Commentary

Anaesthesia in septic patients: good preparation and making the right choice?

Colin F Royse[1,2]

[1]Department of Pharmacology, Level 8, Medical Building, University of Melbourne, Carlton, Victoria 3010, Australia
[2]Royal Melbourne Hospital, Melbourne 3050, Australia

Corresponding author: Colin F Royse, colin.royse@unimelb.edu.au

Published: 6 November 2009
This article is online at http://ccforum.com/content/13/6/1001

Critical Care 2009, **13:**1001 (doi:10.1186/cc8133)

See related research by Zausig *et al*., http://ccforum.com/content/13/5/R144

Abstract

Septic patients may require anaesthesia for surgery or to facilitate endotracheal intubation for respiratory failure. These patients frequently start with a deranged haemodynamic state, including vasodilation with hypotension, and cardiomyopathy, making induction of anaesthesia a potentially hazardous task. Anaesthetic agents are well known to decrease contractility and to cause vasodilation – in part from direct effect of the drugs, and in part due to the state of anaesthesia, that causes reduced sympathetic tone. Before induction, the physician should understand the haemodynamic state (especially using echocardiography), should restore cardiovascular reserve with inotropes and vasopressors, and should induce anaesthesia with the smallest dose of the safest drug. In the previous issue of *Critical Care*, Zausig and colleagues show that propofol may not be the safest choice of induction agent in septic patients.

Septic patients requiring induction of anaesthesia for surgery or mechanical ventilation frequently have severe haemodynamic derangement, which is likely to be made worse by anaesthesia. Anaesthetic drugs are well known to decrease contractility and to cause vasodilation both from direct effects on the heart and vasculature, and from the loss of sympathetic tone induced by the state of anaesthesia. In the previous issue of *Critical Care*, Zausig and colleagues examined the direct effects of intravenous anaesthetic agents on the myocardium, using an isolated septic rat heart preparation [1].

Prior to induction of anaesthesia, the physician should attend to three aspects of care. Firstly, the physician should understand the underlying haemodynamic state to determine how bad the myocardial performance is. This is easily achieved using focused transthoracic echocardiography [2-4], assessing the ventricular filling, function, and left atrial pressure state to determine whether the primary abnormality is vasodilation, systolic failure, systolic and diastolic failure, or right ventricle failure [5]. Secondly, the physician should attempt to restore cardiovascular reserve in these patients using vasopressor or inotropic drugs prior to induction. Thirdly, the physician should choose the safest induction agent and use as little of it as possible to achieve unconsciousness.

It is in this setting that we must question whether different anaesthetics have different cardiovascular profiles for different pathological conditions. Do septic patients behave differently from patients with dilated cardiomyopathy? Unfortunately, Zausig and colleagues did not compare the cardiovascular effects of the same anaesthetics in the sham operated hearts [1], so we cannot identify whether the magnitude of cardiovascular deterioration was the same for sepsis hearts versus normal hearts. In normal rabbits, propofol was shown to cause dose-dependent reduction in contractility *in vivo* (measured using pressure–volume loops) and had a slower recovery profile than that of sevoflurane or desflurane [6]. It may be that the same rules apply for any patient with a deranged haemodynamic state and that care and attention to the dose and supportive therapy may be more important than the choice of anaesthetic. It is also disappointing that the volatile anaesthetics were not investigated, as these have been shown to be protective at least for ischaemia–reperfusion pathology [7].

Nonetheless, Zausig and colleagues' paper raises the question of whether propofol use may be significantly worse in patients with sepsis, and highlights that one must be careful with both the induction dose as well as the maintenance infusion [1]. Propofol is one of the most commonly used drugs in the intensive care unit both for induction and for longer-term sedation. Further research is required for *in vivo* animal studies as well as human studies to better determine whether the effect size identified in the

present paper will translate to differences in the septic patient. Although ketamine is strongly recommended by Zausig and colleagues, the spectre of emergence delirium may negate the otherwise beneficial cardiovascular profile. Further research is also required to identify whether combinations of drugs will produce less cardiovascular depression, and whether longer-term infusions will produce differences in cardiovascular outcomes and patient recovery.

Competing interests

CFR has received research funding grants from Baxter Healthcare, who manufacture anaesthetic drugs, and has also received equipment support from Sonosite Australia for echocardiography studies. There are no other competing financial or nonfinancial interests.

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