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The sono-ocular test

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ERASMUS Darwin in his Zoonomia (1794) referred to the vertigo induced in certain patients by the sound of bells and waterfalls. This phenomenon later came to be known as the Tullio phenomenon after the work of Tullio (1929), who showed in the pigeon that the ampulla of a semicircular canal can be excited by an acoustical stimulus. This response is increased if the bony canal is fenestrated, and persists after destruction of the cochlea. Following this pioneering physiological work of Tullio and of Huizinga (1935), the phenomenon was rediscovered in man by Benjamins (1938) in a patient with a cholesteatoma and a fistula in the horizontal semicircular canal.

Since that time the phenomenon became more widely found with the advent of the two-stage operation for labyrinthine vertigo, and the idea became prevalent that a further mobile window in the vestibular side of the vestibular membrane was necessary for such a reaction to occur. More recently, however, Land (1957), Chadwick (1966), and Kacker and Hinchcliffe (1970) have reported Tullio's phenomenon in patients with sensorineural hearing loss, although only in the case of the last-named authors were detailed investigations made of the patients. Gerlings and Kleyn (1942) had referred to patients with congenital syphilis reported by other authors who appear to have shown this phenomenon. Kacker and Hinchcliffe (1970) also coined the term 'the sono-ocular test' for a presentation of acoustical stimuli during electronystagmographic recording with the patient's eyes closed.

It has been observed that noise of sufficiently high intensity will induce nystagmus even in normally-hearing people (Dickson and Chadwick, 1951) and recently Evans *et al.* (1971) have proposed high-intensity low-frequency sound stimulation as a method of testing vestibular function. Other tests

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have been proposed in which nystagmus has been induced by a moving sound field (Hennebert, 1960; Watson, 1968).

The present paper concerns itself with a clinical application of the technique proposed by Kacker and Hinchcliffe (1970) and which is now currently used in the audiology clinic of the Institute of Sound and Vibration Research, Southampton. Some results will be presented for a group of normally-hearing subjects, four patients with Tullio's phenomenon, and a series of patients with vestibular disorders, in some of whom spontaneous nystagmus was enhanced by acoustic stimulation.

Method

Testing of the patients and normal subjects was conducted in the following manner. The patient was seated lengthwise on a couch in an electrically insulated room which was used for the other electronystagmographic and positional tests performed in the department. The backrest was elevated to an angle of 90° with the rest of the couch.

Surface electrodes were positioned for normal electronystagmographic recording of horizontal nystagmus using an Elema Mingograph 24B ENG machine.

Initially for the purpose of the test a short recording of twenty seconds was performed with patient positioned to be looking ahead, but with his eyes closed, and any spontaneous nystagmus was noted. TDH39 headphones with MX41/AR cushions were then fitted and tones presented from a Peters SPD5 audiometer to one ear. Initially a 1,000 Hz tone at 110 dB hearing level (ISO) was presented for five seconds. If there was no nystagmus induced, following rest of about ten to twenty seconds, five seconds of 500 Hz tone at 110 dB HL was presented, then 250 Hz at 95 dB HL, 2,000 Hz at 110 dB HL and 4,000 Hz at 110 dB HL. This was then repeated on the other ear. If the patient found these levels unpleasantly loud he was presented with the highest intensities which he could tolerate.

A positive response was said to have been obtained if nystagmus was induced where none was present previously or if there was a significant increase in the spontaneous nystagmus. Following such a response, further stimuli were withheld until it had completely subsided. Stimuli of the same frequency were then presented at progressively 10 dB lower levels until no response was obtained, in order to measure the threshold for the response.

In the case of the first twenty patients tested, testing was performed using a continuous test tone. This group included three of the four patients to be described in greater detail below.

In the subsequent forty patients tested pulsed test tones, pulsing at 2 pps were used following a report that normal subjects are more sensitive to the induction of nystagmus by high-intensity acoustical stimuli if they are pulsed.

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The sono-ocular test

Normally-hearing subjects

An experimental group of ten normally-hearing subjects with no history of hearing loss or vertigo was tested using both pulsed and continuous test stimuli. This group consisted of five males and five females. The pulsed stimuli were presented before the continuous stimuli in half the cases, in the other half the order was reversed. In all cases both ears were tested before the nature of the stimulus was changed.

