REFERENCES

Sir,—As well as suggesting a useful application of transsacral block, the high frequency of dural puncture experienced by Dr Ryan in his series will be of considerable interest to others using the technique. Although the subarachnoid space in the normal subject ends at the lower border of S2, extensions to S3 and S4 present an occasional hazard in the use of caudal analgesia. Finger-like projections of the subarachnoid space along the course of the sacral nerves have been noted at dissection (Hingson and Southworth, 1946), but these were caudad in direction and considered to be relevant only to the caudal approach to the extradural space. The current widespread popularity of caudal analgesia, particularly in children, appears to indicate that, while this hazard undoubtedly exists, its frequency is low.

Whereas the caudal needle is advanced through the sacro—coccygeal hiatus cephalad towards the dura, transsacral block using Moore’s approach (Moore, 1965) is accomplished by vertical insertion of the needle to a predetermined depth through the appropriate foramen. Frequent dural puncture under the circumstances can only be explained by an unexpectedly high frequency of CSF containing meningeal projections accompanying the sacral nerves as far as their respective foramina. Although I have not encountered this problem in 54 S4 blocks, a lower frequency at this level is to be expected because of the relative distances of the S3 and S4 foramina from the normal termination of the dura. I note, however, that it was not a feature of the recently published series of S3 blocks for bladder pain (Simon, Carron and Rowlingson, 1982) or for incontinence (Essenhig and Ryan, 1982) and the former authors commented on the negligible morbidity and safety of the procedure for outpatient use.

It is difficult to assess the extent of the problem without exact knowledge of the technique used. Although stating in his letter that his approach is essentially the same as that used by myself, Dr Ryan specified the use of a Tuohy needle in the original series to which he referred. The possibility of dural puncture could be considerably increased by the use of such a needle directed medially into the sacral canal and maximized by further manipulation cephalad.

Although resting urethral sphincter tone has been shown to increase after bilateral S4 block (Torrens and Griffith, 1974), my own series was confined to unilateral blockade at this level and resulted in no reports of change in bladder control in any of the patients studied.

D. H. ROBERTSON
Stirling

REFERENCES

TABLE I. Summary of results of S3 block. Minimum follow up 3 months

<table>
<thead>
<tr>
<th>Clinical condition/Technique</th>
<th>n</th>
<th>Dry</th>
<th>Improved</th>
<th>No change</th>
<th>Comment</th>
</tr>
</thead>
<tbody>
<tr>
<td>Multiple sclerosis</td>
<td>12</td>
<td>6% Phenol</td>
<td>8</td>
<td>2</td>
<td>2</td>
</tr>
<tr>
<td>Instability obstruction</td>
<td>23</td>
<td>Bupivacaine + steroid</td>
<td>18</td>
<td>3*</td>
<td>2</td>
</tr>
<tr>
<td>“Idiopathic” instability</td>
<td>8</td>
<td>Bupivacaine + steroid</td>
<td>2</td>
<td>—</td>
<td>6</td>
</tr>
<tr>
<td>Miscellaneous (Dementia 4, frontal lobe tumour 1, schizophrenia 1)</td>
<td>6</td>
<td>Bupivacaine + steroid</td>
<td>—</td>
<td>2</td>
<td>4**</td>
</tr>
</tbody>
</table>

HARMDYNMIC CONSEQUENCES OF APPLICATION OF ACRYLIC CEMENT DURING INDUCED HYPOTENSION

Sir,—We read with interest the article of Vazeery, Skeie and Anda (1983) describing the changes in cardiac output and systemic arterial pressure after the insertion of acrylic cement during trimetaphan, sodium nitroprusside and glycerol trinitrate-
induced hypotension.

Unfortunately, they did not monitor the end-expiratory carbon dioxide during the operation. Previously, we have observed abnormal decreases in the capnograms after the insertion of hip prostheses using acrylic cement. These represent wash-out curves typical for pulmonary embolism as described by Smalhout and Kalenda (1975). As an example figure 1 shows such a curve obtained from a 78-yr-old female during general anaesthesia after the insertion of a hip prosthesis using acrylic cement.

