Haemodynamic and/or tonometric monitoring in cardiac surgery

Editor—If shock is defined as a systemic stage of oxygen supply-dependency, most sensitively established from the gastric intramucosal acidosis caused by the net accumulation of protons in anaerobiosis and most specifically excluded by the presence of a normal gastric intramucosal pH, then the following statements may be made about Bams, Mariani and Groeneveld’s recent ICU study of ‘haemodynamically compromised’ patients who had cardiac surgery.

Despite being ‘haemodynamically compromised’, two-thirds of the patients were not in shock on admission to the ICU or 12 h after admission. ‘Haemodynamic compromise’ was not a sensitive, specific or accurate indication of the presence or absence of shock and hence of the appropriateness, success or failure of the resuscitative efforts administered either during or after surgery. Nine (19%) patients who were not in shock on admission developed shock in the next 12 h and 10 (45%) who had been in shock were still in shock 12 h later. Therefore, the resuscitative efforts administered in the first 12 h in the ICU, which were directed by global haemodynamics, were often ineffective in shocked patients or caused shock in adequately resuscitated patients. Gastric intramucosal pH reflected appropriately the absence of any net change in mortality risk achieved by 12 h of haemodynamically directed management, as documented by admission and 24-h measurements in other studies of IC patients, but the differences did not achieve statistical significance. Measurements should have been made during surgery and more frequently to fully demonstrate their predictive value as we and others have reported.2 3

The specifics of these statements would change if the cut-off between normal and abnormal measurements, most appropriately determined from the statistical limits of normality, were different, but the point of the statements would not. Having a sensitive and specific measure of the presence or absence and degree of systemic oxygen supply-dependency has the potential to alter the logic of patient management and improve the precision, efficacy and cost-effectiveness of care by avoiding unnecessary, ineffective and harmful interventions and promoting the evolution of increasingly effective management practices.

Measurements of intramucosal PCO\textsubscript{2} or less conveniently but more accurately PCO\textsubscript{2} gap, provide very specific means of detecting acute regional perfusion failure but normal measurements do not exclude the presence of an intramucosal acidosis caused by impairment of oxygen uptake or ‘cytopathic hypoxia’. Haemodynamic measurements add value in so far as they help to define the severity of the shock present in patients with an intramucosal acidosis and the risk of developing myocardial as opposed to other organ dysfunction and failure, but can be very misleading if used without the support of tonometric measurements to direct management.

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Editor—We thank Dr Fiddian-Green for his comments. Although we do not understand all of the points, we would like to clarify some of the issues he raised.

Inclusion of patients was not based on ‘shock’ but on the presence of hypotension (<75 mm Hg) and the need for a relatively high dose of dopamine (i.e. >8 µg kg\textsuperscript{-1} min\textsuperscript{-1}) in the intensive care unit. The reason for these inclusion criteria is that shock is difficult to define. Dr Fiddian-Green erroneously equates a low pH with ‘shock’. A high or increasing PCO\textsubscript{2} gradient (e.g. a low or decreasing pH) measured in the stomach lumen during support of global haemodynamics was associated with a slight increase in mortality risk. A type II error can indeed cause this increase (which was not statistically significant).

Obviously, we may have missed a difference in PCO\textsubscript{2} gradient between outcome groups, which might have become apparent if more serial measurements had been performed in the disease course until death in the ICU or discharge. This does not invalidate our conclusion that, in a relatively large group of post-cardiac surgery patients with an increased risk of haemodynamic abnormalities and mortality, early global haemodynamic variables were of better predictive value for ICU mortality than regional (e.g. tonometric) variables, in spite of treatment being guided by global rather than tonometric variables. Tonometric variables did not have independent and additive predictive value, beyond the systemic haemodynamic abnormalities. Therefore, we cannot exclude the fact that hypotension, relatively unresponsive to treatment and predictive of a poor outcome, was not accompanied, in many of these patients, by global or gastric mucosal hypoperfusion, at least shortly after surgery. Conversely, our results do not support the suggestion of Dr Fiddian-Green that treatment guided by systemic haemodynamics worsens outcome. The proposition that early treatment of haemodynamically compromised patients after cardiac surgery should be guided by tonometric rather than by systemic haemodynamic variables and that this improves outcome was not supported by our study.

Furthermore, there is no consensus to treat patients who are haemodynamically compromised on the basis of tonometric variables rather than on routinely obtained, and normally accepted, haemodynamic variables. However, we cannot exclude a better outcome if treatment decisions had been based on both global and regional variables. This would be difficult to achieve in the absence of a uniformly accepted strategy to decrease an elevated gastric mucosal to blood PCO\textsubscript{2} gradient.

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