CORRESPONDENCE

SPINAL ANAESTHESIA AND CARDIAC DISEASE

Sir,—May I take issue with Dr Mostafa as to the suitability of spinal anaesthesia for the management of Caesarean section in the patient with severe cardiac disease (Mostafa, 1984).

Extradural blockade has rightly been advocated in the management of patients with cardiac disease for labour (Ostheimer and Alper, 1975) and Caesarean section (McMurray and Kenny, 1982; Linter and Clarke, 1984). This is because, by careful incremental dosage, the block can be instituted with minimal cardiovascular disturbance.

This is precisely what you cannot do with spinal anaesthesia. In a patient with limited cardiac output and pulmonary oedema, the circulation is being supported by endogenous catecholamines. The rapid onset of spinal blockade is extremely likely to result in hypotension. Pulmonary oedema will be exacerbated by the supine position. Hypoxia and restlessness will be common and difficult to manage. Dr Mostafa found that, by attention to detail, you can avoid these problems in some patients some of the time—but you cannot expect to avoid them in all the patients all of the time (Clark, Thompson and Thompson, 1976; Crawford, 1978).

The advantage of a careful, monitored general anaesthetic in these circumstances lies in the ease with which crises can be managed when they occur. In contrast to spinal anaesthesia, ventilation and oxygenation may be optimised, central venous catheters can be inserted and inotropic agents given.

Lunn and Mushin (1982) in their study “Mortality Associated with Anaesthesia” found that errors in the administration of spinal and extradural anaesthetics caused a disproportionately large number of anaesthetic deaths. They commented that: “There is no evidence that a method which abolishes autonomic and sensory response to surgery is particularly beneficial to the poor risk or moribund patient in, or on the verge of, cardiorespiratory failure. Indeed simple logic suggests that this would be a poor choice in these circumstances”.

I agree.

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REFERENCES


Sir,—Thank you for giving me the opportunity to reply to Dr Prince's letter.

It appears to me that he chose to ignore what was clearly stated in the article that “spinal anaesthesia is not normally recommended as a first choice for Caesarean section in parturients with mitral valve disease”. Furthermore, his statement that one cannot avoid problems associated with a particular technique (in this case spinal blockade) in all patients all of the time, clearly equally applies to any other anaesthetic technique. I will proceed to answer his remarks and I hope I will be forgiven for quoting some of his references.

Although hypertension may occur, its incidence has been significantly reduced (Clark, Thompson and Thompson, 1976) and spinal blockade for Caesarean section “has undoubtedly become a safer procedure for both mother and infant since the requirement to avoid caval compression has been realised” (Crawford, 1978). Hypotension in cases similar to ours is preferably treated by a pure α-adrenoreceptor stimulant, for example phenylephrine, not inotropes (Sakar and Marx, 1976). Our patient was not supine as Dr Prince described. Management of hypoxia and restlessness will be equally difficult, but similar, whether extradural or spinal block is administered.

Dr Prince's comments on general anaesthesia are interesting. The main disability of patients with mitral stenosis is essentially one of mechanical obstruction and not myocardial failure. Tachycardia shortens diastole, interferes with left ventricular filling and may precipitate further pulmonary oedema and hypotension. Even tachycardia greater than 110 beat min⁻¹ may cause pulmonary oedema (Barnes, 1976). The difficulties associated with induction of general anaesthesia are known (Churchill-Davidson, 1978). Anaesthesia- and intubation-induced tachycardia are difficult to prevent or treat, particularly in an urgent and unprepared patient. i.v. induction, particularly with barbiturates, is potentially lethal. Induction with nitrous oxide will be prolonged, difficult and require a low inspired oxygen concentration, while cyclopropane is contraindicated (Churchill-Davidson, 1978). Establishing such a patient on a mechanical ventilator is likely to be associated with a period of cardiovascular instability which is difficult to treat or control. High inspiratory inflation pressure will have to be used. Tachycardia and hypotension as a result of reduced cardiac output are likely to follow. These patients will be little helped by inotropic agents such as digoxin (Barnes, 1976) and β-adrenoreceptor stimulants which increase cardiac output or rate, or both, are contraindicated (Sakar and Marx, 1976). Insertion of central venous catheters will be not only time-consuming, but also unlikely to give clear indication of the
The state of left ventricular filling or performance. Pulmonary oedema can be, and is effectively, treated by reducing cardiac preload. The benefit of extradural block in this respect is recognized (Moir and Willocks, 1968; Barnes, 1976). Similar gains can be obtained from spinal block and this was exploited in our patient, with success.

