INTERMITTENT VS CONTINUOUS EXTRADURAL INFUSION OF BUPIVACAINE DURING LABOUR

Sir,—The absolute significance of the findings of Eddleston and colleagues [1] would have been much clearer if they had compared the two extradural control groups which did not receive extradural bupivacaine. Although this control group would have been self-selected and not subject to randomization, the group could have been matched as closely as possible to the two study groups.

In addition, although the incidence of oxytocin use was recorded in this study, an analysis of the association between oxytocin use and the fetal heart rate abnormalities would have provided additional information. This association is recognized widely, and ascribing heart rate abnormalities to different extradural techniques irrespective of the use of oxytocin may not be valid.

Whilst it is tempting to attribute fetal heart rate abnormalities to uptake of bupivacaine by the fetal myocardium, the evidence for this is far from conclusive. Although Abboud and colleagues [2] also observed fetal heart rate abnormalities after extradural bupivacaine, other factors may have been contributory. In the large majority of cases, abnormalities are associated with aortocaval compression which is not always diagnosed or recognized [3]. The umbilical venous:maternal venous concentration ratio of bupivacaine in the umbilical vein at one minute in some of the women in this study may be an index of fetal myocardial toxicity. Moreover, although the evidence for fetal myocardial toxicity from bupivacaine is strong, this does not indicate that the fetal myocardium is much more sensitive than adult myocardium.

The predominant reason for the low fetal:maternal ratio of bupivacaine is that bupivacaine causes fetal myocardial toxicity, and the resultant difference in the proportion of bupivacaine bound to alpha-1-acid glycoprotein across the placenta, and the resulting relative difference in the proportion of bupivacaine bound to glycoprotein [6]. Moreover, fetal acidosis, which may be associated with the use of oxytocin, promotes placental transfer of bupivacaine by ion trapping of the basic drug [7].

Finally, the conclusion of Eddleston and colleagues [1] that there is no advantage in either extradural intermittent bolus or infusion techniques must be re-examined. If it is hypothesized that bupivacaine causes fetal myocardial toxicity in these small concentrations, then it suggests that the fetal myocardium is much more sensitive than adult myocardium.

Sir,—Thank you for the opportunity of replying to Dr Laishley’s detailed letter. Our study was an attempt to examine, in particular, the effect of continuous extradural infusion of bupivacaine compared with intermittent injections of 0.25% bupivacaine. Exhaustive studies comparing intermittent and continuous infusion extradural analgesia with bupivacaine failed to demonstrate any maternal advantage associated with either regimen [1, 2]. To date, no attention has been paid to the effect of the two regimens on the fetus, in particular fetal heart rate pattern.

In our study, we documented 71 deceleration episodes in the intermittent group and 69 in the infusion group. We found no difference between groups in the incidence of deceleration occurring within 30 min of an extradural top-up, but we did detect a discrepancy in its severity and duration. In the intermittent group, the majority of decelerations (73.8%) were recorded as transient, whereas in the infusion group 61.1% of episodes lasted in excess of 10 min.

Indeed, there are several other potential causes for fetal heart rate decelerations, including aortocaval compression producing hypotension, the use of oxytocics and fetal acidosis, all of which are referred to in Dr Laishley’s letter. I can restate that none of the deceleration episodes referred to our paper occurred in the presence of maternal aortocaval compression. Obviously, this does not exclude the presence of concealed aortocaval compression. In addition, there was no difference in the mean dose of oxytocic required by each treatment group in the acid-base status of the two groups at delivery.

Finally, we accept Dr Laishley’s comments concerning a control group. Such a group almost universally would have received another form of analgesia, most probably parenteral pethidine.

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DOES ETOMIDATE CAUSE HAEMOLYSIS?

Sir,—I read with interest the article “Does etomidate cause haemolysis?” by Nebauer and colleagues [1], as it reminded me of an unreported finding after etomidate use in domestic cats. We conducted research several years ago at the Veterinary Teaching Hospital at the University of Illinois to determine the anaesthetic effects and pharmacokinetics of etomidate in domestic cats [2, 3]. We used Amidate i.v. (Abbott Laboratories) in a dose of 3 mg kg⁻¹.