In two of these subjects there was some minimal spontaneous nystagmus in the resting position with no acoustical stimulation. This was not increased by the stimulation. In the other eight subjects the acoustic stimulation did not induce any nystagmus.

It may be concluded from this very limited normative study that the stimuli used do not induce or potentiate nystagmus in normal subjects.

Patients with a positive Tullio-type response

Case 1: A fifteen-year-old schoolboy (R.H.) was referred to the audiology clinic of the Institute of Sound and Vibration Research on 26 January 1972 with a history of congenital bilateral severe sensorineural hearing loss, and more recently of attacks of vertigo while involved in his hobby of campanology.

The bell-ringing in his church was of the more modern type in which small ropes directly ahead of the player were pulled, rather than the more traditional type involving long ropes coming directly down from the bell tower. This playing therefore did not involve neck extension.

Attacks of swaying vertigo generally come on towards the end of a session of bell ringing. The patient wore his postaural hearing aid for these sessions in order to hear the instructions given by the group leader.

The aetiology of his hearing loss had not been completely established and it dated from birth or his early years. Of relevance perhaps is the fact that he was born one month prematurely and was jaundiced postnatally.

On examination of the patient, the appearance and mobility of his tympanic membranes were normal. The Weber test at 512 Hz was not referred and Rinne and Bing tests were positive in both ears. He wore a post-aural hearing aid and heard speech well by means of this prosthesis. No abnormalities were found in his cranial nerves.

His air conduction audiogram is shown in Figure 1. Bone conduction testing revealed no air-bone gap. In fact his audiogram was identical to audiograms obtained previously in 1966 and 1969. Sweep-frequency Békésy audiometry showed a Jerger type II pattern with the pulsed threshold similar to that obtained by manual testing. The thresholds of uncomfortable loudness are shown on the audiogram.

Speech audiometry showed the half-peak levels to be elevated by 30 dB in both ears with optimal discrimination scores of 71 per cent on the right and 97 per cent on the left.

Most comfortable loudness-level testing on the Békésy audiometer gave results compatible with end-organ pathology but elevated acoustic reflex thresholds were obtained on testing with the Madsen electroacoustic impedance meter.

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Electronystagmographic testing showed no spontaneous nystagmus present with eyes open in the light, but 1° nystagmus to the right with eyes closed or eyes open in the dark. Neck position testing accentuated the nystagmus to the right, as did positional testing using the Cawthorne technique. Optokinetic and pendulum testing gave normal results. Caloric testing with eyes open in the darkness and ENG recording showed normal results with no significant canal paresis or directional preponderance and a normal visual fixation index.

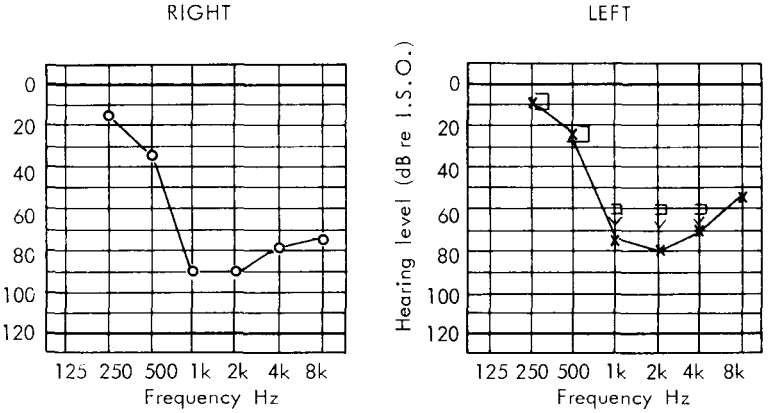


FIG. 1.
Audiogram of R.H. 26 January 1972.

Sono-ocular testing showed nystagmus induced by acoustic stimulation at 95 dB HL at 250 Hz, and 100 dB HL at 500, 1,000, 2,000 and 4,000 Hz in the left ear (Fig. 2). There was some response to the offset of a 500 HL stimulus at 110 dB in the right ear.

Re-testing three months later he showed a less-marked response and he had given up his bell ringing. However, positive sono-ocular responses were obtained on this occasion to 95 dB HL stimulation of this right ear at 250 Hz, and 110 dB at 4,000 Hz.

His personality as revealed by the Eysenck personality inventory was of a normal ambivert. (N = 12, E = 8, L = 1.)

