Correspondence

"Preservatives" in propofol

To the Editor:

The addition of 0.005% disodium edetate to the formulation of propofol was mandated by the United States Food and Drug Administration (FDA) in 1997. Disodium edetate, in this concentration, can be expected to retard microbial growth, but not to confer complete antimicrobial "preservation".¹ This change has not been required by the Health Protection Branch (HPB) in Canada.

Propofol preparations support microbial growth² and outbreaks of postoperative infection have been linked to extrinsic contamination of the agent.^{3,4} There are no reports from Canada.

The importance of using strict aseptic techniques when drawing up propofol is emphasized widely. In addition, the interval between drug withdrawal and administration should be as short as possible, < 6 hr for single dose injections and 12 hr for continuous infusions (Propofol Product Monographs). The HPB is aware of the FDA-mandated change, but has decided not to follow their example.⁵ Reasons include: 1) concern that the presence of the "preservative" may encourage complacency and an inappropriate relaxing of the required strict aseptic handling practices; 2) knowledge that correct use of the product, as labelled (i.e. following strict aseptic technique) will prevent complications related to extrinsic contamination; 3) lack of evidence confirming a benefit from the US change in formulation; 4) absence of Canadian reports of infections due to propofol contamination; and 5) lack of evidence confirming the disodium edetate addition does not create an independent hazard.⁵

The merits of including a preservative in propofol formulations remain to be determined. Intuition suggests that a "partially preserved" product should offer a benefit over a product without preservatives which has infectious risks linked to extrinsic contamination, this has not been demonstrated and the absence of risk from the added preservative has not been confirmed. HPB deserves credit for its careful approach on this issue.

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End-tidal CO₂ after CO₂ embolism

To the Editor:

Rudston-Brown and colleagues described their interesting findings concerning the haemodynamic state and gas exchange after gas embolism with CO₂ vs He in pigs.¹ The authors found more severe deleterious effects with He than with CO₂ embolism and a decrease in P_{ET}CO₂ was associated with both conditions. However, in disagreement with their results, previous experimental studies^{2,3} demonstrated an initial increase in P_{ET}CO₂ after CO₂ embolism, mostly after the injection of rapid bolus of small volumes of CO₂. This may occur as a result of the excretion of CO₂ by the lungs, after its rapid absorption into the blood. In addition, some anecdotal cases^{4,5} confirm the observations made in the mentioned experimental settings. It is possible that the adjusting of the mechanical ventilation, as described in the study by Rudston-Brown *et al.* may have increased CO₂ excretion, leading to lower P_{ET}CO₂ levels. Since CO₂ embolism presents some particular characteristics, clinical anaesthetists should be aware of this transient increase in P_{ET}CO₂ levels after CO₂ embolism in order to make an early diagnosis.

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