The Journal of Laryngology and Otology
September 1993, Vol. **107**, pp. 780–786

Cortical evoked potential criteria in the objective assessment of auditory threshold: a comparison of noise induced hearing loss with Ménière's disease

DEEPAK PRASHER, MOHAMED MULA, LINDA LUXON* (London)

Abstract

Amplitude of the N1 component of the cortical response was used to objectively determine threshold of hearing at 1 kHz and 4 kHz in a series of consecutively referred medicolegal cases with alleged occupational noise induced hearing loss and a control group of patients with Ménière's disease who were not seeking compensation for their hearing loss. The cortical response thresholds were compared with the subjective pure tone audiometric (PTA) thresholds at the same frequencies. The cortical and PTA thresholds were 'within 10 dB' for 84 and 92 per cent of the cases of noise induced hearing loss (NIHL) and Ménière's disease respectively, confirming the validity of CERA as a means of defining accurately the frequency specific thresholds and the audiometric configuration. Of the remaining 16 per cent of NIHL, 13 per cent exaggerated their PTA thresholds at 1 kHz and 10 per cent at 4 kHz whilst the error in cortical threshold estimation was beyond the 10 dB level for three and six per cent of cases at those frequencies respectively. The median exaggeration of threshold was 25 dB. For eight per cent of the Ménière's patients, thresholds exceeded 10 dB at both 1 and 4 kHz, four per cent of whom exaggerated their PTA thresholds and four per cent had a test error greater than 10 dB. A similar percentage (four per cent) of both groups revealed a cortical test error greater than 10 dB whereas three times as many cases of noise induced hearing loss (13 per cent) revealed exaggeration of their subjective audiometric thresholds compared with the Ménière's group (four per cent).

The value of latency criteria in the assessment of cortical threshold was established. The latency of N1 close to threshold was invariably greater than 150 ms and at 30 dB above threshold, irrespective of absolute levels, it was within 10 ms of 100 ms, thereby providing another criterion for the improved measurement of threshold.

Key words: Hearing loss, noise induced, occupational; Evoked potentials, auditory; Legal medicine

Introduction

It is well recognized that workers exposed to hazardous levels of noise (>85 dB) develop a loss of hearing sensitivity. Although standard pure tone audiometry (PTA) provides a subjective assessment of the degree and configuration of hearing impairment, any exaggeration, either intentionally or subconsciously, may preclude accurate identification of the true extent of the deficit. It has been suggested (Gleason, 1958) that as many as 30 per cent of claimants deliberately conceal the true threshold of hearing.

Compensation for occupational noise induced impairment of hearing was considered in the UK as early as 1907 and disablement benefit became payable in 1975 for deafness due to noise at work. The range of occupations at risk of developing noise induced hearing loss (NIHL) was extended in 1980, leading to an explosion in the number of compensation claims. Although several special audiometric procedures have been designed to reveal the presence of a non-organic component of a loss (Martin, 1972; Coles, 1982), a rapid objective and validated assessment has not been established and incorporated into routine practice.

Objective verification of the audiogram has been sought for some time and evoked potential recordings offer several responses which may fulfill this role (Picton *et al.* 1977). Although brain stem potentials can be useful in the estimation of threshold (van der Drift *et al.* 1987) in certain groups such as children who may be difficult to test, they do not provide frequency specific thresholds which can be directly compared with the standard audiometric evaluation (Stappels *et al.* 1985; Starr and Don, 1988) and may miss islands of low frequency hearing (Picton and Durieux-Smith, 1988).

Beagley and Kellogg (1968) first compared the vertex response and subjective auditory thresholds. Later, several studies (Keidel, 1976; Jones *et al.* 1980; Alberti, 1981; Coles and Mason, 1984; Hyde *et al.* 1986) showed that thresholds obtained by cortical evoked audiometry were similar to those obtained by pure tone audiometry,

From the Medical Research Council, Section of Neuro-otology, Human Movement and Balance Unit, and the Department of Neuro-otology,* National Hospital for Neurology and Neurosurgery, London.
Accepted for publication: 8 March 1993.

although others (Rose *et al.* 1971) disputed the extent of agreement between the two test procedures. Despite this, it is generally agreed that N1 of the late cortical response is best suited to assessing sensitivity at specific frequencies and providing objective evaluation of the configuration and severity of hearing impairment at each of the frequencies routinely assessed.