There was some family history of hearing loss in that his paternal great-uncle and great-aunt had hearing losses, and in an attempt to establish an element of genetic carriers the parents were tested. The patient's mother had normal hearing in both ears on Békésy audiometry, although there was evidence of old otitis media in her right ear. Normal acoustic reflexes were obtained with the impedance probe in her left ear but neither acoustic nor non-acoustic reflexes were obtained with the probe in the right. The patient's father had a mild bilateral high-frequency sensorineural hearing loss falling off above 2 kHz to 25 dB at 4,000 Hz and 48 dB HL at 8,000 Hz. There was no obvious cause in his history for his hearing loss. He had normal acoustic reflexes. There would thus appear to be little in the parents in line with Anderson and Wedenberg's (1968) suggestions

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of the characteristics of carriers of recessive disorder apart from the father's high-frequency hearing loss.

It would thus appear that he may well have had some hereditary predisposition to hearing loss and this was aggravated by his neonatal jaundice.

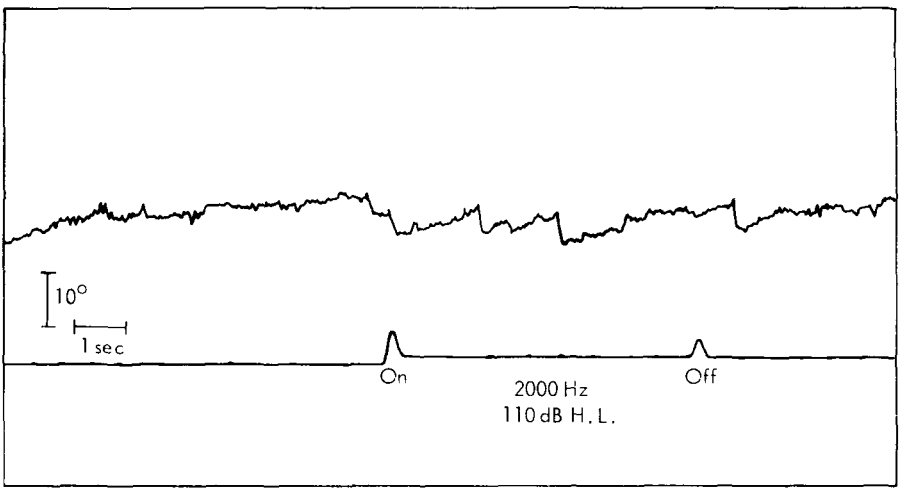


FIG. 2.
ENG response of R.H. to acoustical stimulation by 2,000 Hz tone 110 dB HL.
26 January 1972.

Case 2: A fifty-nine-year-old planning engineer (H.D.) was referred suffering from recurrent Ménière's disorder. He had previously had a right labyrinthine destruction (1957) for persistent Ménière's disease. This had relieved his vertigo until three months before he was seen in February 1972. In those three months he had suffered eight attacks of swaying vertigo.

He had had a complete right-sided hearing loss since his labyrinthine destruction in 1957 and had poor hearing in his left ear.

On examination his right tympanic membrane was scarred with a small dry anterior perforation, and his left tympanic membrane was somewhat scarred with reduced mobility. The Weber was not referred, and, in the left, the Rinne test was positive and the Bing test negative. The only incidental abnormal finding in the cranial nerves was some reduced sensitivity to pinprick in his right tragus. Electrogustometric thresholds were 54 μA on the right, 58 μA on the left.

Personality testing with the Eysenck personality inventory revealed a normal ambivert but with a high lie score (N = 10, E = 13, L = 7), not an unusual finding in Ménière's disorder. WR, VDRL and Reiter protein CFT were negative.

His audiogram is shown in Figure 3. Of note are the thresholds in his right ear marked J—'Jump' threshold, at which he was observed to respond as though being presented with a noxious stimulus.

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Further testing of his left ear showed most comfortable loudness levels and thresholds of uncomfortable loudness indicative of a marked degree of recruitment. His optimal discrimination score for speech was 94 per cent.

