Declaration of interest

None declared.

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doi:10.1093/bja/aeu423

Cardiac output decrease and propofol: what is the mechanism?

Editor—I read with interest the correspondence regarding the haemodynamics of induction agents and bispectral (BIS, Covidien, USA) index-guided induction of general anaesthesia.1 2 As far as I am aware, the paper by Moller Petrun and Kamenik3 is the first to look at this period of change in haemodynamics using BIS-guided anaesthesia and a minimally invasive cardiac output monitoring device (LiDCOrapid, LiDCO Ltd, UK).

It is interesting to note that the paper referred to by Kakazu and Lippmann relates only to an abstract published nearly 30 yr ago.4 However, they are quite correct in their proposition that the decrease in cardiac output (CO) may be quite substantial in high-risk patients after induction of general anaesthesia. Indeed, after the abstract published in 1986, Lippmann and colleagues5–9 have been very keen to highlight the deleterious cardiac effects of propofol in a number of articles (mainly correspondence) to this and other journals.

Most anaesthetists continue to regard the decrease in mean arterial pressure (MAP) on induction as due either to cardiac depression, as Kakazu and Lippmann propose, or due to decrease in systemic vascular resistance. However, we believe that the decrease in CO has little to do with an effect of reduction in cardiac contractility as they suggest, but rather on an effect on preferentially reducing venous rather than arterial tone.

Elegant studies in the early 1960s demonstrated that venous tone was significantly raised in precisely those patients who would be most affected by venodilation and included patients with cardiac failure and the anaemic.10 Although alluded to in some publications,11 little attention has been directed to the known effects of propofol on venous smooth muscle. This pioneering work was carried out nearly 30 yr ago by Colin Goodchild and his colleagues at Leeds.11 13 They clearly demonstrated that veno relaxation and an increase in venous capacitance resulted in diminished venous return and stroke volume leading to a decrease in CO and MAP. Venous relaxation due to propofol can very easily be offset by the simple expedient of administering a low-dose phenylephrine infusion immediately before the administration of propofol. We have shown, albeit in abstract form only, that this simple manoeuvre will markedly reduce the decrease in CO and MAP during induction with propofol.14 Indeed, it may well be that most of the effects on reduction in stroke volume, CO, and MAP and increase in stroke volume variation seen post-induction may be due to venous relaxation. Although the effects of venous capacitance can also be counteracted by the use of fluids,13 it is surely not the correct strategy in elective patients who are not fluid depleted and have not been subjected to many hours of fluid restriction.15

Moller Petrun and Kamenik are to be congratulated for pointing out the decrease in CO drives the decrease in MAP post-induction and is evident with both propofol and etomidate. Further work is being done in this area to see whether our supposition that phenylephrine will markedly reduce this decrease by its administration prophylactically during surgery in high-risk patients is correct. It also emphasizes the importance of measuring CO preinduction and getting a baseline reading. As Professor Jean Louis Vincent and Dr David Fagnoul have elegantly pointed out ‘The main reason why reliable cardiac output monitoring can be useful during surgery is to be able to establish a baseline for high-risk patients in whom complications, such as hypoxemia, tachycardia or oliguria, arise after the immediate postoperative period, and therapeutic interventions become more complex. Some would argue that there is still time to introduce a cardiac output device at this point, but most would agree that it is preferable to be able to make a trend evaluation when such a problem occurs’.16

Declaration of interest

D.W.G. has received subsistence and travelling expenses from LiDCO Ltd and also honoraria for speaking at meetings from Covidien.

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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.1 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 phenylephrine 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 19852 3 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), LCSI (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 phenylephrine as advocated by Goodchild and Serrao,4 Bentley and colleagues,5 Bidd and colleagues,6 which may cause hypertension in many patients, just to offset presumptive propofol hypotension? Furthermore, the degree of hypotension is unknown and an overshoot causing hypotension 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 phenylephrine followed by propofol is ‘playing with fire’.

Phenylephrine 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 Serrao6 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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