adequate. Since these conflicts are about differences in values rather than differences in facts, is it really valid to label such therapies as futile?

The courts in Australia and other jurisdictions have not recognised the right of doctors to make unilateral decisions regarding withdrawal of "futile" treatment. In *Marchlewski v Hunter Area Health Service*, the New South Wales Supreme Court found against the doctors who had withheld therapy in a brain injured neonate against the wishes of her parents, "notwithstanding what would appear to the Hospital to be a meaningless prolonging of human life".¹⁴ In the case of John Thompson, it would appear that Justice O'Keefe ordered the reversal of a decision to withdraw therapy from a man with hypoxic brain injury because of the objection of the patient's family.¹⁵ Overseas, in almost every case where the patient or family have objected to a medical decision to withdraw or withhold therapy, the courts have found in favour of the patient.¹⁶ It appears

that the courts are supporting the rise of patient autonomy and the decline of medical paternalism.

It is clear that “futility” means many things to many people and it continues to defy definition, despite years of debate in the literature. Such a situation is likely to result in confusion, misinterpretation and potential litigation. Rather than making continued efforts to redefine futility and achieve consensus, reference to “futile” treatment should be avoided completely. The use of the concept of futility as a basis for withdrawal of therapy has not proven to be useful. It should not, and does not, remove the duty of the doctor to talk to the patient and family, and to explain that further treatment will be of no benefit.

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REFERENCES

1. Schneiderman LJ, Jecker NS, Jonsen AR. Medical futility: its meaning and ethical implications. *Ann Int Med* 1990;112:949-954.
2. Helft PR, Siegler M, Lantos J. The rise and fall of the futility movement. *N Engl J Med* 2000;343:293-296.
3. Poses RM, Bekes C, Copare FJ, Scott WE. The answer to “What are my chances, doctor?” depends on whom is asked: prognostic disagreement and inaccuracy for critically ill patients. *Crit Care Med* 1989;17:827-833.
4. Curtis JR, Park DR, Krone MR, Pearlman RA. Use of the medical futility rationale in do-not-attempt-resuscitation orders. *JAMA* 1995;273:124-128.
5. McCrary SV, Swanson JW, Youngner SJ, Perkins HS, Winslade WJ. Physicians' quantitative assessments of medical futility. *J Clin Ethics* 1994;5:100-5.
6. Knaus WA, Wagner DP, Lynn J. Short term mortality predictions for critically ill hospitalised patients; science and ethics. *Science* 1991;254:289-394.
7. Teres D, Lemeshow S. Why severity models should be used with caution. *Crit Care Clin* 1994;10:93-110.
8. Luce JM, Wachter RM. The ethical appropriateness of using prognostic scoring systems in clinical management. *Crit Care Clin* 1994;10:229-241.
9. Lemeshow S, Clark J, Teres D. Outcome predication for individual intensive care patients: useful, misused, abused? *Intens Care Med* 1995;21:770-776.
10. The Ethics Committee of the Society of Critical Care Medicine. Consensus statement of the Society of Critical Care Medicine's Ethics Committee regarding futile and other possibly inadvisable treatments. *Crit Care Med* 1997;25:887-891.
11. Halevy A, Brody BA. A multi-institutional collaborative policy on medical futility. *JAMA* 1996;276:571-574.
12. The Ethics Committee of the Society of Critical Care Medicine. Consensus statement of the Society of Critical Care Medicine's Ethics Committee regarding futile and other possibly inadvisable treatments. *Crit Care Med* 1997;25:887-891.
13. Council on Ethical and Judicial Affairs, American Medical Association. Medical futility in end of life care: Report of the Council on Ethical and Judicial Affairs. *JAMA* 1999;281:937-941.
14. *Marchlewski v Hunter Area Health Service* (Unreported, NSW Supreme Court, Dowd J, 14 August 1998) NSWSC 771.
15. Back from the dead after sister's plea. *The Advertiser*. May 4, 2000.
16. Daar JF. Medical futility and implications for physician autonomy. *Am J Law Med* 1995;21:221-40.