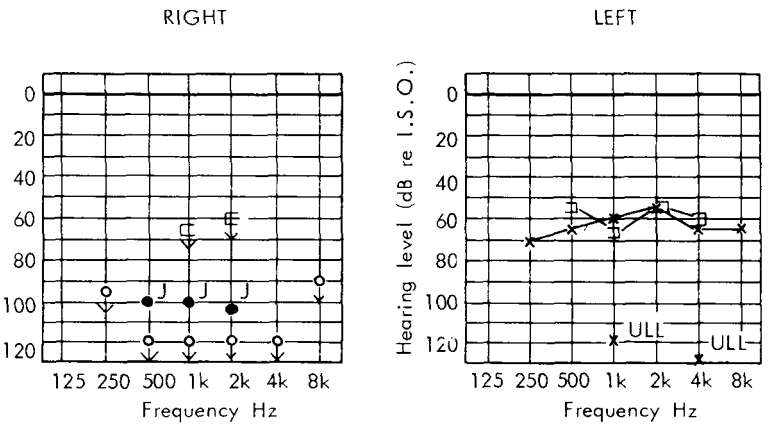


FIG. 3.
Audiogram of H.D. 9 February 1972.

Electronystagmography showed no spontaneous nystagmus with his eyes open in the light, in the darkness or closed. Positional testing with the Cawthorne technique revealed nystagmus to the left in all three positions of a peripheral type. Neck position testing showed a nystagmus to the left when his head was turned to the right with his eyes closed. Pendulum and optokinetic testing gave normal results. Caloric response in the left ear was normal. Cold air caloric testing of the right ear produced a marked nystagmus to the left of immediate onset.

Sono-ocular testing of the right ear induced nystagmus at 500 Hz with a 110 dB HL tone, 1,000 Hz (100 dB) and 2,000 Hz (110 dB). No nystagmus was induced on stimulating the left ear.

It was concluded that the labyrinthine destruction performed in 1957 had not been complete and that a mobile window remained on the vestibular side of the vestibular membrane, together with a number of intact sensory receptor cells.

Case 3: A seventy-nine-year-old retired army officer (H.W.) was referred with an eighteen-month history of attacks of swaying vertigo, left-sided hearing loss, and tinnitus. He complained of an intermittent hearing loss in the right ear.

He gave a history of considerable exposure to gunfire during World War I and of temporary hearing loss arising between the wars from large doses of quinine

On examination his right tympanic membrane was scarred with hypermobile patches, his left tympanic membrane was retracted and scarred. The Weber test at 512 Hz was referred to the right, the Rinne test was positive in his right ear but not heard on the left. No abnormality was detected in his other cranial

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nerves and his electrogustometric thresholds were 80 μA (right) and 75 μA (left). Personality testing with the Eysenck personality inventory high-extraversion and lie scores (N = 4, E = 16, L = 6). His WR, VDRL, Reiter protein CFT were all negative.

His audiogram is shown in Figure 4. The right–left threshold difference at 500 Hz was supported by the results of Stenger testing. He had Jerger type II sweep frequency, Békésy audiograms in both ears and the Owens 'tone decay' test showed a moderate degree of 'decay' at 500, 2,000 and 4,000 Hz in the left ear. Further testing with the modified Carhart test showed rapid 'decay' at 250, 1,000 and 4,000 Hz in the left ear but in the right ear no 'decay' at 250 and 1,000 Hz, and 30 dB 'decay' at 4,000 Hz. SISI testing of the left ear at 110 dB HL showed a 50 per cent discrimination score. Speech testing revealed an optimal discrimination score of 20 per cent in the left ear and 59 per cent in the right.

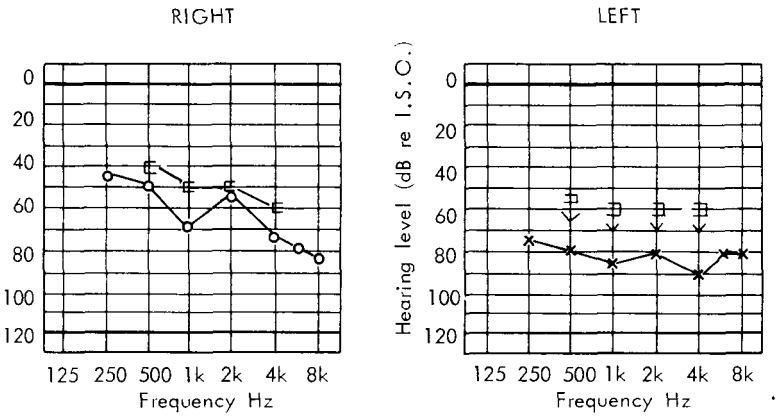


FIG. 4.
Audiogram of H.W. 7 March 1972.