If equitable assessments of disability are to be established and used as the basis for compensation then reliable and reproducible documentation of auditory function is obligatory as the current compensation ratings are based on averages across a range of frequencies and even minor inaccuracies in threshold values can result in significant differences in compensation levels.

Therefore, the aims of this study were threefold:

(a) To validate the sensitivity and reliability of the N1 cortical response as a means of defining auditory thresholds by a comparison with the PTA in cases of alleged NIHL and in a group of patients with Ménière's disease, who had no reason to give unreliable test results.

(b) To evaluate the cortical response and establish parameters of value in identifying accurately the threshold value.

(c) To devise a rapid objective means of verifying pure tone audiometry which could be incorporated into routine clinical assessment of NIHL.

Cases

Sixty-two males who had worked in a broad spectrum of industries including railways, mining, car manufacturing, printing and paper mills and who had been exposed to an equivalent continuous noise level of more than 85 dB (A) for a minimum of five years and up to 50 years were assessed.

Twenty-seven patients with Ménière's disease were also examined. The mean age and range of the two groups are given in Table I.

Methods

Pure tone audiometric (PTA) thresholds were measured in all subjects with a Madsen OB802 clinical audiometer using the standard technique for pure tone audiometry including 3 kHz and 6 kHz (British Society of Audiology, 1981). Tympanometry and acoustic reflex thresholds were measured with a GrayStad (GSI33) impedance bridge.

Cortical potentials were recorded and analysed blind to the audiometric evaluation. The subject sat in a comfortable chair in a single-walled Industrial Acoustics Company (IAC) sound attenuated chamber, listening to the tone bursts of 200 ms duration, with a rise and fall time of 10 ms presented monaurally through TDH 49 earphones at a rate of one every second. To reduce test time, only responses at 1 and 4 kHz were obtained. These frequencies were selected as in both the cases of Ménière's disease and NIHL, they represented frequency ranges where maximal and minimal auditory loss would be expected.

If the responses confirmed the subjective thresholds at these two frequencies it seemed unlikely that subjective thresholds at other frequencies would be inaccurate. The responses were recorded beginning with a stimulus intensity of 90 dB HL and adjusting the intensity in 10 dB steps for subsequent recordings according to the response elicited. If no response, which was based on the N1–P2 amplitude being greater than any other peak within the first 200 ms of the response window, was judged to be present after a repeat run, the intensity was increased by 5 dB. Threshold was taken as the lowest intensity at which a response was present. The contralateral ear was masked with white noise 30 dB below the ipsilateral stimulus intensity. The electrical activity was recorded from Cz with respect to A1 and A2 with the contralateral (with respect to the stimulated ear) mastoid acting as ground and inter-electrode impedance less than 2 kohms. This activity was filtered with a bandpass of 0.1 to 30 Hz. An analysis window of 500 ms was used with cursor resolution of 1 ms. Latency of the response was defined as the interval between the stimulus presentation and the N1–P2 peak.

Threshold estimates were analysed and compared using SPSS (Statistical Package for Social Sciences).

TABLE I

Group	Number	Mean age (SD) (years)	Age range (years)
NIHL	62	55 (10)	34–78
Ménière's disease	27	59 (10)	39–73

Results

The range of hearing assessed (Figure 1) was very similar for both the cases with noise induced hearing loss (NIHL) and patients with Ménière's disease.

A comparison of the mean thresholds at the two frequencies (1 and 4 kHz) studied showed no significant difference (Table II) between the cortical and PTA thresholds. Although the mean thresholds at 1 kHz were significantly different between the two groups (27.8 dB for NIHL and 48.6 dB for Ménière's disease), the cortical response thresholds matched closely the PTA thresholds. The mean discrepancy between the two threshold estimations for the NIHL group was 0.34 and −0.56 for 1 and 4 kHz respectively. The mean difference in the thresholds for the Ménière's group was −2.3 and −1.3 for 1 and 4 kHz respectively.

The mean hearing at 4 kHz was similarly affected for both groups and again showed no significant difference in the mean thresholds obtained with PTA or cortical responses.

