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''I would have everie man write what he knowes and no more.''—Montaigne

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Editorial

Wet, dry or something else?

The great fluid debate continues to rage. There is still no clear consensus or agreement as to whether the perioperative patient should be treated according to a liberal fluid regimen, or a restrictive one. Fortunately, the evidence base continues to mount, and it is to be hoped that, little by little, we are able to move closer to a logical and evidence-based position.

The debate began a number of years ago in the intensive care unit. Early work by Shoemaker and colleagues[1] demonstrated that patients with supra-normal value for tissue-oxygen delivery tended to have superior outcomes after critical illness as compared with those who did not.[2] This led to the (not unreasonable) hypothesis that therapeutic intervention to increase tissue-oxygen delivery or perfusion would in turn result in improved outcomes in critical illness.[3] Shoemaker and his group pursued this theme over several years, as did other groups. The results of these studies were somewhat mixed.[4] Part of the problem was that there was little standardization of approach of protocol, either in terms of the fluid and inotropic regimens used, or the timing of interventions.

Despite the often conflicting and confusing outcomes of studies in the critical care unit, many groups made the logical leap of extending optimization strategies to the perioperative surgical population.[5] 'Optimization' does not necessarily mean achieving supra-normal pre-defined goals, but is the logical development of that concept—tailoring the goal to the best achievable values (whether for perfusion or oxygen delivery) in the individual patient. Shoemaker's group published a randomized perioperative 'goal-directed therapy' study aiming at supra-normalization, with positive results.[6] Although criticized for lack of standardization of inotropic and optimization strategies, this study led to a flowering of interest in this area, and numerous perioperative 'optimization' studies followed. A number have demonstrated that either inotropes and fluids together, or fluid loading alone can produce a startling reduction in morbidity and mortality extending throughout the duration of the hospital stay and for at least a month after major surgery.[7–]11 Claims have been made that optimization with specific pharmacological agents such as dopexamine may be superior to other agents, or to fluids alone. This has yet to be substantiated, and has been questioned.[12]

At the same time as the widespread interest in fluid optimization,[13] there has been an apparently competing school of thought. Many surgeons have long suspected that excessive perioperative fluid administration may lead to their patients becoming 'waterlogged'. Even those patients who do not appear to be clinically fluid-overloaded may suffer adverse consequences as a result of overenthusiastic hydration. This point of view is well established in thoracic surgical circles, where there is good evidence for liberal fluid regimens contributing to poor outcomes after lung surgery.[14] More recently, there has been growing support for this view in the general surgical population. Recent reports of the National Confidential Enquiry into Perioperative Death have highlighted over-hydration as a contributory cause in the genesis of postoperative problems leading to death. Carefully considered case histories have led to specific recommendations regarding careful fluid management (the implication being restriction) in vulnerable patients and those most at risk, such as the elderly.[15] The evidence goes well beyond the observational, however. There is a growing literature in other surgical populations comparing liberal with restrictive fluid management, many of which show better outcomes in fluid-restricted patients.[16–21]

So how are these two schools of thought to be reconciled? Logically, of course, there may be very little difference between their positions. Let us suppose that the relationship between risk and volume loading is a U- or J-shaped curve (Fig. 1), with perioperative risk (on the *y*-axis) decreasing with increasing volume load (on the *x*-axis), up to a critical point. Beyond this point, further volume loading would result in a rapid increase in the risk of morbidity and mortality. Studies comparing liberal with restrictive fluid policies are comparing risk in patient cohorts to the left and to the right of a vertical line drawn through the curve, parallel to the risk axis and intersecting the fluid-loading axis. If this line is drawn close enough to the nadir of the curve, then it could result in optimal fluid balance. The optimization studies take a different approach; they compare patients below a line drawn parallel to the fluid loading axis and perpendicular to the risk axis, with patients who may be above this line. The ideal approach should define the nadir of the risk curve fairly tightly. This requires physiological measurement tailored to the individual patient in order intuitively more likely to define the volume optimization point. Notably, the fluid volume required to achieve this may vary dramatically between patients subjected to the same protocol.[8]

The second major consideration relates to the timing of fluid administration. In the critical care setting, circulatory optimization is effective at preventing subsequent morbidity and mortality only when it is given at an early stage, before organ failure has developed, that is at a time of tissue vulnerability or crisis,[22] using crisis in its original sense meaning a turning point (etymologically, a fulcrum). Late circulatory optimization after the establishment of organ failure or tissue damage is probably ineffective and may be harmful.[4] The same could be true in the perioperative setting.

