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Mean intracranial pressure monitoring by a non-invasive audiological technique: a pilot study

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SUMMARY Intracranial pressure is normally transmitted to the perilymph of the cochlea via the cochlear aqueduct. The relationship between perilymphatic pressure, indirectly measured by tympanic membrane displacement, and mean intracranial pressure defined either clinically or by direct measurement has been examined in 58 patients (aged 5–77 years), with hydrocephalus, benign intracranial hypertension, intracranial tumours, subarachnoid haemorrhage and head injuries. The most consistent results were obtained in young patients with hydrocephalus and benign intracranial hypertension. However, the technique was not suitable when the stapedial reflex was absent as a result of middle ear/brainstem dysfunction and did not reflect intracranial pressure when the cochlear aqueduct was not patent. This pilot study suggests that the tympanic membrane displacement technique may provide a useful non-invasive method for serial monitoring of intracranial pressure in young patients with hydrocephalus or benign intracranial hypertension.

The direct measurement of intracranial pressure involves either surgical intervention, if an intracranial mass lesion is present, or lumbar puncture. However, if the cochlear aqueduct is patent, intracranial pressure is transmitted to the perilymph of the cochlea.[1] Perilymphatic pressure can be assessed indirectly by recording displacement of the tympanic membrane during stapedial reflex contraction elicited by a loud sound. In a preliminary study[2] we reported the effects of shunting for either hydrocephalus or benign intracranial hypertension on tympanic membrane displacement in three patients. We wished to explore the problems of using this technique in a wide variety of patients with possible intracranial hypertension and compare it wherever possible with direct measurements of cerebrospinal fluid pressure.

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Patients

Fifty eight patients (aged 5–77 years) were examined. Their final diagnosis included a combination of hydrocephalus (34), intracranial tumours (13), benign intracranial hypertension (5), subarachnoid haemorrhage (6), head injuries (4) and miscellaneous (5), table 1. Thirty two were diagnosed as having raised intracranial pressure and 12 subsequently

Table 1 *Category of intracranial pressure via different bases*

				Direct measurement via			
Diagnosis (a combination of)	*N*	*Clinical assessment*	*Ventricular drain (≤200 mm)*	*Ventricular catheter*	*Ventricular reservoir*	*Subdural catheter*	*Lumbar puncture*
Hydrocephalus	34	24	2	4	2	0	2
Intracranial tumours	13	12	1	0	0	0	0
Benign intracranial hypertension	5	0	0	0	0	0	5
Sub-arachnoid haemorrhage	6	4	2	0	0	0	0
Head injury	4	0	0	0	0	4	0
Miscellaneous*	5	4	0	0	0	0	1 (Normal)

*Miscellaneous included: Cerebral Thrombosis (1), Alzheimer's Disease (2), Sagittal Sinus Thrombosis (1), Normal (1).

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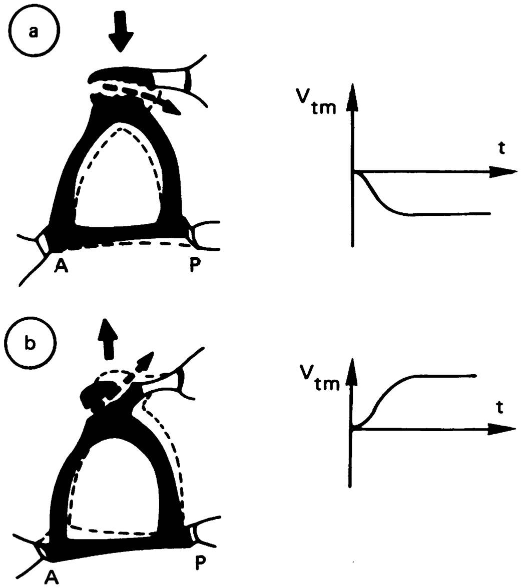

Fig 1 *Stapes movement and resulting tympanic membrane displacement when the acoustic reflex is induced in conditions of (a) raised and (b) normal/low perilymphatic pressure. V represents the volume displacement of the tympanic membrane, negative when the membrane moves inward and positive when outward.*

underwent a lumbar/ventriculo-peritoneal shunt operation. Direct measurement of cerebrospinal fluid pressure or intracranial pressure, by lumbar puncture or intraventricular or subdural pressure monitoring, was compared with the results of tympanic membrane displacement in 17 patients. In an additional six patients where direct measurements were undertaken no tympanic membrane displacement could be recorded. These patients included those with head injuries (4), subarachnoid haemorrhage (1) and hydrocephalus (1). In the remaining patients the evidence of raised or normal intracranial pressure was based upon clinical history and findings, the presence of papilloedema and evidence of midline "shift" or acute ventricular dilatation on computed tomography.

Methods

Perilymphatic pressure will reflect cerebrospinal fluid pressure when the cochlear aqueduct is patent. Changes in the perilymphatic pressure produce small but measurable variations in the kinematics of the middle ear ossicles and tympanic membrane. Alterations in pressure influence the resting position of the stapes footplate in the oval window, and consequently the degree of freedom of the ossicles and tympanic membrane to move in an inward or outward direction. Contraction of the stapedius muscle, attached by a ligament to the head of the stapes, moves the ossicle, fig 1. High perilymphatic pressure displaces the resting position of the stapes footplate laterally, thereby allowing a higher degree of freedom for motion in a medial direction, and correspondingly a more inward-going tympanic membrane displacement on stapedial contraction, fig 1, a. Low perilymphatic pressure will have an opposite effect, fig 1, b. Thus the pattern of tympanic membrane displacement during stapedial reflex contraction is a measure which relates to perilymphatic pressure,[3] and possibly to intracranial pressure.[2]

Table 2 *Tympanic membrane displacement and intracranial pressure*

(a) Tympanic membrane displacement	*CSF pressure* ≤ 200 *mm* (normal)*	*CSF pressure* > 200 *mm* (high)*
Inward high	2	6
One ear inward and one outward	0	1
Outward (normal)	7	1

*Includes patients on ventricular drainage.

