Correspondence

Etomidate for induction of the septic patient

Editor—We commend Eissa and colleagues' review article; however, we wish to draw attention to the selection of induction agent for septic patients. The ideal haemodynamic properties of etomidate use in this population are countered by lingering concerns about subsequent impaired adrenal steroidogenesis with its attendant consequences—a situation described as an ultimate Faustian bargain. Two recent systematic reviews have examined effects of single-dose etomidate in critically ill patients, and those with suspected sepsis. They both conclude that single-dose etomidate is associated with transient suppression of the adrenal axis. However, neither study reported a significant effect of etomidate on mortality. In fact, no prospective randomized trials to date have reported that etomidate has a significant adverse effect on mortality in patients with sepsis. We feel that while uncertainty remains, consideration should be given to using alternative induction agents, such as ketamine, in the patient with severe sepsis.

Conflict of interest
None declared.

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Reply from the authors

Editor—I appreciate the authors comments on our review article. The articles they cite support the case for etomidate use, on the grounds that there is little evidence of any detrimental effect other than transient adrenocortical suppression. Ketamine is also indicated in the induction of the haemodynamically compromised septic patient. However, I fear that formal comparison of these two agents in a randomized controlled trial with mortality or even major system dysfunction as an outcome measure would be extremely difficult to achieve.

Conflict of interest
None declared.

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Anaesthetic management of patients with severe sepsis

Editor—we read with interest the review article on the anaesthetic management of patients with severe sepsis. While informative, we were disappointed that no reference was made to the use of activated protein C (APC). This would be particularly relevant to the consideration of neuraxial blocks. The only real benefit we can see with epidural catheters in septic patients taken to critical care postop is to aid with weaning. However, epidurals on a background of sepsis is not only high risk but may preclude the use of APC which might be potentially life saving at a later stage. Although initial enthusiasm for APC has waned, the Surviving Sepsis Guidelines 2008 still recommends that adult patients with sepsis-induced organ dysfunction associated with a clinical assessment of high risk of death receive APC if there are no contraindications. We hope that the results of the two current randomized controlled trials underway (one funded by the French government due to be completed in March 2012 the PROWESS-SHOCK trial) will address the issue of APC and severe sepsis. Until this time, we believe that the placement of epidural catheters in patients with severe sepsis should be discussed with the on-call intensivist before insertion.

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Reply from the authors

Editor—I thank Dr Gibson and Dr Terblanche for their interest in our article. I disagree that weaning is the only benefit of epidurals in these patients, as attenuation of the surgical stress response, optimum analgesia, avoidance of high-dose opioids, reduction in thromboembolic phenomena, etc. are among the many potential benefits of an epidural when indicated after an individual risk–benefit analysis. Denying the septic patient an epidural in this scenario on the currently remote possibility of a subsequent theoretical indication to use activated protein C in these patients seems unjustified. I am more confident that the anaesthetist looking after the patient in theatre is better placed to decide on the merits of an epidural after an individual risk–benefit analysis than an intensive care-based colleague, possibly liaising by telephone, who may or may not be familiar with the patient.

Conflict of interest
None declared.

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Some old truths are still true ... succinylcholine in spinal cord injury

Editor—I was perturbed by the study by Yoo and colleagues on the cardiovascular responses to tracheal intubation in patients after spinal cord injury (SCI). While I agree with their conclusion that patients with cervical cord injuries are commonly hypotensive after induction, I have serious concerns about the anaesthetic techniques used in this study.

Since the early 1970s, there have been reports of hyperkalaemia and life-threatening arrhythmias in SCI after succinylcholine administration. The mechanisms responsible for hyperkalaemia are described in the 2006 review referenced by Yoo and colleagues in their paper. In fact, the review states that ‘Quadriplegics and paraplegics with persistent paralysis, therefore, could have the potential for succinylcholine hyperkalaemia throughout life’. Despite this, Yoo and colleagues administered succinylcholine to 214 SCI patients at various stages after SCI. The indication for rapid sequence induction seems particularly unclear: only 50 of the 214 were ‘acute’, defined as within 4 weeks of injury. In previous similar studies, the authors used vecuronium: there is no explanation for the change to succinylcholine in this study. In my own experience, rapid sequence induction is rarely indicated in this group, and I would question whether the use of this technique simply to fit a study protocol is ethical. At the very least, serial plasma potassium concentrations should have been provided by the authors.

Most anaesthetists regard avoidance of cardiovascular instability at induction as a laudable aim, and routinely administer opioids to help achieve this. This is particularly important in neuroanaesthesia. I am confused as to why Yoo and colleagues chose to use an opioid-free technique which not surprisingly produced the massive swings in heart rate and arterial pressure they report. I would not be proud of an anaesthetic chart showing that systolic arterial pressure and heart rate had increased by up to 60 mm Hg and 60 beats min⁻¹, respectively, when I intubated the patient. They have previously demonstrated that patients with tetraplegia have a blunted cardiovascular response to laryngoscopy and intubation, but conversely, they found that patients with paraplegia had at least a similar, and in some cases enhanced, response. Even in the (uninjured) control group, two in 20 patients had transient ventricular arrhythmias: the incidence in the injured patients reached up to 20% in some groups. Such cardiovascular instability is particularly undesirable in patients with chronic paraplegia who may be increased risk of cardiac disease: the authors acknowledge this in their discussion but made no attempt to avoid this very instability when actually looking after the patients!

Intubation was performed 60 s after the administration of succinylcholine using direct laryngoscopy and manual inline stabilization of the head. The text states that ‘Patients in whom intubation took more than 15 s were excluded from analysis’, that is, 105 s after giving thiopental. I wonder how many patients were excluded from the study for this reason and how they were eventually intubated. In the context of spinal instability attempting laryngoscopy if both muscle relaxation and anaesthesia are beginning to wear off is likely to put the spinal cord at risk of further injury. I believe that this risk is increased by the anaesthetic technique used in this study.

Overall I believe that the techniques described in this study put patients at increased risk of life-threatening complications. I am surprised that it received ethical approval in the first place and in particular that the BJA agreed to publish it.

Conflict of interest
None declared.

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