The scatter was examined further by computing the correlation between cortical and PTA thresholds in NIHL group which was 0.79 at 1 kHz and 0.89 at 4 kHz. Furthermore the regression analysis shows that a linear regression line can be used to summarize the relationship and the statistic r^2 accounts for 62 and 81 per cent of the variance in the data for the NIHL group for 1 and 4 kHz respectively. A scatter plot with the regression line for the NIHL group is shown in Figure 2a and a similar scatter plot of the thresholds with the regression line for the Ménière's group is shown in Figure 2b. The correlation between cortical and PTA thresholds for the Ménière's group was 0.94 at 1 kHz and 0.89 at 4 kHz with the coefficient of determination r^2 accounting for 89 and 80 per cent of the variance in the data respectively for each frequency. These measurements clearly indicate that a closer relationship between the cortical and PTA thresholds is

https://doi.org/10.1017/S0022215100124429 Published online by Cambridge University Press

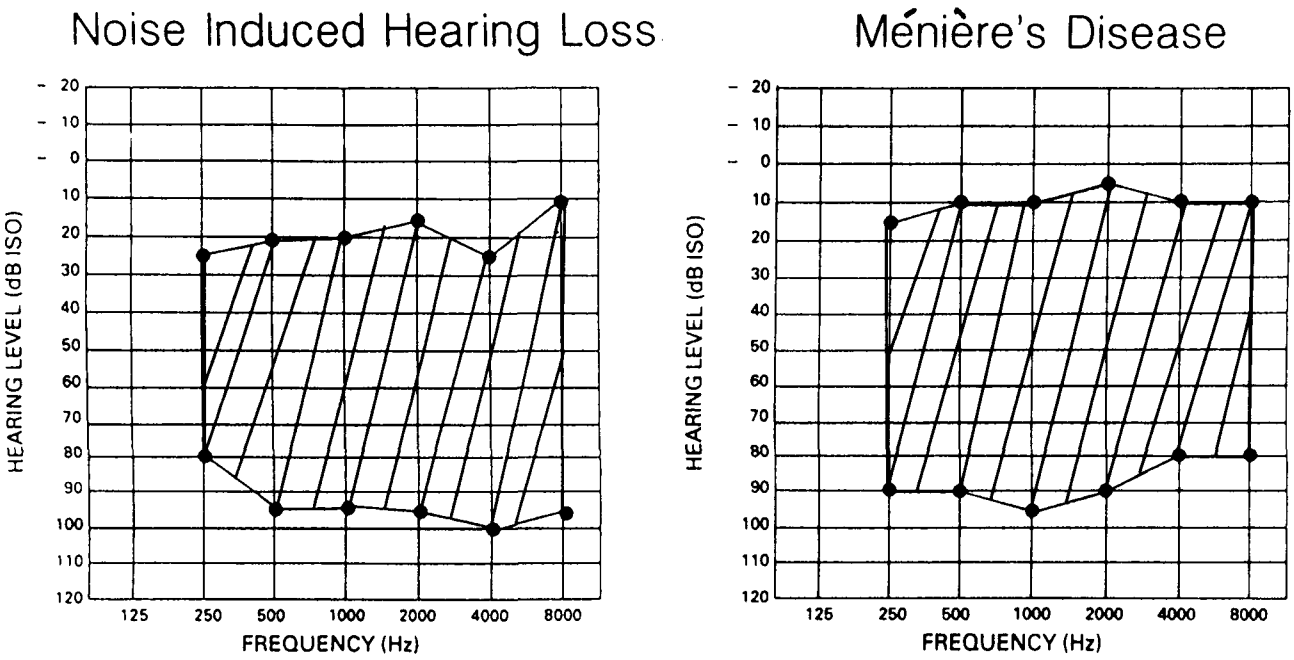

FIG. 1

Range of hearing for cases tested who have noise induced hearing loss or Ménière's disease.

achieved at 1 kHz for the Ménière's group and at 4 kHz for the NIHL group. The regression equations for the relationship between the two methods of threshold determination are shown in Table III for the two groups for both 1 and 4 kHz. In estimating the pure tone threshold from the cortical response threshold the equations for the NIHL group show that, for both frequencies, the predicted PTA thresholds are almost the same as the cortical thresholds. However, for the converse, the estimation of the PTA from the cortical thresholds, the values are consistently predicted to be lower particularly at 1 kHz, indicating a greater exaggeration at this frequency. For the Ménière's group, the predictions for either method based on the other indicate thresholds within 5 dB for both 1 and 4 kHz.