(b) Tympanic membrane displacement	*Intracranial pressure (clinical)* Normal	*Intracranial pressure (clinical)* Raised
Inward (high)	6	11
One ear inward and one outward	2	5
Outward (normal)	22	4

The movement of the tympanic membrane was measured with special computer-based instrumentation which will resolve volume displacements as small as a nanolitre.[3] A transducer probe, attached to a headset, was placed into the patient's external auditory meatus. A 1000 Hz stimulus at sound pressure levels of 100, 110 and 115 dB SPL induced controlled stapedial muscle contraction and ossicular and tympanic membrane movement. Using this method changes in perilymphatic pressure were *indirectly* quantified in terms of the mean displacement, Vm. With shunt patients, measurements were made 1–2 days before and 3–5 days after the operation. Tympanometry was performed to ensure that the middle ear pressure was normal and that no change occurred between or within tests. In 17 patients cochlear aqueduct patency was determined by measuring the change in Vm from sitting to lying; a decrease in Vm of at least 20% at 110 and 115 dB SPL is taken to indicate patency.

Results

The procedure was generally well tolerated except in two patients who were too restless for tympanic membrane displacement measurements to be made. In the 17 patients in whom comparison with direct measurements of cerebrospinal fluid pressure was possible tympanic membrane displacement correctly categorised 13 patients (76%) and incorrectly placed three (18%), table 2, a. In one patient the tympanic membrane displacement was inward in one ear and outward in the other. Pressures greater than 200 mm cerebrospinal fluid were regarded as raised.[4] In table 2

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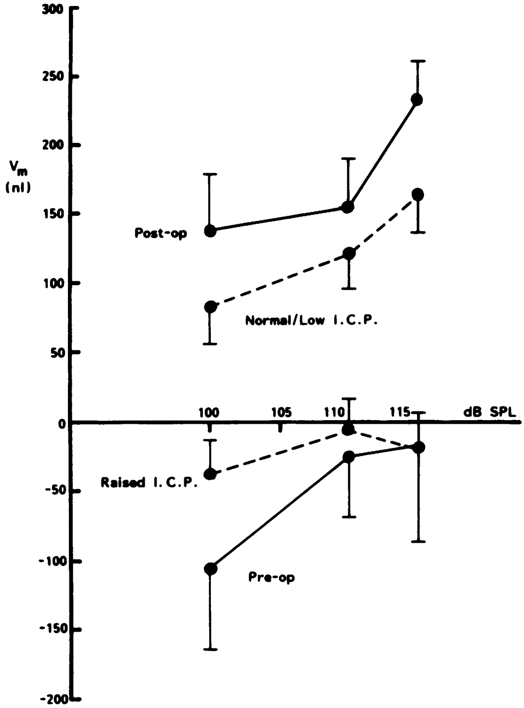

Fig 2 *The mean tympanic membrane displacement, Vm, averaged across patients with raised or normal intracranial pressure (dashed lines) and across shunted patients (solid lines) at three intensity levels (with standard error bars).*

(b) tympanic membrane displacement was related to the clinical assessment of intracranial pressure. Tympanic membrane displacement correctly categorised 33 patients (66%), incorrectly categorised 10 patients (20%) and was equivocal in seven patients (14%). Statistical analysis on table 2 (b) using the chi-square test indicated that the direction of tympanic membrane displacement was correlated with whether intracranial pressure was raised or normal ($p < 0{\cdot}005$).

Figure 2 shows the mean displacement, Vm, measured in patients with raised or normal intracranial pressure (dashed lines) in terms of volume displacement against stimulus intensity, with standard error bars. The average value of Vm in the raised intracranial pressure group is negative whereas the average value in the normal intracranial pressure group is positive from 100 to 115 dB SPL. Analyses of variance showed that the change in Vm with shunting in the 12 patients (solid lines) is highly significant ($p < 0{\cdot}001$). This is indicative of a marked decrease in pressure. In two of the shunted patients marked changes were observed on the left but not on the right following operation. Postural tests suggested that the cochlear aqueduct was not patent on the right side in either patient.

Discussion

Tympanic membrane displacement depends upon a normal middle ear and an intact acoustic stapedial reflex. If intracranial pressure is to be monitored by this technique then the cochlear aqueduct has to be patent. The most consistent results were obtained in young patients with hydrocephalus and benign intracranial hypertension. In normal subjects the tympanic membrane displacement in a particular ear remains stable over many years and we are currently examining children with shunts over a long term. On one occasion when clinically it was unclear if the patient's deterioration was due to failure of the shunt or to intercurrent infection, the tympanic membrane displacement clearly suggested raised intracranial pressure and shunt dysfunction was confirmed at operation.

The least reliable results were obtained in the head injured patients who were on ventilators and in the elderly subjects. In the former group the combination of significant negative middle ear pressures and the use of muscle relaxants meant that acoustic reflexes could not be elicited. In the elderly it is more likely that the cochlear aqueduct is non-patent, in which case intracranial pressure is not reflected in the perilymphatic fluid pressure.[1]

The measurement technique cannot at present measure absolute intracranial pressure, although mean intracranial pressure can to a certain extent be classified as normal or raised. Research is underway to establish calibration procedures which would allow absolute measurements to be made. It is not suitable currently for continuous intracranial pressure monitoring nor for the detection of waves of pressure.

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