Electro-acoustic impedance testing revealed normal middle-ear pressures with normal compliance on the left and high compliance on the right. Acoustic and non-acoustic reflexes were absent.

No spontaneous nystagmus was present on electronystagmographic recording with eyes open in the light, in the darkness nor with eyes closed. None was induced by the Cawthorne positional tests, nor on neck-position testing. The pendulum test showed normal results as did the optokinetic test, although drum rotation from right to left was followed by some afternystagmus to the right in the darkness. Caloric irrigation failed to elicit any response from the left ear at 30 °C and 44 °C and irrigation at 25 °C produced a response of 3 °/sec. (maximum velocity of slow phase) and at 20 °C a response of 4 °/sec. Irrigation of the right ear, too, produced only small responses (7 °/sec. at 44 °C, 6 °/sec. at 30 °C).

Sono-ocular stimulation of the right ear produced no nystagmus but stimulation of the left ear at 95 dB (HL) 250 Hz, 110 dB at 500, 1,000, 2,000 and 4,000 Hz

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all induced nystagmus to the left. On repeated testing six weeks later nystagmus to the left was induced only by stimulation with a 500 Hz tone at 110 dB HL.

As the patient was suffering from generalized Paget's disease polytomography of his temporal bones was performed, but this revealed no direct evidence of Paget's disease there. This does not however exclude this as a diagnostic possibility.

Case 4: A girl (K.W.) aged four years, who had a normal birth with no prenatal or perinatal complications and showed normal development until the age of eighteen months. She then became generally ataxic and was investigated neurologically and a diagnosis of acute cerebellitis was made. She recovered well and continued to develop normally.

At the age of three-and-a-half her speech development stopped and her mother wondered whether or not she was deaf. She was investigated audiologically and considered to have a complete right-sided sensorineural hearing loss. Over the next three months it was noticed that the hearing in her left ear was deteriorating and at the time of referral was severe. X-rays of her internal acoustic meatuses showed bilateral dilatations at the canalicular ends. The deterioration of her hearing was not associated with ataxia nor with nystagmus.

On examination her tympanic membranes appeared normal. Her speech was generally that of a deaf child, although certain words were very clear.

Impedance testing showed normal middle-ear pressures and compliance curves with definite responses on both sides to aural puff stimulation. No acoustic reflexes could be elicited. On speech testing she seemed happy with words at 90 dB in the left ear but was unable to repeat them, but she found speech at 110 dB was uncomfortable. She showed no definite reaction to speech in the right ear.

Evoked response audiometry was difficult due to the patient's restlessness. On stimulation of the right ear, no response was obtained at 500 Hz to the limits of the equipment (100 dB HL). A definite response was obtained from the left ear at 80 dB, but repeated testing failed to show any response at 70 dB.

Electrocochleography showed a small short latency response to 100 dB click stimulation from the left ear but no response from the right ear.

On electronystagmographic testing she had no spontaneous nystagmus with her eyes open in the light nor in the darkness. She showed normal responses to the pendulum and optokinetic tests, but would not tolerate caloric testing.

Sono-ocular testing with eyes open in the dark elicited a nystagmus to the left when stimulated with 110 dB (HL) tones at 1,000 and 2,000 Hz in the left ear. Stimulation of the right ear induced no nystagmus.

Testing of her parents was performed although there was no family history of hearing loss. The father was normally-hearing but with a small dip to 20 dB bilaterally at 2,000 Hz on sweep-frequency Békésy audiometry. He had normal acoustic reflexes. The mother had normal hearing but with dips bilaterally at 5,000 Hz to 30 dB in the left and 20 dB in the right ear. She also had normal acoustic reflexes.

In conclusion she was considered probably to have a predominantly sensory lesion in her left ear, experiencing discomfort from the speech material at 110 dB, and with a small amplitude but short latency response on electrocochleography. In the subsequent year she has been managed conservatively and appears to be

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making good progress in a school for the partially hearing. No definite aetiology for her hearing loss has been established.

Other vertiginous patients tested

Over sixty patients have been tested with the sono-ocular test in the general clinical work of the department. Many of these patients were referred for investigation of their vertigo, but a number had no veriginous symptoms and were being tested as part of our routine investigation of patients with sensorineural hearing loss. Of these patients, forty-seven completed the Eysenck personality inventory (Eysenck and Eysenck, 1964) and twenty-six the Middlesex Hospital questionnaire (Crown and Crisp, 1966). These are routinely completed by patients in this department as part of an ongoing study of hearing and personality, and are of relevance in this case in view of the suggestion of Mitchell (1957) that a psychological factor may play a part in the Tullio phenomenon.