Although the numbers in the Ménière's group are small, there is a tendency for greater scatter in the frequency less affected by the pathology in each group, namely 1 kHz for the NIHL group and 4 kHz for the Ménière's disease group. There appears to be a greater correspondence between cortical and PTA thresholds for the more affected frequencies. This may be due to less exaggeration of PTA thresholds at the affected frequencies. The number of ears showing an exact correspondence between cortical and PTA thresholds at 1 kHz (Table IV) is 24 and 38 per cent for NIHL and Ménière's respectively and at 4 kHz are 28 per cent for NIHL and 27 per cent for the Ménière's group. Thresholds lying within 5 dB are 70 and 85 per cent for 1 kHz and 65 and 67 per cent for 4 kHz respectively, and those within 10 dB amount to 84 per cent for NIHL and 92 per cent for the Ménière's group for both 1 and 4 kHz. Thus a total of 16 per cent of the ears have a greater discrepancy than 10 dB for NIHL and, of these, 13 per cent exaggerated their PTA thresholds for 1 kHz and 10 per cent for 4 kHz. The remaining three and six per cent accounted for the error in cortical threshold estimation beyond 10 dB. Of the remaining eight per cent of the Ménière's ears, only four per cent exaggerated their thresholds and four per cent accounted for the error beyond 10 dB.

TABLE II

COMPARISON OF MEAN CORTICAL RESPONSE AND PTA THRESHOLDS IN MÉNIÈRE'S DISEASE AND NIHL

	1 kHz	4 kHz	*t*-test
NIHL			
PTA	27.8 (17.2)	52.8 (22.1)	NS
Cortical	27.5 (13.6)	53.4 (20.7)	NS
PTA–Cortical	0.34 (10.5)	−0.56 (9.7)	
Ménière's disease			
PTA	48.6 (22.6)	57.9 (14.7)	NS
Cortical	50.9 (21.6)	59.2 (17.3)	NS
PTA–Cortical	−2.3 (7.65)	−1.3 (8.16)	

NS = not significant.

The cortical N1 responses for both 1 and 4 kHz are illustrated in Figure 3a for a case with NIHL and in Figure 3b for a patient with Ménière's disease. It can be clearly seen that with decreasing stimulus intensity the latency of the N1 peak increases particularly near the true threshold. The latency intensity function for the whole group was examined by calculating the mean latency of N1 across all subjects for stimulus intensities from 5 to 30 dB above threshold irrespective of absolute threshold. This mean latency of N1 is shown plotted against sensation level in Figure 4a for 1 kHz and Figure 4b for 4 kHz. It can be seen that at 30 dB above threshold irrespective of absolute levels the latency of N1 approaches 100 ms. Closer to threshold the latency of N1 is significantly prolonged and the mean level tends to be greater than 150 ms. The upper and lower 95 per cent confidence limits suggest that the latency measurement may be used as an additional criterion in the assessment of cortical threshold.

Discussion

It is clear from this study that verification of the subjective hearing thresholds can be achieved by using the

https://doi.org/10.1017/S0022215100124429 Published online by Cambridge University Press

