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AWARENESS DURING CAESAREAN SECTION

Sir,—Many of us are deeply concerned about the incidence of awareness during Caesarean section. The problem is almost certainly greater than would appear, since not all cases are reported.

When one of these incidents occurs, the anaesthetist concerned usually pleads that he followed routine practice, that is induction with thiopentone, neuromuscular blockade, intubation of the trachea followed by ventilation with a 50% nitrous oxide in oxygen mixture plus 0.5% halothane. In the absence of premedication with a narcotic analgesic, inhalation of such a mixture whilst breathing spontaneously will not produce or maintain unconsciousness in the majority of people. Certainly, hyperventilation will produce a degree of analgesia and impairment of consciousness and this no doubt accounts for some of the successes. However, present day preoccupation with the possible effects of PaCO₂ on the fetus has led to the avoidance of hyperventilation, often with careful monitoring of end-tidal PaCO₂ to maintain it at normal values.

Surely, the answer is to provide the patient with a gas mixture guaranteed to maintain unconsciousness. For more than 30 years I have used 60% nitrous oxide plus 1% trichloroethylene in oxygen. Trichloroethylene is a good analgesic, whereas halothane is not, and although it has a higher blood solubility than halothane this is offset by its remarkably high potency: oil/gas solubility 960; MAC 0.17%. During all the time I have used this mixture, I have never had a problem with awareness and the babies' Apgar scores seem to compare well with those obtained using other techniques. Certainly I have never lost a baby.

However, I will go further. Even if it could be shown that my technique had a marginal adverse effect on the fetus, I would find it acceptable if the alternative was possible awareness in the mother.

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CARDIAC ARREST IMMEDIATELY AFTER VECURONIUM

Sir,—Recent reports have brought to our attention a possible association between the new cardiovascularly "clean" myoneural blockers and serious bradycardia/arrhythmias (Kirkwood and Duckworth, 1983; Milligan and Beers, 1985). Common in the majority of these reports have been: (a) the omission of an anticholinergic agent either as premedication or at induction, (b) a relatively slow heart rate before the arrhythmia and (c) a delay of up to 20 min between the administration of the myoneural blocker and the onset of the arrhythmia. Any actual association has been called into question (Maestrone and Pradella, 1985).

Recently a 79-year-old female (45 kg) presented with an actively bleeding gastric ulcer, having been admitted as an emergency 7 days previously with a haematemesis. Following initial resuscitation and a negative endoscopy, the patient had