Future requirements for quality assurance, assessment and reporting in Australasian ICUs

Three recent contributions to this journal address quality assurance (QA) issues. Fraenkel¹ comments on adverse events analysis and reviews Australian council for health care standards (ACHE) clinical indicators, the national intensive care database and the now defunct national Australian incident monitoring systems in intensive care units (AIMS-ICU system). Worthley² discusses adverse events and suggests that quality of care incorporates three aspects: 1. customisation, 2. provision of services consistent with current knowledge and 3. provision of service free from accidental injury. McHugh³ raises three other important issues - 1. the optional approach to QA in level II and small intensive care units (ICUs), 2. the utility of audit, and 3. the resources for audit.

The contributions take ICU centred views. However, Fraenkel¹ points, out that “safety and quality issues are not going to fade away and whatever the preferences and fears of clinical practitioners, some degree of public reporting and accompanying accountability seems almost inevitable” Driven by the need to play a wider role in hospital services,⁴ by potential requirements for active clinical governance,⁵ and the spread of the concept of “Report Cards”,⁶ ICUs are likely to be, if they are not already, subject to pressure from the community, the media, and various hospital health care area or state government quality councils, committees, etc to demonstrate that:

- there is data capture relevant to the type of service that the intensive care unit (ICU) provides
- from the data, it is possible to measure risk adjusted outcomes (not only mortality) and customer satisfaction (e.g. patients, families, staff, managers)

- the data are used to change practices, improve outcomes and increase customer satisfaction
- performance is compared to a defined set of theoretical optimal practice indicators (e.g. best practice guidelines, event thresholds, performance indicators, standards, clinical indicators, etc)
- evaluation is undertaken of the impact of the quality of care before admission to ICU on the outcomes of care in the ICU, and of the clinical course of patients after discharge from ICU and the long term outcomes after discharge from hospital.

Whether the pressures will have a positive or negative impact on the quality of care and outcomes in ICU's remains to be seen.

What is high quality intensive care? The Faculty of Intensive Care, Australian and New Zealand College of Anaesthetists (FICANZCA) defines it as "an organised process that assesses and evaluates health services to improve practice or quality of care".⁷

There are many definitions of the quality of general health care. A useful one is given by the international organisation for standardisation in ISO 8402⁸ - "the totality of features and characteristics of a product or service that bear on its ability to satisfy stated or implied needs". The "stated or implied needs", in the case of ICU's could become the foci of quality assessment and reporting. A putative but unproven list of the implied needs of different stakeholders is:

PATIENT

- Highest probability of survival consistent with pre-admission problems
- Relief of suffering and prevention of further suffering during management
- If survival cannot be achieved, a dying process free of suffering
- Consultation in end of life decisions
- Maintenance of dignity as a person
- Interruption of the precipitating disease process by error free management
- Return to the quality of life before the acute episode
- Earliest access to life saving resources and/or admission to the ICU without delay which could decrease probability of survival
- Equity in access to life saving resources within the ICU

FAMILIES⁹

- Assurance of quality of care
- Up to date information
- Proximity to the patient in place and time
- Support from, and sensitivity of, staff members
- Knowing the staff members and having confidence in them
- Comfort in waiting areas

- Consultation in decision making

STAFF

- Satisfaction of the patient's needs
- Access to evidence for practice
- Accurate diagnosis
- Appropriate, effective interventions
- Optimal patient outcome
- Provision of holistic care
- Provision of incident-free care
- Intellectual challenge
- Sense of being in control
- Professional development
- Use of new technologies
- Challenging interventional procedures
- Achievement of best practice
- Status as the best of the best

FICANZCA, ANZICS and ACHS

- Appropriate physical and organisational structures for quality practice and reporting
- Professional development of trainees in quality practice and assessment
- Development and use of relevant guidelines and standards

