Playing with fire: debate about propofol-induced hypotension

Reply from the authors

Editor—We thank Dr D. Green for his interest in our correspondence and his comments regarding propofol administration, especially in high-risk patients. Giving a vasopressor prophylactically before propofol administration to mitigate its side-effects of vasodilation and hypotension is debatable. A large number of references quoted by Dr Green involve investigations using phentylephrine in animal studies. The main problem is that one should not extrapolate these data to outcomes studies in humans. Moreover, there is no consensus among the anaesthesia community concerning routine use of this technique.

With cardiac depression and reduced myocardial contractility, cardiac output is decreased. Our study in 1985, was the first haemodynamic study of propofol in the USA. This study highlighted propofol side-effects on anaesthesia induction of ASA class II and III patients. Because of these findings, ASA class IV and V patients are at greater risk from haemodynamic collapse. Venous return to the heart is also reduced, further worsening the hypotension and decreasing the cardiac index.

Results from the study are as follows: MAP (mean arterial pressure) decreased by –23 (12)% at 2–3 min (some recovery at 5 min), CO (cardiac output) decreased by –18 (13)% at 2–3 min (recover at 5 min), LCSWI (left ventricular cardiac stroke work index) decreased by –35 (16)% at 2–3 min mark and LSWI (left ventricular stroke work index) decreased by –35 (18)% at the 2–3 min mark (a function of lower CI and lower MAP).

Why administer phentylephrine as advocated by Goodchild and Serrao, Bentley and colleagues, Bidd and colleagues, which may cause hypertension in many patients, just to offset presumptive propofol hypotension? Furthermore, the degree of hypotension is unknown and an overshoot causing hypertension places the patient at risk for myocardial ischaemia and stroke. Playing such ‘games’ is potentially dangerous without an arterial line before induction of anaesthesia. Without an arterial line to guide induction of high-risk patients (ASA IV or V), utilizing the technique of first phentylephrine followed by propofol is ‘playing with fire’.

Phentylephrine is an α-adrenergic receptor agonist. This peripheral vasoconstriction (increase SVR and PVR) and reflex bradycardia with accompanying propofol myocardial depression can result in increased cardiac ‘back pressure’—worsening pulmonary oedema in congestive heart failure patients.

In conclusion, we would like to thank Dr Green for his comments and thoughtful debate on our correspondence relative to the use of propofol for induction of anaesthesia. Debate is always good in the anaesthesia community. The next big debate should focus on the use of an i.v. vasopressor before propofol and the pros and cons of this technique as proposed by Goodchild and Serrao in their letter. The bottom line is ‘patient safety’ and currently, there are no outcome studies to support prophylactic or concurrent vasopressor use with propofol.

Declaration of interest

None declared.

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Airway management strategy for odontogenous abscess

Editor—In their study of patients with odontogenous abscesses and reduced mouth opening, Schumann and colleagues report successful tracheal intubation with the Glidescope device (Verathon Medical Europe, Ijsselstein, The Netherlands) for all 50 subjects randomized to this form of laryngoscopy; additionally, the Glidescope was used successfully for rescue in the group of patients randomized to Macintosh laryngoscopy but had failed. Readers may see this as good news for the Glidescope in managing difficult airways complicating dental abscess; some may extend this as good news for videolaryngoscopy in general. While this may be true, we have concerns that the authors’ overall approach may not be generally applicable for patients with this dangerous condition.

First, this study was performed by practitioners with experience in anaesthesia for head and neck surgery and with acquired skill in videolaryngoscopy. This is therefore a competent group and so their results may not be directly transferable to others, a form of ‘context sensitive airway management’. We feel that this factor should be emphasized more; experts do things that others cannot.

Secondly, the authors acknowledge that this is a high-risk group of patients to manage safely. The authors recognize this with description of a back-up plan, namely awakening the patient with subsequent awake fibreoptic intubation. The notion of planning for failure is a consistent theme throughout the NAP4 report. Planning is often, however, limited to the so-called ‘Plan A, Plan B’ approach. If these two plans share modes of failure which can spoil each technique, the notion of binary planning for airway management may not be sufficient, since factors leading to failure of Plan A may prevent effective execution of Plan B. For example, if Plan A (videolaryngoscopy) fails due to instrumental bleeding (a real possibility in the context of airway infection with swollen, hyperaemic tissues), Plan B (fibreoptic management) is also compromised. The two plans are too tightly linked or coupled to the same failure mode. So, for the authors, failure of both of their plans is possible as a consequence of both plans sharing a common failure mode, namely bleeding.

Thirdly, to the element of risk, we can add the element of uncertainty. In their study of airway management planning in dental abscess, Darshane and colleagues report that ‘problems did occur... and ‘these always came as a surprise requiring a total re-think’. This is a possibility for Schumann and colleagues. They may be confronted with a deteriorating, perhaps dangerous airway requiring immediate, reactive responses. Facemask ventilation may not be effective (efficacy of mask ventilation in the authors’ study was not reported), use of a supraglottic device may be impossible with high-grade trismus, and emergency cricothyroidotomy is associated with low success. Succinylcholine, used by the authors for airway control, may not wear off before hypoxia develops, especially in patients with high oxygen consumption (young patients with infection and fever). Darshane and colleagues call this ‘on the hoof’ airway management, stating it ‘must be considered a recipe for disaster’.

We suggest that safety is increased if the second (and even better, a third) plan has a method of execution whose risks are distinct from the other selected. For Schumann and colleagues, this could be insertion of a precautionary cricothyroid cannula, a proactive method of particular utility in patients with high-risk airways. This would plausibly increase safety not only for the intubation sequence, but also for tracheal extubation, a period of risk which is often underestimated. Postoperative bleeding is a concern for all the study patients, particularly for those whose mouth opening remained limited after anaesthesia. Indeed, airway bleeding is recognized as a major source of morbidity and mortality in the NAP4 report.

In general, odontogenous (dental) abscess with fascial space involvement are difficult to manage and those affecting the floor of the mouth are even more difficult and may be dangerous. Trismus is an extra and major complication. This may not resolve with anaesthesia and is associated with symptom duration, as the authors have usefully shown. We conclude that videolaryngoscopy may be safe and effective in this challenging group of patients, but only in the context of a robust, reliable airway management strategy. This would best be devised using proactive and reactive components whose effective implementation, if needed, use methods that are not compromised by sharing the same failure mode.

Declaration of interest

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