From his remarks it is difficult to tell which technique Dr Prince would use: incremental extradural or general anaesthesia. This is not surprising. Management of such complicated cases is always open to criticism. Retrospectively, it is easy to say that other techniques would have been more effective. However, in an unprepared emergency Caesarean section the time factor is of paramount importance. It seems to me that Dr Prince did not appreciate this urgency. Not only maternal distress was present, but the fetus, who incidently spent 3 weeks in the special baby care unit with meconium aspiration, was facing imminent intra-uterine death. It is my view that neither technique he described would have been suitable for both mother and fetus.

Lunn and Mushin (1982) expressed their opinion that the "quality of the anaesthetist is more important in terms of outcome than the drugs or techniques he chooses to use". Their adverse comments in connection with spinal and extradural blocks are related to mismanagement and failure to recognize their disadvantages. The successful outcome in our case is evidence that the technique was fully exploited to the benefit of both mother and infant. Given the knowledge of the disadvantages of other techniques, spinal anaesthesia was not a poor choice; it was, in our view, the only choice in such circumstances.

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REFERENCES

THE PROBLEM OF INTRATECHAL KETAMINE
Sir,—I have read with interest the letter of Brock-Ume and his colleagues (1985). In their first publication they had reported that, after extradural ketamine, pain relief was obtained in every patient suffering from cancer pain (Mankowitz et al., 1982).

Other authors (Saissey et al., 1984) also reported on the analgesic effects of extradural ketamine in postoperative pain. In some of these patients the duration of pain relief was increased when repeated injections, or an infusion, was used. However, in one alcoholic patient the extradural ketamine failed.

Although complications after extradural ketamine in man have not been reported as yet (Mankowitz et al., 1982; Rubin et al., 1983; Saissey et al., 1984), clinical trials should be postponed temporarily, until any possible toxicity has been investigated fully.

In my opinion the differing views as to the toxicity of intrathecal ketamine when used in small or large animals may result from the spinal effects of the different concentrations of the drug used. In rats the volume of the solution injected intrathecally was restricted to 4–6 ml. (Ahuja, 1983) and the required dose of ketamine had to be injected in a very high concentration. The drug may be diluted by the CSF; however, in small animals this will not be as significant as in larger animals. This high drug concentration in small animals might explain why 9.1% of rats died in Ahuja’s experiment. In rats, the concentration of intrathecally administered ketamine was more than 100 times higher than in humans if given extradurally. This might explain the fact that Brock-Ume found nerve degeneration in small monkeys, but not in larger baboons (Brock-Ume, Kallichurum et al., 1982; Brock-Ume, Mankowitz et al., 1982). It seems reasonable, therefore, to devise experiments in which the histotoxic effects of different drug concentrations will be examined at spinal and supraspinal levels both microscopically and electronmicroscopically.

Since, as yet, we do not have a safe, widely used method with which to control pain, it would be very important to examine not only opioids, but also the role of other neurotransmitters in analgesia. As ketamine is acting not only on opioid receptors, but also on the noradrenaline and serotonin systems (Martin, Bouchal and Smith, 1982; Martin and Smith, 1982; Lundy and Jones, 1983), the proposed research may be interesting.

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REFERENCES