Role of hypotension in the cerebral haemodynamic effects of sufentanil and fentanyl

Sir,—In an abstract by Jamali and colleagues from the European Society of Anaesthesiologists Annual Congress [1], the authors concluded that sufentanil 0.8 μg kg⁻¹ caused a significantly greater reduction in cerebral perfusion pressure than fentanyl 4.5 μg kg⁻¹. The comparative potency ratio of sufentanil to fentanyl in this study was therefore 5.6:1.

However, sufentanil is 11.5–12 times as potent as fentanyl based on both the IC₅₀ value (plasma concentration necessary to produce 50% of the maximal EEG effect) [2] and studies of the MAC reduction of isoflurane by sufentanil [3] and fentanyl [4]. In order to compare the effects of these two opioids on any clinical variable, including their effects on cerebral perfusion pressure, it is important to select dosing schemes which reflect their relative potencies. In this study, a relative overdose of sufentanil was given. A fentanyl dose of 9.2-9.6 μg kg⁻¹ should have been given compared with a dose of sufentanil 0.8 μg kg⁻¹ to give a potency ratio of 11.5-12:1, thereby allowing a fair comparison between these opioids to be made.

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Carcinoid syndrome and anaesthesia

Sir,—I read with interest the recent article by Veall and colleagues [1] on the anaesthetic management of patients undergoing laparotomy for carcinoid syndrome. The review article was very good and gave a very concise account of how to manage this disease.

The authors made reference to an article that I had published in Anesthesia and Analgesia in 1973 [2] in which I stated that "the drug aprotinin is one of the most effective antagonists of the bradykinin–kallikrein system. It is a potent inhibitor of kallikrein–kinin and trypsin systems, specifically blocking the activity of the protease kallikrein and preventing further proteolytic action on alpha, globulin, resulting in elaboration of bradykinin. However, as aprotinin is not yet available for clinical use, some other agent should be used." The authors, in referencing my paper, also stated that its role in this disease entity was "questioned" by me. This gives the misleading impression that I did not wish to use the drug. May I point out that in 1973 aprotinin was an experimental drug in the USA, as stated in the paper, and therefore could not be used clinically. If it had been approved for use by the regulatory government agencies at that time I may have indeed used the drug on my patient.

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