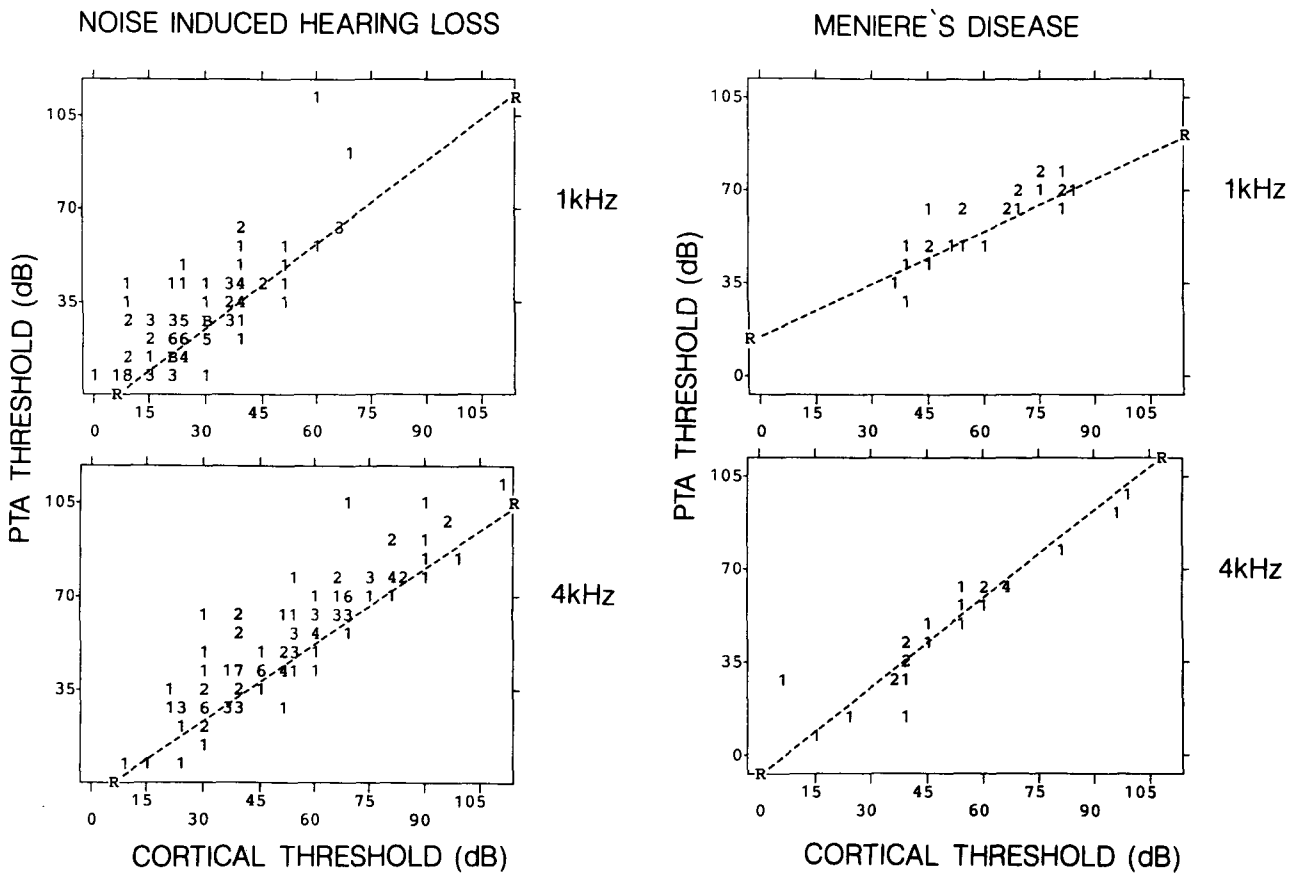

FIG. 2

A linear regression line summarizes the relationship between subjective and objective threshold measurements for: (a) NIHL and (b) Ménière's disease. Numerals and letters (A=10, *etc*) indicate the number of cases with a particular combination of cortical and subjective thresholds. The 'R' on the ordinate and abscissa indicate the regression line. A greater scatter is seen at 1 kHz for cases with NIHL and at 4 kHz for patients with Ménière's disease.

late cortical potential N1, which provides an objective frequency specific evaluation. The correspondence between the thresholds obtained by each method is very high with correlation values around 0.9. The 'within 10 dB' correspondence between the PTA and cortical thresholds was better at 92 per cent in those not seeking compensation (Ménière's group) compared to 84 per cent for the medicolegal cases with NIHL. The mean discrepancy between the two methods of threshold estimation for the NIHL group was close to zero. Similar mean differences in the subjective and objective responses were reported by Coles and Mason (1984).

The mean exaggeration of threshold in the NIHL group is shown to be greater at 1 kHz than at 4 kHz by 10 dB although the percentage of ears exaggerating threshold was very similar. This would imply a tendency to level subjectively the hearing loss across the frequency range such that the threshold at 1 kHz approaches that at 4 kHz. Equating loudness across the frequency range in this way leads to a raised threshold at 1 kHz as most cases have a worse threshold at 4 kHz than at 1 kHz. A greater scatter in the unaffected frequency of 4 kHz is also seen in patients with Ménière's disease suggesting that perhaps the attempt at bringing the threshold level up to that at the worse affected frequency is not deliberately executed by those with NIHL, but is a phenomenon common to all with sloping hearing loss.

TABLE III
REGRESSION EQUATIONS

NIHL
Regression equations for 1 kHz:
Cortical threshold (CT) = 10.0 + 0.6 PT
Pure tone threshold (PT) = 0.33 + 1.0 CT
Regression equations for 4 kHz:
Cortical threshold (CT) = 8.7 + 0.80 PT
Pure tone threshold (PT) = 1.8 + 0.96 CT
Ménière's disease
Regression equations for 1 kHz:
Cortical threshold (CT) = 6.7 + 0.91 PT
Pure tone threshold (PT) = −0.9 + 0.97 CT
Regression equation for 4 kHz:
Cortical threshold (CT) = 8.1 + 0.86 PT
Pure tone threshold (PT) = 17.2 + 0.70 CT