How should circulatory optimization be judged? A number of techniques have been used in different studies, and found to be effective. Early studies relied on a pulmonary artery catheter. In modern practice, however, this is unlikely to be a first choice monitoring modality because of its perceived high complication rate.[23] Many less invasive alternatives are available, and have already demonstrated their value in circulatory optimization studies. These include such techniques as pulse power analysis,[11] pulse contour analysis, oesophageal Doppler monitoring[24 25] and others. These have been applied in a range of clinical settings including but not limited to abdominal surgery. Despite the demonstrable economic and clinical advantages of fluid optimization,[8 26] suitable monitoring devices have not become universally available in the operating theatre. Neither has their use become commonplace, perhaps because of a perception that they involve additional time, complexity or difficulty with an attendant risk to the patient.

In this edition of the *British Journal of Anaesthesia*, Solus-Biguenet and colleagues[27] have demonstrated that very simple, non-invasive or minimally invasive monitoring can be used to predict fluid responsiveness in a highly predictable and accurate way. In their elegant study, they have demonstrated that, where an arterial line is present, changes in respiratory pulse pressure variation can be used to predict responsiveness to a fluid challenge in patients undergoing major liver resection surgery—a patient group particularly susceptible to major fluid shifts. The accuracy with which they were able to predict this was high, with an area under the receiver operating characteristic (ROC) curve of 0.79. Not only this, a similar degree of predictive accuracy could be obtained by a completely non-invasive technique, using pulse pressure variability detected by the Finapres device. Using this device, the area under the ROC curve reached 0.81. The authors were able to derive their results using simple in-house software (Fig. 1).

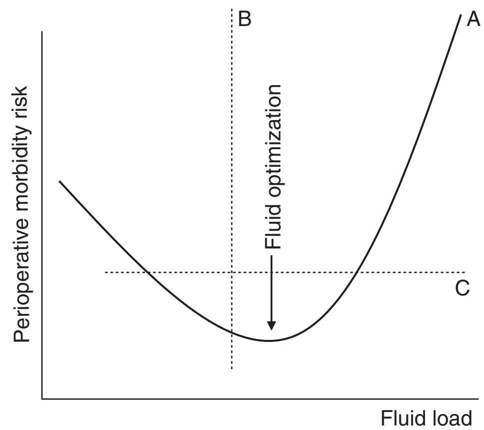

Fig 1 Curve A represents the hypothesized line of risk. Broken line B represents a division between patient groups in a 'wet *vs* dry' study. Broken line C represents a division between patient and groups in an 'optimized *vs* non-optimized' study

Techniques such as these can bring accurate prediction of fluid responsiveness, and hence, fluid optimization within the reach of all patients, without the need for either invasive monitoring or expensive additional equipment. It remains to be seen whether circulatory optimization using the simple techniques described by Solus-Biguenet and colleagues will translate into similar improvements in morbidity and hospital stay observed in previous studies using more invasive monitoring. If they do, we could be one step closer to protecting our patients from the Scylla and Charybdis of fluid therapy.

M. C. Bellamy

Professor of Critical Care Anaesthesia,
Intensive Care Unit,
St James's University Hospital,
Leeds LS9 7TF, UK
E-mail: m.c.bellamy@leeds.ac.uk

1 Shoemaker WC, Montgomery ES, Kaplan E, Elwyn DH. Physiologic patterns in surviving and nonsurviving shock patients. Use of sequential cardiorespiratory variables in defining criteria for therapeutic goals and early warning of death. *Arch Surg* 1973; **106**: 630–6

2 Matsuda H, Shoemaker WC. Survivor's and nonsurvivors' responses to dextran 40. Hemodynamic and oxygen transport changes in critically ill patients. *Arch Surg* 1975; **110**: 301–5

3 Shoemaker WC, Appel PL, Waxman K, Schwartz S, Chang P. Clinical trial of survivors' cardiorespiratory patterns as therapeutic goals in critically ill postoperative patients. *Crit Care Med* 1982; **10**: 398–403

4 Gattinoni L, Brazzi L, Pelosi P, *et al.* A trial of goal-oriented hemodynamic therapy in critically ill patients. SvO_2 Collaborative Group. *N Engl J Med* 1995; **333**: 1025–32

5 Shoemaker WC. Cardiorespiratory patterns of surviving and nonsurviving postoperative patients. *Surg Gynecol Obstet* 1972; **134**: 810–14

6 Shoemaker WC, Appel PL, Kram HB, Waxman K, Lee T. Prospective trial of supranormal values of survivors as therapeutic goals in high risk surgical patients. *Chest* 1988; **94**: 1176–85

