PENTAZOCINE AND TREMOR FOLLOWING HALOTHANE ANAESTHESIA

Sir,—Muscular spasticity and tremor occurs during recovery from general anaesthesia, especially following halothane (Dawkins, 1961), and may be associated with hypoxaemia (Jones and McLaren, 1965). This condition may be prevented or controlled by methylphenidate (Brichard and Johnstone, 1970). However, this drug is an unreliable analgesic, may provoke adrenergic overactivity (Brichard and Johnstone, 1970), causesamphetamine-like drug dependence (Willis, 1974) and is usually included in the D.D.A. drug schedule.

To test the hypothesis that pentazocine might be an effective substitute for methylphenidate in controlling muscular spasticity and tremor following halothane we studied 100 consecutive patients who developed this condition. Fifty-four patients were given pentazocine 30 mg i.v., and 46 received methylphenidate 20 mg i.v. The patients were allocated randomly to the groups which were comparable in terms of age of the patient and type of operation. The same anaesthetic technique was used although this was not standardized and the duration of anaesthesia was not recorded. The patients were observed in the recovery room for 30 min by a trained nurse who did not know which drug had been given. The nurse was asked to record only the presence of tremor, which was regarded as a more objective assessment to make than spasticity.

In all except one of the patients given methylphenidate tremors stopped within a few minutes of the drug being administered and they did not recur. In the one exception, tremor ceased immediately after administration of methylphenidate but recurred slightly. With pentazocine the tremors stopped quickly and did not recur in 46 of the patients, but recurred slightly in seven and were not affected in three.

Pentazocine does not control tremor following halothane anaesthesia as well as methylphenidate, but, where hypoxia is not a serious problem and pain is present, pentazocine 30 mg i.v. reduces both rigidity and tremor and provides useful analgesia with less book-keeping for the nursing staff.

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REFERENCES

TRACHEAL CYLINDROMA: ANAESTHETIC MANAGEMENT

Sir,—After the publication of our article "Tracheal cylindroma: anaesthetic management" (Lippmann and Mok, 1977), a further literature survey revealed an article by Geffin, Bland and Grillo (1969).

Your readers should be made aware also that in the forthcoming Proceedings of the 14th Scandinavian Society of Anaesthesiologist's meeting (1977) Dr A. Baraka's presentation on "Anaesthetic problems during the tragic civil war in Lebanon" deals with a case report of a transsected trachea and its management which is similar to the description in both our article and that of Geffin, Bland and Grillo (1969).

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REFERENCES

TOXICITY OF LOCAL ANAESTHETICS

Sir,—Recent advances in the field of local anaesthesia have stirred lively interest in the tissue distribution and metabolism of these drugs. Unfortunately, although much valuable information has been gained from these studies, often much of this work has been done in a clinically irrelevant setting or conclusions have been reached that are at the moment still speculative, or both.

The recent study by Malagodi, Munson and Embro (1977) on the effect of infusion rates on the acute toxicity of etidocaine and bupivacaine in Rhesus monkeys concluded that etidocaine was less toxic than bupivacaine.

Accidental intravascular injection of a local anaesthetic solution is the most obvious possibility for acute systemic toxicity. Therefore, it is desirable that acute toxicity studies duplicate infusion rates used clinically in extradural block or peripheral nerve block. In the clinical studies where extradural infusion rates are indicated (Bromage, O'Beirn and Dunford, 1974; Tucker and Mather, 1975) they are in the range 150-450 mg min~1. The low end of this spectrum would correspond to the fastest infusion rate in Malagodi's study. Contrary to the overall conclusion of the authors, at these injection rates, etidocaine appeared more toxic than bupivacaine, with the seizure dose for etidocaine being 4.76 mg kg~1 as opposed to 5.33 mg kg~1 for bupivacaine. Clinical studies (Scott, Jebson and Boyes, 1973; Scott, 1975) have shown also that the etidocaine toxicity threshold decreases as the infusion rate increases.

The toxicity from systemic absorption of the local anaesthetic also deserves brief consideration. A number of studies have disregarded the behaviour at fast infusion rates, and assumed that the characteristics of the slow infusion rates simulate the systemic absorption from the injection site and that serum concentrations correlate with the appearance of toxicity.