MANAGERS

- Cost effective practice and staffing
- Satisfaction of guidelines or standards that lead to hospital and/or ICU accreditation
- Minimal clinical pathway variance
- Elective clinical governance
- Coordination of ICU and hospital wide assessment of ICU practice (eg. medication errors, infection control)

What is an appropriate framework for needs based quality assessment and reporting? The New South Wales health department¹⁰ has described such a framework. It appears applicable to ICU's. In the framework, there are six performance areas - safety, effectiveness, appropriateness, consumer participation, efficiency and access. There are also five cross dimensional issues - competence of personnel, information management, continuity of care, education and training for quality and accreditation. It is tempting to propose that a model could be developed from a combination of the perceived needs approach and the NSW quality framework to provide a goal for strategies to develop optimal quality assessment and reporting in ICU's. The model would be applicable to level 1, 2 and 3 ICU's¹¹ once a unit has defined its patient population and casemix. FICANZCA's guidelines on quality assurance⁷ and the quality assurance section of the maintenance of professional standards guidelines¹² provide some presently suggested QA activities but not the reporting framework.

Whilst the theoretical models for assessing and reporting QA continue to develop¹³ and many units conduct high quality QA, there are difficulties. The Australian and New Zealand intensive care society (ANZICS) survey¹⁴ for the period 1995-1997 found problems in the organisational structure and staffing for QA in some of the responding ICU's. There were also problems with limited access to information technology. Until the survey is repeated it must be assumed that such problems still exist in some, if not many, units.

Quality intensive care is probably based on:

- a unit culture built around the goal of achieving excellence and achieving the optimal outcome for the patient with a unit organisation or structure that facilitates excellence
- the use of valid evidence that is applicable to the unit's population of patients to drive clinical management
- the collection of data to assess the outcomes of care and the quality of processes, with the use of the data to improve both
- where it is difficult to measure outcomes of care, the measurement of valid surrogate markers
- the sharing of the information derived from data amongst all members of staff who impact on the quality of care
- the regular performance of research or clinical "small change" studies¹⁵
- an establishment of staff that are willing and able to pursue the first six principles under effective leaders committed to high quality care
- the provision of adequate resources to the unit to achieve the principles.

However, there are barriers to the achievement of the principles in some units. For example:

- there is growing debate on the optimal outcome of intensive care for some subgroups of patients
- there is uncertainty on whether we are measuring all the outcomes that are important to the patient and families
- resources (computers, software, people, time) to undertake optimal data capture and use may be inadequate in some units and there may be competition with resources for clinical management
- in some areas, there is difficulty in the attraction and retention of intensive care nurses. Paradoxically, whilst the understaffed units are most in need of data to assess the quality of care, well staffed units for clinical care are likely to have most resources in quality activities.
- outcomes in ICU's may be impacted upon by the quality of care in the hospital wards from which they

are admitted to ICU and the wards to which they are discharged to return to ICU at a later date.^{16,17,18}

Smaller units have particular problems.³ There are wide variations in casemix, severity of illness and throughput between such units. Annual throughput may be too small to allow staff to link changes in practice to changes in outcomes. Tailoring quality assessment, to local activities, casemix and objectives would appear to be essential. Audit may be less useful than adverse event monitoring and specific local clinical indicators may be necessary. Benchmarking outcomes with similar ICU's rather than large tertiary referral hospital units may be a better option. However, the principles outlined previously in the article would appear to apply to the smaller units.

As a specialty group, intensive care specialists have shown they wish to be in the forefront of the provision of high quality clinical care in units with appropriate organisational and physical structures. The development of data capture and sharing of data on quality has been in advance of many specialties. However, pressures will continue to mount for intensivists to show extramural groups that the expensive, high technology ICU care changes outcomes favourably in various subgroups of patients and those outcomes and the quality of life which result are acceptable to the Australasian community. The reports required will need many ICU's to adopt a wider vision of their role in the continuum of hospital care and their contribution to long term outcomes.