7 Lobo SM, Salgado PF, Castillo VG, *et al*. Effects of maximizing oxygen delivery on morbidity and mortality in high-risk surgical patients. *Crit Care Med* 2000; **28**: 3396–404

8 Wakeling HG, McFall MR, Jenkins CS, *et al*. Intraoperative oesophageal Doppler guided fluid management shortens postoperative hospital stay after major bowel surgery. *Br J Anaesth* 2005; **95**: 634–42

9 Boyd O, Grounds RM, Bennett ED. A randomised clinical trial of the effect of deliberate perioperative increase of oxygen delivery on mortality in high risk surgical patients. *JAMA* 1993; **270**: 2699–707

10 Wilson J, Woods I, Fawcett J, *et al*. Reducing the risk of major elective surgery: randomised controlled trial of preoperative optimisation of oxygen delivery. *Br Med J* 1999; **318**: 1099–103

11 Pearse R, Dawson D, Fawcett J, Rhodes A, Grounds RM, Bennett ED. Early goal directed therapy after major surgery reduces complications and duration of hospital stay. A randomised, controlled trial. *Crit Care* 2005; **9**: R687–93

12 Takala J, Meier-Hellmann A, Eddleston J, Hulstaert P, Sramek V. Effect of dopexamine on outcome after major abdominal surgery: a prospective, randomized, controlled multicenter study. European Multicenter Study Group on Dopexamine in Major Abdominal Surgery. *Crit Care Med* 2000; **28**: 3417–23

13 Grocott MP, Mythen MG, Gan TJ. Perioperative fluid management and clinical outcomes in adults. *Anesth Analg* 2005; **100**: 1093–106

14 Moller AM, Pedersen T, Svendsen PE, Engquist A. Perioperative risk factors in elective pneumonectomy: the impact of excess fluid balance. *Eur J Anaesthesiol* 2002; **19**: 57–62

15 National Confidential Enquiry into Patient Outcome and Death. 1999 Report: Extremes of Age. http://www.ncepod.org.uk

16 Joshi GP. Intraoperative fluid restriction improves outcome after major elective gastrointestinal surgery. *Anesth Analg* 2005; **101**: 601–5

17 Kremer M, Ulrich A, Buchler MW, Uhl W. Fast-track surgery: the Heidelberg experience. *Recent Results Cancer Res* 2005; **165**: 14–20

18 Bergman S, Feldman LS, Carli F, *et al*. Intraoperative fluid management in laparoscopic live-donor nephrectomy: challenging the dogma. *Surg Endosc* 2004; **18**: 1625–30

19 Moug SJ, Smith D, Wilson IS, Leen E, Horgan PG. The renal sequelae of a novel triphasic approach to blood loss reduction during hepatic resection. *Eur J Surg Oncol* 2006; **32**: 435–8

20 Kita T, Mammoto T, Kishi Y. Fluid management and postoperative respiratory disturbances in patients with transthoracic esophagectomy for carcinoma. *J Clin Anesth* 2002; **14**: 252–6

21 Nisanevich V, Felsenstein I, Almogy G, Weissman C, Einav S, Matot I. Effect of intraoperative fluid management on outcome after intraabdominal surgery. *Anesthesiology* 2005; **103**: 25–32

22 Rivers E, Nguyen B, Havstad S, *et al*. Early goal-directed therapy in the treatment of severe sepsis and septic shock. *N Engl J Med* 2001; **345**: 1368–77

23 Pinsky MR, Vincent JL. Let us use the pulmonary artery catheter correctly and only when we need it. *Crit Care Med* 2005; **33**: 1119–22

24 Noblett SE, Snowden CP, Shenton BK, Horgan AF. Randomized clinical trial assessing the effect of Doppler-optimized fluid management on outcome after elective colo-rectal resection. *Br J Surg* 2006; **93**: 1069–76

25 Sinclair S, James S, Singer M. Intraoperative intravascular volume optimisation and length of hospital stay after repair of proximal femoral fracture: randomised controlled trial. *Br Med J* 1997; **315**: 909–12

26 Fenwick E, Wilson J, Sculpher M, Claxton K. Pre-operative optimisation employing dopexamine or adrenaline for patients undergoing major elective surgery: a cost-effectiveness analysis. *Intensive Care Med* 2002; **28**: 599–608

27 Solus-Biguenet H, Fleyfel M, Tavernier B, *et al*. Non-invasive prediction of fluid responsiveness during major hepatic surgery. *Br J Anaesth* 2006; **97**: 808–16