A comparison of the arterial and venous drug concentrations after giving etidocaine and bupivacaine in equal dose (milligram for milligram) have suggested that the resulting plasma concentrations tend to be greater for bupivacaine. However, these differences seem to be less at the higher doses (Bridgenbaugh et al., 1974a; Moore et al., 1976). These data are again of questionable clinical relevance since they assume that the two local anaesthetics are equipotent. There are a number of studies indicating that this is not so. The potency of bupivacaine has been estimated to be 1.5 to 2 times that of etidocaine (Bromage,
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O’Beirn and Dunford, 1974; Morgan and Russell, 1975; Renck and Edstrom, 1975. Even at the higher etidocaine doses, visceral analgesia was less effective, behaviour was unpredictable when the drug was used for peripheral nerve block, and the intensity of motor block was a problem (Bromage, O’Beirn and Dunford, 1974) or a potential problem (Renck and Edstrom, 1975). Studies that attempted to compare the two drugs at the same milligram doses (Moore et al., 1974, 1975) indicated a greater analgesic efficacy of bupivacaine. In addition, the intense motor blockade produced by etidocaine disturbingly outlasted its analgesic action (Moore et al., 1975). Consequently, equipotent, not equivalent, doses should be compared if a clinically meaningful conclusion is to be drawn.

The practising physician is interested in the performance and safety of a drug in the actual clinical setting. Taking individual observations out of context of this clinical reality is to run the danger of coming to medically misleading conclusions.

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——— (1976). Arterial and venous plasma levels of bupivacaine following epidural and intercostal nerve blocks. Anesthesiology, 45, 39.


Sir,—Thank you for the opportunity to reply to Drs Young and Luna.

From a careful reading of this letter, it appears that the arguments raised by Drs Young and Luna are based on the mistaken assumption that we stated that etidocaine was less toxic than bupivacaine. However, nowhere in our paper did we make such a statement. As shown clearly in table I of our paper, the only statistically significant differences among any of the measurements made were between seizure doses for etidocaine at 0.5 and 2.0 mg kg\(^{-1}\) min\(^{-1}\) and seizure thresholds for etidocaine at 0.5 and 1.0 mg kg\(^{-1}\) min\(^{-1}\).

In our discussion we stated “Thus, in clinical practice, where relatively large doses of local anaesthetic drugs are administered and are absorbed slowly—for example, during regional nerve block—etidocaine would be tolerated better than bupivacaine.” This statement was based primarily on the findings of others, as stated in the preceding two sentences of the paragraph, and secondarily, on results obtained with the slowest infusion rate of the two agents. It should be noted clearly that we showed only a tendency for a better (and not a statistically significant) tolerance to etidocaine than bupivacaine.

The third paragraph of the letter from Drs Young and Luna focuses entirely on erroneous conclusions drawn from statistically insignificant differences between seizure doses measured during the rapid (2.0 mg kg\(^{-1}\) min\(^{-1}\)) infusion rate experiments (see table I). In the discussion section we concluded: “However, following rapid administration . . . as would occur following inadvertent intravascular injection, differences in rates of redistribution and hepatic extraction would be minimized, tending to equalize the toxicity of the two agents.” This is the conclusion, and the only conclusion, which may be drawn from the data presented. Drs Young and Luna have misread this statement also.

Our study, which correctly appeared in the experimental section of British Journal of Anaesthesia, was designed primarily to evaluate the effect of infusion rate on the toxicity of both etidocaine and bupivacaine in Rhesus monkeys. The small number of non-human species studied must be considered by the readers in the appropriate context. We hope that most careful readers would be perspicacious enough not to draw conclusions which the authors have been most cautious to avoid making, on the basis of lack of statistically significant data; and to view this animal study within the appropriate and intended context.

The only part of the letter from Drs Young and Luna which pertains to our study is the third paragraph. The remainder of the letter deals with areas remote from the object of our paper. The sole exception may be their comment, “Taking individual observations out of context of this clinical reality is to run the danger of coming to medically misleading conclusions.” We agree with this statement and respectfully suggest that Drs Young and Luna reread our paper with this thought in mind.

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