

America, and Australia.¹⁰ The belief that continuous monitoring of the patient is beneficial has led to recommendations throughout the world that it should be used whenever possible.⁵

Equally, it is accepted that oxygen saturation should not be permitted to fall below 90%—that is, an arterial oxygen tension of 7.9 kPa—as below this value a minor fall in oxygen tension results in a rapid fall in oxygen content. Pulse oximeters are subject to error, but they are convenient and accurate in most cases when used within the clinical range and will detect hypoxaemia. A review of 2000 anaesthetic incidents showed that pulse oximetry detected more incidents than any other monitor. The authors of this study considered that proper use of pulse oximetry would have alerted the anaesthetist in over 80% of all applicable incidents during anaesthesia and recovery had the incidents remained undetected by other means.¹¹

Many endoscopic techniques require sedation and thus carry the risk of loss of consciousness. Every effort should be made to ensure patients' safety, and this should include the routine use of oxygen supplementation and monitoring of arterial oxygen supplementation by pulse oximetry. Indeed, these recommendations should be seen as a bare minimum, as a mortality of one in 2000 requires urgent action. If any doubt exists about the use of supplemental oxygen and pulse oximetry it follows that the only logical step to improve patients' safety during endoscopy is to widen the remit of the

national confidential inquiry into perioperative deaths. This will permit thorough assessments of all factors contributing to mortality and morbidity after endoscopy.

J E CHARLTON

Consultant in pain management and anaesthesia

Department of Anaesthesia,
Royal Victoria Infirmary,
Newcastle upon Tyne NE1 4LP

- 1 Bell GD, McCloy RF, Charlton JE, Campbell D, Dent NA, Gear MWL, *et al*. Recommendations for standards of sedation and patient monitoring during gastrointestinal endoscopy. *Gut* 1991;32:823-7.
- 2 Royal College of Surgeons of England. *Commission on the provision of surgical services. Report of the working party on guidelines for sedation by non-anaesthetists*. London: Royal College of Surgeons, 1993.
- 3 Bell GD, Reeve PA, Moshiri M, Morden A, Coady T, Stapleton PJ, *et al*. Intravenous midazolam: a study of the degree of oxygen desaturation occurring during upper gastrointestinal endoscopy. *Br J Clin Pharmacol* 1987;23:703-8.
- 4 Murray AW, Morran CG, Kenny GNC, Anderson JR. Arterial oxygen desaturation during upper gastrointestinal endoscopy. *Gut* 1990;31:270-3.
- 5 Association of Anaesthetists of Great Britain and Ireland. *Recommendations for standards of monitoring during anaesthesia and recovery. Revised edition 1994*. London: AAGBI, 1994.
- 6 Quine MA, Bell GD, McCloy RF, Charlton JE, Devlin HB, Hopkins A. Prospective audit of upper gastrointestinal endoscopy in two regions of England: safety, staffing, and sedation methods. *Gut* 1995;36:462-7.
- 7 Bell GD, Morden A, Brown S, Coady T, Logan RFA. Prevention of hypoxaemia during upper gastrointestinal endoscopy by means of oxygen via nasal cannulae. *Lancet* 1987;i:1022-3.
- 8 Bowling TE, Hadjiminas CL, Polson RJ, Baron JH, Foale RA. Effects of supplemental oxygen on cardiac rhythm during upper gastrointestinal endoscopy: a randomised controlled double blind trial. *Gut* 1993;34:1492-7.
- 9 Moller JT, Wittrup M, Johansen JH. Hypoxemia in the post anesthesia care unit: an observer study. *Anesthesiology* 1990;73:890-5.
- 10 Runciman WB, Ludbrook GL. Monitoring. In: Nimmo WS, Rowbotham DJ, Smith G, eds. *Anaesthesia*. 2nd ed. Oxford: Blackwell Scientific Publications, 1994:704-39.
- 11 Webb RK, Van der Walt J, Runciman WB, Williamson JA, Cockings J, Russell WJ, *et al*. Which monitor? A review of 2,000 anaesthetic incidents. *Anaesth Intensive Care* 1993;21:529-42.

Wonderful albumin?