TABLE IV
COMPARISON OF PTA AND CORTICAL THRESHOLD: PERCENTAGE OF EARS WITHIN AN ACCURACY BAND

	Ménière's disease		NIHL	
	1 kHz	4 kHz	1 kHz	4 kHz
Exact	38	27	24	28
Within 5 dB	85	67	70	65
Within 10 dB	92	92	84	84
PTA > Cortical (>10 dB)	4	4	13	10
Cortical >PTA (>10 dB)	4	4	3	6

https://doi.org/10.1017/S0022215100124429 Published online by Cambridge University Press

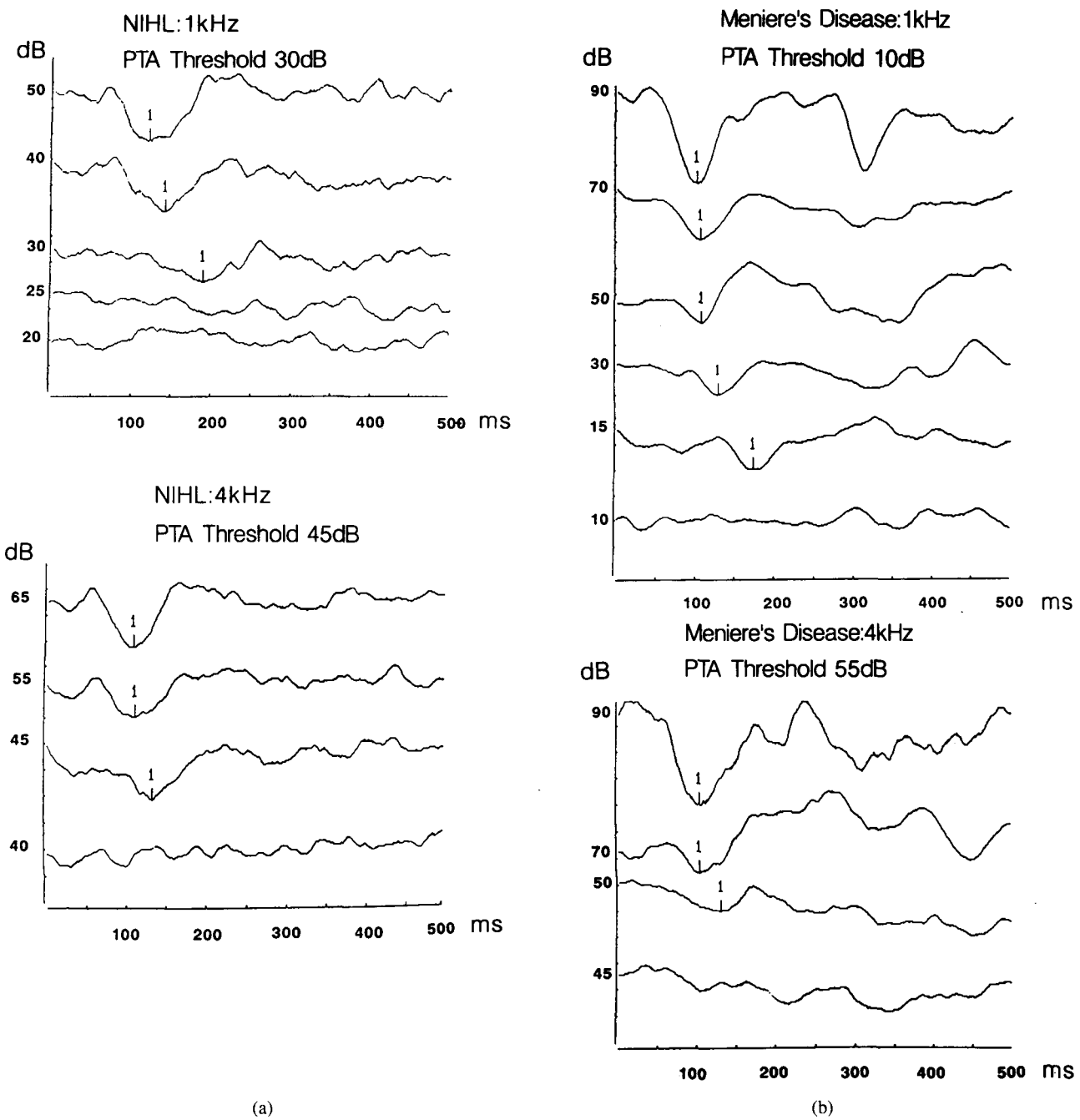

FIG. 3

Representative recordings of the cortical response N1 at 1 kHz and at 4 kHz for a case with: (a) NIHL and (b) Ménière's disease. Note the prolongation of latency with decreasing stimulus intensity.

