Extradural blockade is commonly used in our clinic to provide anaesthesia, pain relief and suppression of the sympathetic nervous system in critically ill patients. The technique is also recommended during labour for pain relief and the control of arterial pressure in patients with severe eclampsia (Bonica, 1980).

In patients with a head injury and increased ICP, extradural anaesthesia may be relatively contraindicated because of the risk of tentorial herniation after accidental puncture of the dura. In women with severe eclampsia, the presence of cerebral oedema with a reduction in intracranial compliance must be considered. To date, no direct measurements of the effect of extradural injection on ICP have been published. We had the opportunity to measure the ICP response to lumbar extradural injection in two patients.

PATIENTS AND METHODS

Patients

Case Report 1. A 39-year-old woman presented with multiple injuries, which included a blunt head injury. There was no intracranial space-occupying lesion and the patient's initial Glasgow Coma Score was 6. An extradural pressure transducer was inserted through a burr hole in the skull on the first day to monitor ICP and the alterations resulting from therapy. Four weeks later, an extradural catheter was inserted at the 3rd lumbar space to aid surgical management of femoral fractures. At this time the patient was still comatose, but no localizing neurological deficit was present. Mean ICP varied between 5 and 20 mm Hg. After surgery, the extradural technique was continued, to provide analgesia. At 4-hourly intervals, 0.25% bupivacaine 10 ml was injected over 20–30 s. Once increases in ICP had been recognized, the regimen was changed to 0.5% bupivacaine 5 ml, injected at the same rate, but the use of extradural analgesia was abandoned soon afterwards. The possibility that the catheter had entered the subarachnoid space was excluded clinically. Sixteen single ICP responses to 10 ml, and four responses to 5 ml, were evaluated from the patient's record.

Case Report 2. ICP monitoring was instituted because of severe head injury with diffuse oedema (no space-occupying lesion) in a 21-year-old...
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TABLE I. Summary of results. ICP$_1$ = ICP before extradural anaesthesia; ICP$_p$ = peak pressure; $\Delta p$ = absolute change in ICP; time = duration of ICP increase

| Agent          | Patient 1 (ICP$_1 <$ 20 mm Hg) | Patient 1 (ICP$_1$ > 20 mm Hg) | Patient 2 | | |
|----------------|---------------------------------|---------------------------------|-----------|---|---|---|---|---|---|---|---|
|                | ICP$_1$ (mm Hg)                | ICP$_p$ (mm Hg)                | $\Delta p$ (mm Hg) | Time (s) |
|                | Mean | Range | SD    | Mean | Range | SD    | Mean | Range | SD    | Mean | Range | SD |
| Bupiv. 10 ml   | 9    | 15.5  | 11–19 | 3.13 | 33.0 | 22–41 | 5.81  | 17.44 | 11–23 | 3.92 | 285 | 145–365 | 70 |
| Bupiv. 5 ml    | 7    | 25.0  | 20–28 | 7.54 | 47.85| 33–63 | 10.04 | 22.86 | 13–35 | 2.95 | 238 | 165–335 | 53 |
|                | 4    | 17.0  | 15–19 | 1.83 | 23.25| 21–25 | 1.79  | 6.25  | 6–7   | 0.43 | 169 | 90–245 | 69 |
| Bupiv./NaCl 10 ml | 6    | 9.33  | 6–11  | 1.89 | 15.66| 10–18 | 2.75  | 6.33  | 4–10  | 1.89 | 140 | 85–280 | 71 |
| Bupiv. 15 ml   | 1    | 5     | —     | —    | 22   | —     | —    | 17    | —     | —   | 295 | —     | —   |
| Bupiv./NaCl 20 ml | 2    | 6     | 6–6   | —    | 26.5 | 26–37 | —    | 20.5  | 20–21 | —   | 220 | 155–330 | — |
| NaCl 20 ml     | 1    | 6     | —     | —    | 26   | —     | —    | 20    | —     | —   | 350 | —     | —   |
| NaCl 10 ml     | 1    | 8     | —     | —    | 18   | —     | —    | 10    | —     | —   | 185 | —     | —   |

FIG. 1. Intracranial pressure response to bupivacaine hydrochloride 10 ml in patient 1 with increased baseline ICP.

FIG. 2. Intracranial pressure response to bupivacaine hydrochloride 10 ml in patient 1, continuing on a plateau with B-waves.

Patient with multiple injuries. During the 2nd week after injury, an extradural catheter was inserted at the 3rd lumbar space with the aim of resolving a paralytic ileus by producing sympathetic blockade. At the time, ICP was normal, there were no pathological pressure waves and there was no neurological deficit. The patient was sedated with a barbiturate. The first two injections were of 0.25% bupivacaine 15 and 20 ml. Because of unacceptable increases in ICP, the dose was reduced to 10 ml given over 20–30 s every 4 h. In this patient, measurements were also recorded after the injection of 10 and 20 ml of physiological saline.

**ICP measurement**

ICP was monitored continuously using an extradural pressure transducer (Gaeltec), a Hellige amplifier (Servomed 5S) and a paper recorder (Servomed 130T). The transducer was calibrated regularly.

**RESULTS**

In both patients reproducible increases in ICP (summarized in table I) were produced by the lumbar extradural injection of bupivacaine 10 ml, the ICP trace following a characteristic course (fig. 1). On one occasion the increase in ICP lasted for 11 min and B-waves were observed (fig. 2). A specific effect of bupivacaine was excluded by the injection of the same volume of physiological saline in the second patient.