Not all it is cracked up to be

Albumin solutions are commonly used to treat low serum albumin concentrations and hypovolaemia. Human albumin effectively replaces volume and supports colloid oncotic pressure. Unlike synthetic colloids, it has transport functions and binds reversibly with anions, cations, and some substances that are active or toxic only in the free form. It is a scavenger of free radicals¹ and improves prognosis in the sheep model of the adult respiratory distress syndrome.² It has anticoagulant properties, inhibiting platelet aggregation and enhancing the inhibition of factor Xa by antithrombin III.^{3,4} It may also have a role in preserving microvascular integrity, which is possibly mediated by glycoproteins distributed through the capillary membrane.⁵

Common reasons for using albumin rather than synthetic alternatives are generally clinical associations and beliefs related to the importance of hypoalbuminaemia. Hypoalbuminaemia is associated with a poor surgical outcome and a longer stay in hospital and is a marker of higher risk in critically ill patients. It is associated with oedema and, by implication, with low serum colloid oncotic pressure. Starling's law is commonly invoked to suggest cause and effect. Serum albumin concentration falls rapidly in critically ill patients. This is often blamed on catabolism or failure of synthesis of albumin, which then implies albumin deficiency and that replacement is necessary.

Unsurprisingly, albumin replacement is popular. It has tremendous theoretical advantages while at the same time either preventing or treating the fall in serum albumin concentrations, which carries such fearful connotations.

But let us consider the evidence for such beliefs. Although hypoalbuminaemia may be associated with a poor outcome, there is always an underlying problem. Correcting this

problem—not the serum albumin concentration—influences the outcome. A return of serum albumin concentration towards normal usually indicates improvement.

Oedema in critically ill patients has the same pathophysiological basis as hypoalbuminaemia; hypoalbuminaemia is usually an effect rather than a cause.⁶ Serum albumin concentration correlates poorly with colloid oncotic pressure,⁷ but Starling stated that, even if the correlation was high, the oncotic gradient—not the absolute plasma value—was important. Albumin distributes across the capillary membrane, thereby minimising the transcapillary gradient, and this process is accelerated in critically ill patients.^{6,8} Consequently, the absolute serum albumin concentration may fall with minimal change in the transcapillary gradient. The fall in serum albumin concentration seen in sepsis, trauma, and major surgery occurs too rapidly to be due to either catabolism or failed synthesis, which together account for less than a tenth of circulating albumin a day. Redistribution is more likely, which is probably also relevant in conditions such as pre-eclampsia.

Hypoalbuminaemia is associated with poor gut motility, but increasing the serum concentration by giving albumin does not increase gut motility. Conversely, correcting the underlying problem, and incidentally improving the serum albumin concentration, improves gut motility.⁹

Should we support a particular range of serum albumin concentrations? Undoubtedly, giving albumin will help to maintain the serum albumin concentration, but if this is a marker of rather than a cause of pathophysiology then why treat it? Several studies have failed to show that treatment with albumin improves outcome.¹⁰⁻¹² One study found more infections in patients whose serum albumin concentrations

were not supported¹³; other studies have failed to confirm this.¹⁰⁻¹²

What of the impressive theoretical arguments for using albumin as the preferred colloid solution? If albumin is effective for binding drugs and toxic substances, is a scavenger of free radicals, and is important for vascular integrity and blood coagulation we would expect it to be better than other agents. As administration of colloid is a substantial component in the management of critically ill patients the long term benefits of albumin should be easily identifiable. Curiously, there is a dearth of studies on this. While many studies have shown the comparative haemodynamic efficacy of albumin and synthetic colloids, few have examined longer term use. One study comparing gelatin and albumin found no difference in outcome, length of stay in an intensive care unit, or requirement for blood products and coagulation factors.¹⁰

Should we use albumin for volume replacement? It is expensive. It is a human product. In critically ill patients its impressive array of theoretical advantages over synthetic agents does not translate into overt clinical benefits. If it is unhelpful in this population then whom is it likely to benefit?

What is the role of albumin solutions? There is no convincing evidence that albumin is better than synthetic alternatives for volume replacement; nor is there clear evidence for maintaining the serum albumin value concentration above a certain level. Currently, the widespread use of albumin has more to do with word association and the treatment of items that are marked on the pathology form

with an asterisk than with scientific medical management. At a time when molecular biology and genetic engineering are the focus of our research we would do well to review critically some of our beliefs about more basic science.

NEIL SONI

Senior lecturer in anaesthesia and intensive care

Magill Department of Anaesthesia,
Chelsea and Westminster Hospital,
London SW10 9NH