Errors in cortical threshold estimation were similar in the two groups (three per cent for NIHL and four per cent for Ménière's disease) and occur primarily due to excessive rhythmic activity in the EEG obliterating the response upon averaging but other possible causes may include drowsiness, excessive muscle activity, and increased ambient noise level. The extent of exaggeration of threshold was significantly different in the two groups (13 per cent in NIHL and four per cent in Ménière's disease). The amount of overlay (25 dB) is small but significant in terms of compensation. Using a criterion of a discrepancy of 15 dB or more in either ear at 0.5 kHz, Hyde *et al.* (1986) reported an exaggeration of threshold in eight per cent of their cases. Our findings are in broad agreement with those of Jones *et al.* (1980) who reported 85 per cent of their 37 cases to be 'within 10 dB' at 1 kHz and 73 per cent at 4 kHz. They also observed greater distribution of threshold differences in cases compared to controls.

Although it has been suggested (Naatanen and Picton, 1987) that the cerebral processes underlying the small and broad N1 evoked near the threshold may not be the same as those underlying N1 at higher intensities, in this study no significant difference was observed in the error in cortical threshold estimation as a function of absolute threshold. The estimation of a more severe hearing loss conferred no particular advantage over that of a near normal threshold.

A significant finding of particular value in quantifying threshold is that the latency of N1 close to threshold

https://doi.org/10.1017/S0022215100124429 Published online by Cambridge University Press