No changes in mean arterial pressure (radial artery) were observed in association with the induced increases in ICP.

**Peak ICP**

The peak ICP, which ranged from 11 to 63 mm Hg, exceed the physiological resting value of 5–10 mm Hg (Lundberg, 1972) on every occasion. In the first patient, the injection of bupivacaine 10 ml increased ICP from 18.8 to 39.5 mm Hg (mean of 16 measurements). After 5 ml, ICP increased from 17 to 22.3 mm Hg (four measurements). In the second patient, 10 ml produced an increase from 9.3 to 15.7 mm Hg (six
measurements). The injection of two larger volumes produced even greater increases (5–22 mm Hg after 15 ml; 6–26.5 mm Hg after 20 ml).

Figure 3 shows the ICP responses in three different conditions. Figure 3A shows seven measurements made in the first patient when baseline pressure was > 20 mm Hg and figure 3B shows nine measurements when it was < 20 mm Hg. Figure 3c shows the responses of the second patient, measured on six occasions after 10 ml and twice after 20 ml. The absolute peak pressure and the actual increase correlated well with baseline ICP. The higher the initial ICP, the greater the increase (fig. 4).

Time course of ICP increase

ICP started to increase 5–10 s after the start of the extradural injection. With 10 ml, the mean time to peak pressure was 43.8 s in the first patient and 34.2 s in the second. Thus ICP continued to increase after the end of the injection. Having reached its peak, ICP decreased exponentially. In the first patient, ICP was increased for an average of 4 min 25 s after injection of 10 ml and for 2 min 49 s after 5 ml. In the second patient, with a normal baseline pressure, the increase in ICP lasted for an average of 2 min 20 s after 10 ml. After 15 and 20 ml, the increase persisted for 2 min 35 s and 5 min 50 s, respectively.

Fig. 3. Schematic graphs of the ICP responses to extradural volume load. A: Patient 1. Bupivacaine 10 ml and baseline ICP greater than 20 mm Hg. B: Patient 1. Same volumes but baseline ICP less than 20 mm Hg. C: Patient 2. Responses to bupivacaine 10 ml (●), 0.9% sodium chloride 10 ml (■), bupivacaine 20 ml (○) and 0.9% sodium chloride 20 ml (●).

Fig. 4. ICP increases after 10-ml extradural injection at L3/4, plotted against baseline ICP.
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DISCUSSION

Although measurements comparable to those described here have not been published previously, there have been reports on the relationship between cerebrospinal fluid (CSF) pressure and lumbar extradural pressure. Pollock and Boshes (1936) showed that pressure changes in extradural veins are transmitted to the CSF. Furthermore, jugular vein compression is known to produce an increase in lumbar extradural pressure (Bryce-Smith, 1950; Shah, 1981). This can only be explained by an increase in intracranial pressure being transmitted to the lumbar CSF.

Burn, Guyer and Langdon (1973) pointed out that rapid or large-volume extradural injection can produce discomfort or even unconsciousness in alert patients. This had previously been reported by Evans (1930), who injected up to 140 ml, but could not explain whether the effect was the result of drug intoxication or of a transmitted pressure change. Burn, Guyer and Langdon (1973) implied that it was caused by the latter.

The first measurements indicating that lumbar CSF pressure is affected by an extradural volume load were published by Buchholz and Lesse (1951). They injected up to 150 ml, but did not record any increase in CSF pressure with less than 40 ml. No neurological side effects were reported. Simultaneous measurements of lumbar extradural and lumbar subarachnoid pressures during and after extradural volume load were published by Usubiaga and colleagues (Usubiaga, Wikinski and Usubiaga, 1967; Usubiaga et al., 1967). They found that even 10 ml produced a marked increase in lumbar CSF pressure and that the response was dependent on the volume and rate of injection. The increase in subarachnoid pressure was greater than in the extradural space and lasted for 3–10 min, producing traces similar to the ICP records presented here. A relationship to some cerebral symptoms was noted.

Bromage (1967) has stated that one of the central effects of extradural block is a transient change in CSF pressure, and Greene (1981) recently argued that it is an increase in ICP which induces cerebral symptoms shortly after injection. The relationship of the pressure increase to cerebral symptoms has been stressed by Nolte (1978) and Cousins (1980). In sheep, which have a very low spinal extradural compliance, the injection of 5–10 ml produced syncope lasting 60–90 s (Lebeaux, 1974).

The finding that extradural volume load increases ICP may be explained as follows. The dural sac is compressed by the increased extradural pressure that follows injection and this causes a shift of CSF into the cranium. Depending on the intracranial compliance, some degree of ICP increase is produced. The linear correlation between baseline ICP and its subsequent increase is comparable to the results published by Leech and Miller (1974) and Miller (1975). They studied intracranial pressure responses to defined intracranial volume changes and, in our opinion, the increase in ICP response described here represents the same volume–pressure relationship.

Our results demonstrate that extradural injection may increase ICP. We conclude that extradural anaesthesia should not be used in patients with severe intracranial hypertension or space-occupying lesions, 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. Caution is also necessary in situations of compromised intracranial compliance, for example in women with severe pre-eclampsia requiring extradural analgesia during delivery. In such patients, a very slow injection rate should be used.

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REFERENCES


