In investigating the technique, I chose to use large volumes of 0.5% bupivacaine, but only in an attempt to ensure that volume and concentration of local anaesthetic could not be implicated in failed blockade. I have no doubt, from subsequent personal experience, that smaller volumes and concentrations of local anaesthetic can provide an equally satisfactory and a safer block. Perhaps, with further investigation, one may indeed find, as Dr Dawson suggests, that extradural catheters in alternate intercostal spaces, provide the best answer, notwithstanding the great danger of pleural puncture.

Finally, I would concur with Dr Dawson in saying that Tuohy needles are in general too blunt for this technique. I have found, however, that the disposable Tuohy needles manufactured by Portex (U.K.) Ltd are extremely sharp and admirably suitable for this purpose.

D. F. Murphy
Dublin

REFERENCES


Sir,—The possibility of using pharmacological agents to facilitate recovery from brain damage has been subjected to animal experimentation (Feeney, Gonzalez and Law, 1982). In addition to the case studies in human subjects cited by these authors, we would like to refer to some studies in which catecholamines appeared materially to improve brain defects following head trauma (Luria et al., 1968; Gross, 1977; Marotta et al., 1977). In addition, a case treated by one of us (FJL), in which the patient (who had received extensive head trauma), presented as a "meningal vegetable", responded to the simultaneous administration of amphetamine and chlorpromazine (to control his psychotonic symptoms). On this regimen the patient returned to work after 6 months. Since Feeney, Gonzalez and Law (1982) intimate that catecholaminergic agents and neuroleptic drugs are mutually antagonistic in this situation, it would appear that this conclusion may be unwarranted. Thus, it has been shown that amphetamine is useful in the therapy of chronic schizophrenia, despite the concurrent use of neuroleptic agents (Nyman, 1975).

It is possible that there has been a delay in using these apparently mutually antagonistic treatments for patients suffering brain injury, since their combined use would appear to lack a rational basis. It seems that, during the acute phase of brain damage, amphetamine would be useful, while during the chronic phase (as demonstrated by the case we cite) combination with a neuroleptic agent would be better.

A possible explanation for this paradoxical effect is the finding that there are at least two types of dopamine receptors, one type inhibitory and the other excitatory (Cools and Van Rossum, 1980).

The sequence of head injury may well occur as a result of an imbalance of activity between these two types of receptors. In some cases, dopamine agonists would be sufficient to redress this imbalance, while in others a combination with neuroleptic agents would enhance the response.

Another factor related to this work is that naloxone has been shown to ameliorate the effects of brain injury (Baskin and Hosobuchi, 1981) and also to improve the state of consciousness (Baskin and Hosobuchi, 1981; Symons, Emson and Farman, 1982). Furthermore, coma patients subjected to sensory system stimulation seem to recover more readily (Le Winn and Dimascencu, 1978). It is possible that coma is a reflection of the opioid system (Gillman and Lichtigfeld, 1981). Stress has been shown to cause opioid release (Rossier et al., 1977). Thus, it is possible that the effect of restraint coupled with brain damage causes the release of opioids.

Therefore, the opioid system seems to play a crucial part in the expression of the symptoms of trauma as suggested by previously (Gillman and Lichtigfeld, 1981).

We have further suggested that there is an overall balance between the catecholaminergic and the opiateergic systems. This balance, once disturbed, could result in pathology, which at its most extreme could lead to brain death.

It is vital, we believe, particularly in the acute phase of brain injury, to consider the interplay between these systems.

M. A. GILLMAN
F. J. LICHTIGFELD
Johannesburg

NITROUS OXIDE DISTENDS THE BOWEL

Sir,—In his otherwise excellent article on anaesthesia and bowel surgery, Dr A. R. Aitkenhead (1984) failed to mention an important effect of nitrous oxide: namely, it causes bowel distension.

This is because nitrous oxide, being highly soluble, diffuses into the bowel faster than the less soluble intestinal gases, mainly nitrogen and methane, can diffuse out of it.

Eger and Saidman (1965), using closed segments of dog stomach, ileum and colon, measured increases in volume of
75–100% in 2 h, and 100–200% in 4 h when 70–80% nitrous oxide was added to the respired gases. This was confirmed by Steffey (1979) in dogs and ponies.

Most practising anaesthetists seem to be unaware of this work and continue to use nitrous oxide during abdominal operations. If they will only try omitting it, they will find that intestines are smaller and surgeons more grateful.

In cases of intestinal obstruction and closed pneumothorax, avoidance of nitrous oxide is even more important.

If Dr Aitkenhead knows of evidence which contradicts this work I would be grateful to be told of it. If not, I hope that you will give enough prominence to this letter to draw the attention of practising anaesthetists to this clinically useful information.

D. V. Thomas

REFERENCES


Sir,—I read with particular interest Dr Aitkenhead’s symposium article “Anaesthesia and Bowel Surgery” (Aitkenhead, 1984) and noted his emphasis on the problem of anastomotic breakdown.

I agree that regional anaesthesia offers a number of advantages, but would also suggest that when general anaesthesia is administered one should avoid high concentrations of nitrous oxide. Eger and Saidman (1965) found that intestinal gas volume in dogs increased 75–100% in 2 h and 100–200% in 4 h during nitrous oxide anaesthesia, whereas no volume changes were observed when the animals breathed halothane in oxygen alone. They suggested that nitrous oxide is relatively contraindicated in cases of intestinal obstruction. More recently it was demonstrated (Lewis, 1975) that, in healthy adult patients undergoing non-abdominal surgery, there is a significant increase in girth measurement during anaesthesia with 70% nitrous oxide.

The increase in volume of enclosed gas-filled spaces in the body is related to the alveolar nitrous oxide concentration. The theoretical maximum increase in gas pocket volume may be reduced from 400 to 100% by decreasing alveolar nitrous oxide from 80 to 50% (Eger and Saidman, 1965). Nitrous oxide-induced intraoperative distension may not only make abdominal closure more difficult, but could possibly predispose to postoperative ileus and cause tension on anastomotic sutures. This might partially explain any differences in anastomotic breakdown rate between regional and general anaesthesia.

G. B. H. Lewis

REFERENCES