Table I shows the breakdown of the patients. All of these patients were found to have at least a minimal amount of spontaneous nystagmus in the course of electronystagmographic testing. Of these sixty patients, fifteen were found to show an enhancement of their spontaneous nystagmus when tested with the sono-ocular test at one or more frequency. Of these, seven were patients diagnosed as Ménière's disorder, four with labyrinthitis, two with sudden deafness, one with mumps and one with an 'acoustic neuroma'.

TABLE I.

PATIENTS TESTED WITH THE SONO-OCULAR TEST

Conductive hearing loss	
(a) Otitis media	1
(b) Otosclerosis	4
End-organ disorders	
(a) Noise-induced hearing loss	1
(b) Mumps	2
(c) Ménière's Disorder	25
(d) Labyrinthitis	15
VIII nerve lesions	3
Brainstem lesions	
(a) Brainstem 'bends'	1
(b) Syringomyelia	1
Miscellaneous	
(a) Head trauma	3
(b) Sudden deafness	3
(c) Tinnitus	1

In order to test Mitchell's (1957) idea that Tullio's phenomenon may be related to psychological factors in these patients, the personalities of those

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showing enhancement on acoustical stimulation was compared with those showing no enhancement. On none of the nine scales tested (extraversion, neuroticism and lie scales of Eysenck personality inventory, and anxiety, phobic anxiety, obsessionality, somatic preoccupation, depression and hysteria scales of the Middlesex Hospital questionnaire) did the two groups of patients differ significantly. In order to overcome the criticism that this could be related to slightly d.fferent proportions of patients with different diagnoses within the two groups, the same comparison was made with the patients diagnosed as suffering from Ménière's disorder, and again the two groups were not significantly different.

Discussion

The sono-ocular test described by Kacker and Hinchcliffe (1970) has proved useful in provoking nystagmus in four patients in whom little or none was otherwise present. Although one of the patients (H.D.) had had a simple cortical labyrinthectomy which has been reported as being occasionally followed by Tullio's phenomenon, in the other three cases the aetiology is less usual. In the case of the young child (K.W.) the diagnosis is too uncertain to contribute further to the discussion, but the other two cases are more interesting.

In the case of R.H., who has what would appear to be a stable congenital sensory hearing loss, the phenomenon developed only when he was exposed to high noise intensities when wearing a hearing aid while participating in bell-ringing. That this high-intensity exposure provoked some sensitization of a congenitally-abnormal labyrinthine system is a possibility, and moderate levels of stimulation were subsequently able to provoke this vestibular response. Following medical advice he forewent his campanological hobby and has subsequently been clear of vertiginous attacks even with fairly intense acoustical stimulation.

Davies (1968) reports depressed caloric response as being unusual in Paget's disease, with the labyrinthine capsules being least sensitive to involvement in the bony changes of this disorder. He also put forward some evidence supporting earlier work that the auditory lesion is principally conductive with an end-organ component. H.W. also showed such a mixed picture but with some suggestion of neural involvement. Again there is no obvious cause which could be suggested for his positive sono-ocular response other than a possible hyper-excitability of the vestibular labyrinth by early involvement by the disease process.

This hyper-excitability concept is supported by the fact that 25 per cent of the patients tested who had some spontaneous nystagmus recordable by electronystagmography also responded with an enhancement of this spontaneous nystagmus by the acoustical stimulation. This may be regarded as a lowering of the response threshold of the vestibular labyrinth to acoustical

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stimuli. In all healthy individuals if the stimulus is sufficiently intense such a response can be obtained although this will normally require stimuli exceeding the person's pain threshold. The present study was unable to support the premise that this vestibular threshold to acoustical stimulation was related to the personality of the patient.

Summary

The sono-ocular test of Kacker and Hinchcliffe (1970) has been used to elicit nystagmus in four patients with Tullio's phenomenon, and to enhance the nystagmus of fifteen patients with vestibular abnormalities. The test used entails electronystagmographic recording with the patient's eyes closed and acoustical stimulation with pulsed tones of different frequencies. The personality of the patient was not found to influence the response.

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