- 1 Holt M, Ryall M, Campbell A. Albumin inhibits human polymorphonuclear leucocyte luminol dependent chemiluminescence: evidence for oxygen scavenging. *Br J Exp Pathol* 1984;65: 231-41.
- 2 Emerson T, Redens T, Lindsey C. Human serum albumin pretreatment attenuates the lung dysfunction in the endotoxemic sheep ARDS model. *FASEB J* 1988;2:A977.
- 3 Jorgensen K, Stoffersen E. On the inhibitory effect of albumin on platelet aggregation. *Thromb Res* 1980;17:13.
- 4 Jorgensen K, Stoffersen E. Heparin like activity of albumin. *Thromb Res* 1979;16:573.
- 5 Demling R. Effect of plasma and interstitial protein content on tissue edema formation. *Curr Stud Hematol Blood Transfus* 1986;53:36.
- 6 Fleck A, Raines G, Hawker F. Increased vascular permeability; a major cause of hypoalbuminaemia in disease and injury. *Lancet* 1985;i:781-4.
- 7 Grootendorst AF, van Wilgenburg MH, van de Laet PH, Vander Hoven B. Albumin abuse in intensive care medicine. *Intensive Care Med* 1988;14:554-7.
- 8 Vaughan T, Erdmann A, Brigham K, Woolverton W, Weidner W, Staub N. Equilibration of intravascular albumin with lung lymph in unanaesthetized sheep. *Lymphology* 1979;12:217-23.
- 9 Woods MS, Kelley H. Oncotic pressure, albumin and ileus: the effect of albumin replacement on postoperative ileus. *Am Surg* 1993;59:758-63.
- 10 Stockwell M, Soni N, Riley B. Colloid solutions in the critically ill. A randomized comparison of albumin and polygeline. 1. Outcome and duration of stay in the intensive care unit. *Anaesthesia* 1992;47:3-6.
- 11 Kaminski M J, Williams SD. Review of the rapid normalization of serum albumin with modified total parenteral nutrition solutions. *Crit Care Med* 1990;18:327-35.
- 12 Golub R, Sorrento J, Cantu R, Nierman D, Moideen A, Stein H. Efficacy of albumin supplementation in the surgical intensive care unit: a prospective randomized study. *Crit Care Med* 1994;22:613-9.
- 13 Brown RO, Bradley JE, Bekemeyer WB, Luther RW. Effect of albumin supplementation during parenteral nutrition on hospital morbidity. *Crit Care Med* 1988;16:1177-82.

Professional negligence: a duty of candid disclosure?

Doctors should explain in full when care has gone wrong

Lawyers, whether barristers or solicitors, owe their clients a duty to disclose any conflict of interest that has arisen and to inform their clients that they should thereafter go to another lawyer for advice and help. In legal terms, a conflict of interest arises between a lawyer and client when their interests are no longer the same. There are many ways in which this may arise in practice—for example, if a lawyer has conducted a client's case negligently. Lawyers are in breach of their professional code of conduct if they fail to comply with these duties. Understandably none of us like to find ourselves in such a position; but no client should suffer from a lawyer's mistakes or ineptitude. We all, however, make mistakes. No sensible lawyer thinks that it can never happen to him or her.

Why should the same rule not apply to doctors? Why should it not be part of doctors' ethical code to inform their patients if they have been negligent in their care and treatment? Why should doctors not be subject to disciplinary action if they fail to comply with such a duty?

In 1987 the then master of the rolls, Sir John Donaldson, stated, "I personally think that in professional negligence cases, and in particular in medical negligence cases, there is a duty of candour resting on the professional man. . . . It is but one aspect of the general duty of care, arising out of the patient/medical practitioner or hospital authority relationship. . . ."¹ His views were not, however, necessary for the purposes of deciding the case, and thus far no binding decision of the courts has been given that recognises such a duty in medical practitioners. Should not the medical profession itself recognise such a duty and bring it into effect?

It is invariably the experience of those who practise in the field of medical negligence that if a patient's care has gone

wrong then a full and frank explanation will do much to defuse the anger, upset, and resentment that the patient feels and may substantially reduce the risk that the patient will seek redress in court. Obviously, if major injury has been suffered then, however full the explanation, patients are likely to sue to obtain damages to compensate them—for example, for their continuing loss of earnings or for the cost of future care.

A full and candid explanation will go far to ameliorate the problems that have arisen and also to identify a cause so as to prevent recurrence. A recent study found that one of the four main reasons why people sue doctors is "the need for an explanation—to know how the injury happened and why."² Medical care no longer goes unquestioned; nowadays if something goes wrong patients want to know why.

Although many doctors already give an explanation to their patients, the practice is not universal. The relationship between doctor and patient is based on trust, and doctors may well feel that they do not wish to prejudice that trust or demean themselves in front of their patients or colleagues. In our view, however, the mentality of "never admit anything" (as drivers in car accidents are advised) has no place in the professional world. A doctor would probably wish to discuss the matter with a colleague before giving his or her explanation to the patient and occasionally would want to discuss it first with the defence union. But a prompt explanation is vital; delay will be seen as a cover up.

We believe that the relationship between patient and doctor is likely to be enhanced by a willingness to talk to (not down to) the patient, to explain what has gone wrong, and to show a little humility. Leaving this to "good practice" is not enough; that has not worked in the past. Some doctors will argue that