


Sir,—I admit the omission of the word “randomized” from the methodology and apologize for this error. However, I would have hoped that we were a sufficiently reputable group of workers that it would be realized that this was a mistake.

Increasing the numbers in two series does not introduce bias provided, as we have stated, “neither patients nor observers knew the medication given”. I was not involved in the observations or the anaesthesia, but I kept a constant watch on the findings and without the knowledge of my junior colleagues, withdrew temazepam and zopiclone after 120 observations, restricting the study thereafter to midazolam. With two operating lists per week containing one or two suitable patients, this had already taken about 1 year. Since most lecturers and research fellows have a 1-year contract, any alternative would have introduced a new group of observers.

The very exhaustive survey of our method of evaluation by Morrison, Hill and Dundee (1968) showed the fears of observer difference to be negligible. The alternative of one person making 120 or 144 observations is quite unacceptable because of the problem of boredom and inadvertent attempts at “breaking the code”.

Your correspondent can have little contact with the real world of clinical research to consider lack of “money, availability of staff and time” as excuses for an incomplete study—they are facts of life. It he wants another, our hospital availability of staff and time as excuses for an incomplete study.

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J. W. Dundee Belfast

REFERENCE


BLOCKED EAR AFTER EXTRADURAL INJECTION

Sir,—Hardy (1986) reported three patients with hypoacusis after extradural injection. In fact they complained of a blocked ear (sensation of fullness or cotton wool). While hypoacusis is usually associated with subjective hearing difficulty and low tone hearing loss, these may be absent. Serial pure tone audiometry is needed to establish the hypoacusis.

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Hardy noted that these symptoms of slight endolymphatic hydrops can occur after decreased CSF and perilymph pressures, but still claimed his patients’ problems were caused by increased pressures. However, his findings are quite consistent with the more generally applicable low pressure theory. Note that:

(1) Symptoms occurred immediately after, rather than during, injection at a time CSF pressure is rapidly decreasing (Usubiaga, Wikinski and Usubiaga, 1967).

(2) Injection causes arterial pressure to decrease (Lund, 1971). Hydrops is associated with arterial hypo-, not hyper-, tension (Gordon, 1983).

(3) There may have been undetected dural puncture, with CSF leak or decompression.

(4) Two patients were young women. Injection CSF pressures are higher in older patients (Usubiaga, Wikinski and Usubiagi, 1967), while low pressure headaches are commoner in younger patients (Lund, 1971).

CSF hydraulics are complex and it is easy to make false assumptions. Thus it is generally considered that aqueduct stenosis is caused by hydrocephalus, even though the narrow lumen strongly suggests a centripetal pressure gradient with low pressures in parts of the CSF system (Gordon, 1984). There has long been confusion as to whether postspinal puncture headache is the result of increased or decreased CSF pressure (Lund, 1971). Aboulker (1919) claimed Meniere’s syndrome was the result of intracranial and labyrinthine hypertension and that symptoms (but not deafness) were relieved by decompression. However, instead of the expected jet of CSF at lumbar puncture, it often came out drop by drop or even a dribble. His explanation was that pressures vary at different parts of the CSF system.

There are more general reasons for discounting a causal increase in pressure. Hydrops symptoms do not occur in intracranial hypertension from space-occupying lesions, although vertigo may be a late brainstem symptom (Zulch, Mennel and Zimmermann, 1974). There is no aural analog of papilloedema or congestive inner ear (Kaarber and Zilstorff, 1976). Nor do they occur in pseudotumor cerebri, although a case of hydrops-type deafness has been reported (Simonsen et al., 1985). Significantly, they did not attribute this to increased perilymph pressure, despite immediate remission of deafness and tinnitus upon drainage of CSF, since the deafness also remitted during jugular vein compression, which increases CSF and perilymph pressures.

It is important to determine if high CSF pressure ever causes early hydrodynamic symptoms, since otherwise they will be specific to CSF hypotension, unlike all the other symptoms which are common to CSF hyper- and hypotension (Frederiks, 1976). They would then be a simple and very useful clinical indicator of low pressure in lumbar puncture headache, overshunted hydrocephalus, spaceflight sickness, psychiatric disease, etc.

Furthermore, the misleading high pressure theories of the causation of Meniere’s disease could be discarded. The way to settle the issue is to question patients carefully during CSF pressure measurements for all the symptoms of hydrodynamic symptoms. The full early hydrodynamic syndrome comprises a blocked ear, fluctuant low tone deafness or, on occasion, improved pure tone sensitivity, vertigo, tinnitus, audio sensitivity and lowered acoustic reflex thresholds (Gordon, 1986). The effect of posture should be noted, since low pressure symptoms should improve on lying down, while high pressure ones worsen.

Finally, idiosyncratic differences, in temporal bone anatomy or otological history perhaps, must predominate over general theoretical considerations, since side-effects occur in only a...