We want to draw attention to the fact that, contrary to the authors' opinion, a decrease in cardiac output combined with a decrease in arterial pressure is not necessarily the consequence of peripheral vasodilatation, but can also be the result of the aforementioned embolism in the lung (Halmagyi, Starzecki and Horner, 1964). This can also explain their results in the control group.

If, in dogs, a miliary pulmonary lesion is induced, sodium nitroprusside will cause a decrease in pulmonary artery pressure and pulmonary vascular resistance, but no change in cardiac output (Colley, Cheney and Hlastala, 1979). This is probably the consequence of an increased blood flow to the unaffected parts of the lung (Hill, Sykes and Reyes, 1979).

Thus, in our view the haemodynamic changes following the application of acrylic cement are most probably the consequence of embolism in the lung, which during induced hypotension is masked by some drugs depressing the protective response of hypoxic pulmonary vasoconstriction (Editorial, 1979).

JAAP J. DE Lange
LEO H. D. J. Booij
Amsterdam

REFERENCES


Sir,—In reply to the letter from De Lange and Booij, may I make the following comments:

1. We did not monitor the end-expiratory carbon dioxide. However, in a subsequent investigation we measured blood-gas tensions in 50 patients just before and at 1, 2, 4, 6 and 8 min after the insertion of acrylic cement, in patients operated under normotension, extradural anaesthesia, and trimetaphan, nitroprusside and nitroglycerin hypotension. There were no changes noticed in carbon dioxide, but a definite pulmonary hypoxic response was encountered under normotension and extradural blockade. However, changes in arterial $\text{Po}_2$ were insignificant after the implantation of acrylic monomer during trimetaphan, nitroprusside and nitroglycerin hypotension.

   A. K. VAZERY
   Stavanger

THE LARYNGEAL MASK

Sir,—Dr Brain (1983), in his article on the Laryngeal Mask states that: "During IPPV (with a Manley Blease ventilator) ... the readings obtained from the Wright respirometer (expired minute volume) were all greater than the inspiratory minute volume as measured by rotameter readings, presumably reflecting the known inertial characteristics of this respirometer, ..."

We believe that this explanation is inadequate and offer an alternative.

Measurements of inspired and expired minute volumes were made at different points in the circuit, the rotameters being proximal to, and the respirometer distal to, the ventilator. Thus gas volume measurements were made at different pressures.

Assessment of the accuracy of Wright's mechanical respirometer (Nunn and Ezi-Ashi, 1962) suggests that its inertial characteristics are unlikely to account for the reported difference (14.68%).

Back pressure as a result of the resistance of a ventilator in series causes rotameters to underestimate delivered gas volume, and we believe this to be the major factor operating.

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A. HUTCHINSON
Leeds

VENTRICULAR ARRHYTHMIA OR SUPRAVENTRICULAR ARRHYTHMIA WITH ABDOMINAL CONDUCTION

Sir—Sigurdsson, Werner and Fahrens (1983) reported the occurrence of anomalous QRS complexes in more than half of a group of children undergoing adenoidectomy. The anaesthetic technique included 2.5–3% halothane in 50% nitrous oxide and oxygen, spontaneous ventilation and no tracheal tube. Halothane is known to displace the $\text{PCO}_2$ – ventilation response curve to the right and to reduce its slope (Severinghaus and Larson, 1965). The resulting hypercapnia is likely to be even more severe in the absence of an endotracheal tube. Hypercapnia increases plasma catecholamine concentrations and has been implicated in contributing to arrhythmias occurring during halothane anaesthesia (Benuzumof, 1981). We studied 31 children undergoing cleft palate repair under halothane anaesthesia (end-tidal halothane was 0.8 ± 0.4%), nitrous oxide and oxygen via an endotracheal tube (Karl et al., 1983). We controlled ventilation to produce a normal or low end-tidal carbon dioxide concentration. Despite the addition of adrenaline 0.5–14.3 $\mu$g kg$^{-1}$ (mean value 5.3 $\mu$g kg$^{-1}$; SD 3.2) in 1% lignocaine for local haemostasis, we saw no ventricular or supraventricular arrhythmias.

End-tidal $\text{CO}_2$

MONK PROSTHESIS

FIG. 1. The typical wash-out curve after insertion of a hip prosthesis.