Systolic arterial pressure as a monitor of depth of anaesthesia

Sir,—When Robb and colleagues [1] wrote: "The use of clinical signs (SAP in this case) to guide anaesthetic dosage... was the approach adopted by Suppan in his pioneering studies", they gave the impression that Suppan [2] was the first anaesthetist to use arterial pressure as an indicator of adequacy of anaesthesia. However, when halothane was first introduced, several authors recommended using arterial pressure as an index of depth of anaesthesia. I was taught during my residency that, during anaesthesia, the SAP should always be maintained within 20% of baseline.

Maintaining the SAP within these limits throughout anaesthesia and injecting intermittent volumes of liquid enfurane in the expiratory limb of a closed system [3], we have observed that the accumulated volume of enfurane was, on average, only about 50% of predicted, whereas the use of halothane and isoflurane was found to agree with predicted volumes [4-8]. The reasons for these various deviations from Lowe's theory [3] could not be detected in Brazil because there was no means of measuring end-tidal concentrations.

In 1986 at the University of Wales College of Medicine, an exploratory study confirmed the Brazilian findings [9], and during the IVth International Symposium of the Closed and Low Flow Anesthesia Society, Couto da Silva, Vickers and Mapleson showed a nomogram in which SAP correlated with the depth of anaesthesia. Therefore, the finding of Robb and colleagues [1] confirms that "SAP may be major component of the clinical assessment of the anaesthetic state" [1] observed previously by other authors.

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Sir,—Thank you for the opportunity to comment on the interesting letter from Professor J. M. Couto da Silva.

We feel that it is particularly important to see the comments in our paper in the context of closed loop automatic control of the anaesthetic process, and our comments about Suppan's work were made in this context. Of course we do recognize that others have used arterial pressure as a method of assessing adequacy of anaesthesia, but not in closed loop control.

Furthermore, the words in our paper were chosen carefully to reinforce the fact that SAP is only one part of the range of clinical signs observed regularly by anaesthetists, and that it should be seen in context. We agree that SAP carries considerable information about adequacy of anaesthesia within the confines of our work where, for example, there were no beta-blockers, antihypertensive drugs or major blood loss.

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studied by Hynynen and colleagues. Using their elegant experimental method of the isolated vein segment, Klement and Arndt could have studied the pain-inducing potential of various i.v. anaesthetic agents without the risk of these reaching the general circulation, thereby generating more useful and relevant information for the daily practice of anaesthesia.

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Sir,—It is pleasing that our publication has provoked some discussion, particularly as we can cope easily with any criticism:

Our title would be misleading only if one were to ignore the pronoun “some”, which we chose deliberately to account for other alternatives. In fact, we were already aware of the exceptional properties of propofol and have demonstrated its analgesic action recently [1].

That most of the drugs mentioned in the paper of Bretschneider [2] have osmolalities less than 1 osmol kg⁻¹ is a spurious argument against osmolality as “principal pain stimulus” because most of these drugs do not evoke pain. In fact, some formulations of those which do evoke pain have osmolalities greater than 6 osmol kg⁻¹—the greatest used in our injection experiments. It is mainly a matter of opinion, if our observations have “little (if any) relevance to clinical practice”. In essence, we studied the basic pain-evoking stimuli of injectates, not of drugs. With regard to the clinical implications of our results, we refer to the last paragraph of our discussion.

The reproach of having misquoted Hynynen and colleagues [3] is partially justified. The quotation in the introduction refers to barbiturates in general and to methohexitone in particular but, regretfully, we omitted to add Kawar and Dundee (ref. [4] in our paper) with regard to pain on injections of thiopentone. Admittedly, we misquoted Hynynen and colleagues in the discussion.

We are flattered by the phrase “elegant experimental method of the isolated vein segment”, and we agree that we could have studied the pain-inducing potential of clinically used i.v. anaesthetic agents. However, with the exception of propofol [1], the properties of the pharmaceutical formulations appeared to be more relevant as pain-evoking stimuli than the agents themselves. From a theoretical point of view, we tested and proved the hypothesis that unphysiological osmolality and pH per se may be responsible for pain on injection. Does it not still hold that “theory is the most practical thing conceivable” [4], stated also with regard to medical sciences just 100 years ago?

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