Sir,—Dr Gordon claims that the evidence I presented is consistent with his claimed description of an exclusive low pressure theory of "hydrops syndrome" (Gordon, 1983)—a theory based on anecdotal evidence only and refuted in the same journal (Colman, 1983). If I take each of Gordon's points in turn, it is clear that there is no evidence to support a low pressure origin:

(1) The CSF pressure decrease described by Usubiaga, Wikinski and Usubiaga (1967) is that from a high pressure towards normal. These workers did not demonstrate any subnormal pressures and, in fact, noted increased residual pressures after injection. It has recently been shown that a similar increase in intracranial pressure occurs during extradural injection (Hilt, Gramm and Link, 1986) which may be prolonged in cases of reduced cerebrospinal compliance (Hardy, 1986).

(2) Spinal and extradural analgesia may be associated with a decrease in arterial pressure—well after the time period described. In the cases I described, arterial pressure monitoring showed no change after injection.

(3) Dural puncture in the presence of extradural injection of local anaesthetic would result in massive (total) spinal anaesthesia. This did not occur in the patients reported.

(4) The young patients described were pregnant women at term in labour. This is associated with increased intraspinal pressures (Messih, 1981).

(5) The patients were horizontal throughout which, as Dr Gordon notes, would minimize or negate any low pressure effect.

It would be interesting to know exactly on what experimental data Dr Gordon bases his low pressure theory. In the sources he quotes there is one clear description of "hydrops syndrome" associated with increased intracranial pressure which responded to CSF drainage (Sismanis et al., 1985).

Since my original letter I have noted both auditory and, in particular, vestibular disturbance in a patient during extradural injection of steroid-saline mixture in the treatment of low back pain. The 37-yr-old woman had a history of Menières disease, but was in remission. An extradural puncture was performed at L2-3 with the patient in a left lateral position. Injection of a solution of methylprednisolone 40 mg in 0.9 per cent sodium chloride was performed at a rate of 4 ml min⁻¹. By the time 16 ml had been injected, the procedure had to be abandoned because of disabling waves of vertigo and the feeling of nausea. These symptoms persisted for about 5 min before gradually subsiding. Arterial pressure was stable throughout. The patient would not open her eyes during injection, so that it was not possible to check for nystagmus. After an uneventful recovery she described marked tinnitus and almost complete deafness during this episode. She also remarked that the symptoms were identical with but more severe than those she experienced with the Menières. In this patient, vestibular disturbance predominated although auditory disturbance was present also. The only change in association with these symptoms was a slow extradural injection, a procedure which is associated with increased spinal and intracranial pressures.

Do these symptoms matter? There are two reasons why these observations are important. To the anaesthetist these same symptoms may be caused by systemic local anaesthetic toxicity and not, as in this patient, a purely mechanical effect. To the otologist the mechanism is important, since there have been no studies of intracranial pressure in patients with VIII nerve diseases. The relationship between ICP and vestibular cochlear mechanisms would be worthy of further study.

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