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REFERENCES

1. Fraenkel DJ. Safety and quality in intensive care. *Critical Care and Resuscitation* 2000;2:220-226.
2. Worthley LIG. Quality control, audit, adverse events and risk in the intensive care unit. *Critical Care and Resuscitation* 2000;2:304-307.
3. McHugh G. Development of audit and quality assurance in an intensive care unit. A smaller hospital perspective. *Critical Care and Resuscitation* 2001, 3:52-55.
4. Hillman KM. Restructuring hospital services. *Med J Aust* 1998;169:239.
5. Ayres P. Clinical governance: setting the scene. *Hospital Medicine* 1999;60:505-508.
6. Schneider EC, Epstein AD. Use of public performance reports. *JAMA* 1998;279:1638-1642
7. Faculty of Intensive Care, Australian and New Zealand College of Anaesthetists Quality Assurance 2000; Review IC-8 FICANZCA Melbourne.
8. International Organisation for Standardisation IS08402 quoted in Australian/New Zealand Standard AS/NZS

- 3905;2:1993 Quality System Guidelines; Homebush and Wellington.
9. Wasser T, Pasquale MA, Matchett SC, Bryan Y, Pasquade M. Establishing reliability and validity of the Critical Care Family Satisfaction Survey. *Crit Care Med* 2001;29:192-196.
 10. NSW Health Department. A framework for managing the quality of health services in New South Wales. 1999. Better Health Centre, Gladesville (website <http://www.health.nsw.gov.au>)
 11. Faculty of Intensive Care, Australian and New Zealand College of Anaesthetists. Minimum Standards for Intensive Care Units 1997; Review IC-I FICANZCA Melbourne.
 12. Faculty of Intensive Care, Australian and New Zealand College of Anaesthetists Maintenance of Professional Standards 2000, FICANZCA Melbourne.
 13. Glezeman M, Witznitzer A, Reuvene H, Mazor M. A model of efficient and continuous quality improvement in a clinical setting *Int J Qual Health Care* 1999;11:227-232
 14. Steed K, Warnecke K, Hart G. Australian and New Zealand Intensive Care Society - Intensive Care Unit Registry Report, Descriptive analysis of quality characteristics of Australasian Critical Care facilities 1998; ANZICS, Melbourne.
 15. Rainey TG, Kabcenell A, Bruick DM, Roessner. Reducing costs and improving outcomes in adult intensive care 1998; Institute for Healthcare Improvement, Boston.
 16. Hillman KM. Reducing preventable deaths and containing cost: the expanding role of intensive care medicine. *Med J Aust* 1996;164:308-309.
 17. McQuillan P, Pilkington S, Allan A, et al. Confidential inquiry into quality of care before admission to intensive care *BMJ* 1998;315:1853-1858.
 18. Chen LM, Martin CM, Keenan SP, Sibbald WJ. Patients readmitted to the intensive care unit during the same hospitalisation: Clinical features and outcomes. *Crit Care Med* 1998;26:1834-1841.

Imaging and the intensivist

In the critically ill, ventilated, multi-organ failure patient, more often than not at some stage of the patient's management, there will be some clinically inaccessible area that will require a non-invasive review. The classical 'blind' spots are abdomen and head, although the chest may also at times warrant closer investigation using computed tomography or echocardiography.

However, it is not uncommon that a technician will perform the investigation, particularly when performed 'out-of-hours', and it will be left up to the intensivist to interpret the result. Head CT scans are a case in point when the unconscious patient is admitted in the early hours of the morning, intubated and mechanically ventilated with a CT scan does not look 'quite right'.

It is with this in mind we have decided to include a section in the Journal called 'Radiology for the non-radiologist'. The first of a series of contributions from Dr. Tie presents a review of head CT scans, their interpretation and the abnormalities commonly found in a critically ill patient.¹ We hope that it is useful.

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REFERENCES

1. Tie M. Basic head CT for intensivists. *Critical Care and Resuscitation* 2001;3:35-44.