head lift and a tidal volume greater than 400 ml within 4 min of the onset of ventilation.

All my patients showed a sustained tetanic response to 50 Hz stimulation before nitrous oxide was withdrawn. Elimination of nitrous oxide, extubation and recovery of consciousness all take some time, so further recovery of the neuromuscular junction would have occurred before the patient would be awake enough to experience the "unpleasant subjective sensation" to which Dr Harrop-Griffiths refers.

Howard-Hanson and colleagues (1980) and Engbaek and colleagues (1985) studied blocking drugs other than atracurium in awake preoperative patients and volunteers. To extrapolate their findings to sleepy postoperative patients is unwarranted.

Antagonism of neuromuscular blockade is not without risk (for example bradycardia and disruption of intestinal anastomoses). Following infusions of low dose atracurium, patients should be assessed with train-of-four and tetanic stimulation. Where clinical observation demonstrates satisfactory ventilation, airway protection and reversal of paralysis, I believe that antagonism is not essential.

J. R. Sneyd
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REFERENCES

EXTRADURAL BLOCKADE AND INTRACRANIAL PRESSURE

Sir,—I am not qualified to comment authoritatively on the studies reported by Hilt, Gramm and Link (1986), although their results appear to accord with those of other workers to whom they refer. I do, however, take issue with some of the tentative conclusions drawn from these results by Hilt and colleagues and in your accompanying Editorial (Wildsmith, 1986).

We have in this service alone (and I am sure that we are not unrepresentative in this) administered a continuous lumbar extradural block for the labour of four women with benign intracranial hypertension and two with a space-occupying intracranial lesion, plus several who had been subjected to an intracranial surgical procedure. In none of these patients was there even a hint of an unwanted effect of any of the intradural injections.

Of much greater moment is the question of pre-eclampsia. In common with many of this country and elsewhere, we consider the provision of extradural blockade—for labour or for Caesarean section—to be the outstanding technique of choice in the care of mothers with pre-eclampsia of any grade of severity. The contraindications are well known: the mother declines the offer; coagulopathy; severe hyper-reflexia suggestive of imminent eclampsia; rapidly increasing severity of fetal distress demanding immediate abdominal delivery. Many hundreds of pre-eclamptic mothers have been given extradural analgesia in our service. I have no evidence that there has resulted any instance of the complication under review, and I would be most reluctant to include such a complication in a list of potential hazards relating to the provision of an extradural block to a patient with pre-eclampsia. Of course I would accept that, if one were so unwise as to inject a large volume of anaesthetic solution (more than 10 ml, for instance) at one time, the possible resultant increase in intracranial pressure might lead to unpleasant sequelae (although we never saw even that in the "old days"), but that complication is of minor importance compared with the other potential hazards associated with such injudicious management.

J. Selwyn Crawford
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REFERENCES

INTRACRANIAL HYPERTENSION AND CAUDAL ANAESTHESIA

Sir,—I read with interest the valuable Editorial by Dr Wildsmith (1986) and the original article by Drs Hilt, Gramm and Link (1986). All stressed the relative contraindication of extradural anaesthesia in the presence of increased intracranial pressure (ICP) "... not only because of the risk of tentorial herniation after accidental dural puncture, but also because of the risk of decreasing cerebral perfusion or aggravating brain shifts by increasing ICP." It was suggested that, perhaps, caudal analgesia would be a better technique (Crawford, 1978). However, we encountered two parturients with brain tumours who developed CNS complications after caudal analgesia (Abouleish, 1977). In those two patients the one-dose injection of 1.5% lignocaine 22 or 24 ml through a needle for forceps delivery resulted in apnoea, total loss of consciousness and severe bradycardia. The episodes lasted 5 or 6 min, respectively. Inadvertent intravascular injection was excluded, based on the absence of convulsions, and the presence of adequate analgesia, with T10 or T9 level of sensory block, respectively, and lasting for 90 min following the caudal injection.

The dural sac ends normally at the level of the second sacral vertebra — about 5 cm from the sacral hiatus. Thus the injection of a solution in the caudal canal supposedly affects the ICP less because of this distance from the subarachnoid space. However, with increased ICP, the dural sac may expand further down, and the rapid injection may transmit more pronouncedly than normal the increased pressure in the caudal canal to the subarachnoid space. Therefore, although on theoretical grounds caudal block is safer than extradural anaesthesia in patients with increased intracranial pressure, caution must be taken to minimize the increase in ICP by...
fractionating the dose to small volumes of 3 ml, injecting it over 5–10 min and observing the reaction of the patient to each injection. Another point that needs stressing is that, normally, the risk of dural puncture is less with caudal anaesthesia than with extradural anaesthesia. However, the distension of the subarachnoid space and possible expansion of the dural sac with ICP require more caution than usual. To avoid this problem, one should use a needle-cannula catheter to perforate the posterior sacrococcygeal ligament, and advance only the cannula once the ligament has been perforated (Owens, 1973).

E. ABOULEISH
Houston, Texas

REFERENCES

Sir,—We appreciate Dr Abouleish’s comment on our article and the information about the two parturients developing CNS complications following caudal anaesthesia. This is stressing the problem pointed out in our article.

Furthermore, we agree completely with Dr Crawford that extradural anaesthesia is of outstanding value in the management of patients with pre-eclampsia but, as we discussed, there might be a risk of untoward effects in patients with compromised intracranial compliance. As we could demonstrate in our first patient, the extradural application of 10 ml, the upper limit mentioned by Dr Crawford, can lead to a dramatic increase in ICP. Even 5 ml induced a distinct increase in ICP. If, in these patients the extradural injection is not made with careful fractioning of the dose, as proposed by Dr Abouleish and concluded in our discussion, CNS complications might occur. The frequency of these untoward effects depends on the reduction of the intracranial compliance and the precautions taken.

Regarding the potential hazard, we conclude that extradural anaesthesia has to be administered with caution in patients with restricted intracranial compliance. In patients with intracranial pressure, so that others might avoid the problems method is contraindicated.

H. HILT
H.-J. GRAMM
J. LINK
Berlin

Sir,—Thank you for letting me see the letters from Drs Crawford and Abouleish. The latter’s case serves to support the view I expressed in the editorial, that extradural injections must be made slowly and with particular caution in a patient who may have an increase in intracranial pressure. The second paragraph of Dr Crawford’s letter demonstrates that these patients can still receive the benefits of a continuous extradural technique.

In response to Dr Crawford’s third paragraph, all I can do is quote from my Editorial: “In a severely pre-eclamptic patient the benefits of extradural analgesia during labour are clear....”. The basic point of the editorial was to draw attention to the report that extradural injection can increase intracranial pressure, so that others might avoid the problems encountered.

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