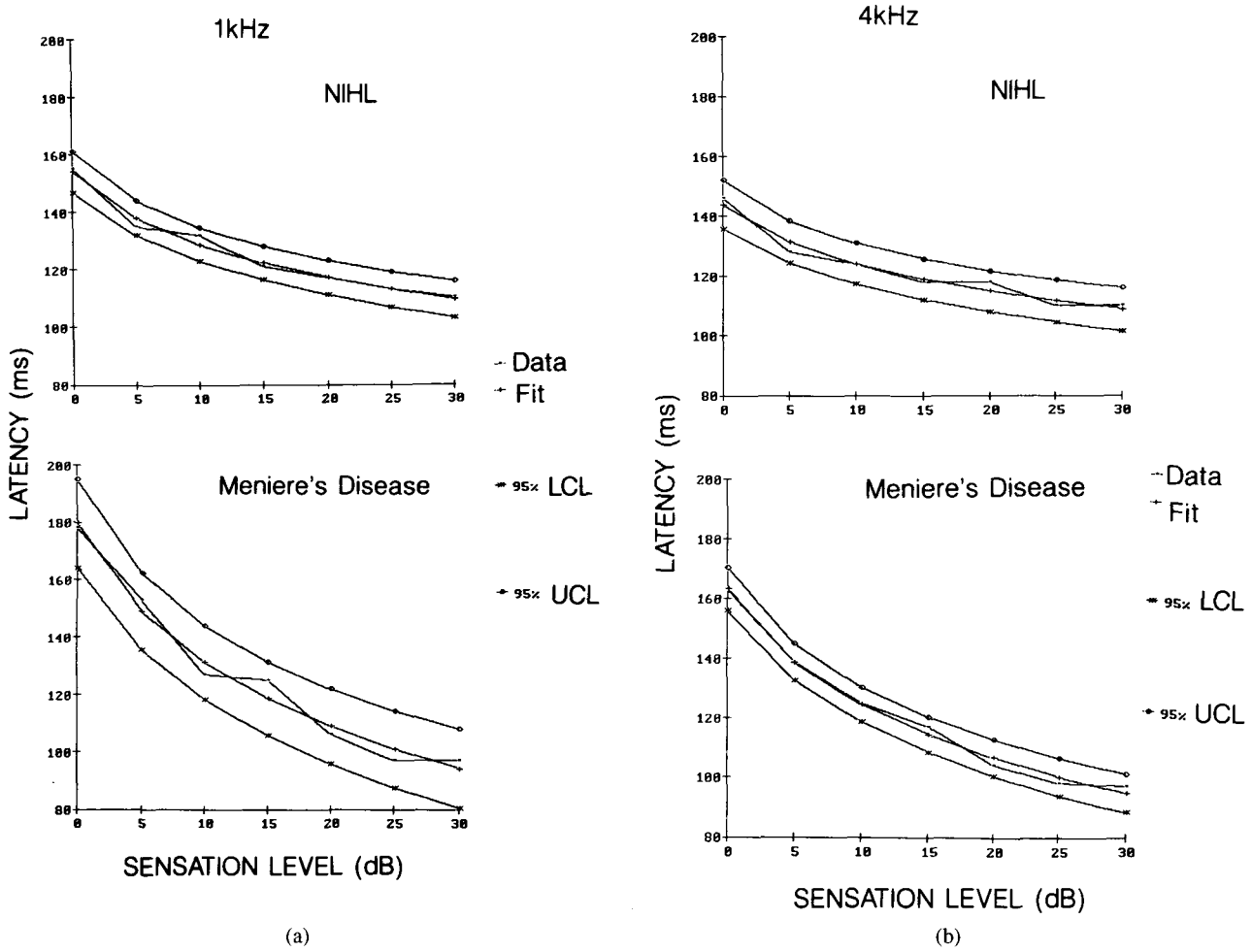

FIG. 4

A comparison of the latency/intensity function of N1. Mean latency is plotted against sensation level of stimulus across all cases comparing: (a) 1 kHz and (b) 4 kHz.

exceeds 150 ms. Thus a latency/intensity function should be used in conjunction with the amplitude criteria. Response latency increases exponentially from near 100 ms at 30 dB above threshold to over 150 ms at threshold. Thus, suspicion should be raised if, as a result of reducing the stimulus intensity, any response rapidly diminishes in amplitude without increasing in latency. This may suggeset that threshold has not been reached and other factors may be responsible for the disappearance of the response. Accurate prediction of threshold from a supra-threshold latency is not possible as the lowest latency limit is reached 30 dB above threshold and remains at that level with increasing intensity. However, a response latency of around 100 ms should indicate a threshold at least 30 dB below the eliciting stimulus intensity. Therefore a supra-threshold response can provide a fast indication of any discrepancy.

Our experience suggests that factors of importance in realizing a closer or better objective threshold than that obtained with pure tone audiometry are: (i) constant monitoring of the patient throughout the test procedure for any overt stimulus related or excessive movements of any kind, particularly blinking or chewing and (ii) ensuring that the patient stays alert during the procedure by asking them to count the number of tones heard as the amplitude of N1 is said to vary directly with the level of confidence of detection of the stimulus (Squires *et al.* 1975; Parsuraman *et al.* 1982). Such active participation avoids any tendency the patient may have for light sleep, which in turn adversely affects response amplitude. The accuracy of count also provides additional information regarding the true threshold level or its concealment.

This study indicates that if the threshold difference between the subjective and objective tests exceeds 10 dB then the test indicating a worse threshold should be repeated.

It is hoped that greater use of hearing protection and recent legislation to reduce levels of noise at work as far as practicable will lead to a reduction in the extent of noise induced hearing impairment. However, if objective verification of threshold is necessary, then the N1 component of the cortical response if appropriately obtained and interpreted, provides the best frequency specific measure of threshold in alert cases and the use of two frequencies provides a fast means of checking any discrepancy in objective thresholds with those obtained by subjective audiometry.

References

Alberti, P. W. (1981) Non-organic hearing loss in adults. In *Audiology and Audiological Medicine*. (Beagley, H. A., ed.), Oxford University Press, Oxford, pp. 910–931.

Beagley, H. A., Kellogg, S. E. (1968) A comparison of evoked response and subjective auditory thresholds. *Audiology* **7**: 420–421.

https://doi.org/10.1017/S0022215100124429 Published online by Cambridge University Press

British Society of Audiology (1981) Recommended procedures for pure-tone audiometry using a manually operated instrument. *British Journal of Audiology* **15:** 213–216.

Coles, R. R. A. (1982) Non-organic hearing loss. In *Otology*. (Gibb, A. G., Smith, M. F. W., eds.), Butterworth's International Medical Reviews, London, pp. 150–176.

Coles, R. R. A., Mason, S. M. (1984) The results of cortical electric response audiometry in medicolegal investigations. *British Journal of Audiology* **18:** 71–78.

Gleason, W. J. (1958) Psychological characteristics of the audiologically inconsistent patient. *Archives of Otolaryngology* **68:** 42–46.

Hyde, M., Alberti, P., Matsumoto, N., Yao, L.L. (1986) Auditory evoked potentials in audiometric assessment of compensation and medicolegal patients. *Annals of Otology, Rhinology and Laryngology* **95:** 514–519.

Jones, L. A., Harding, G. F. A., Smith, P. A. (1980) Comparison of auditory evoked potentials, brain stem evoked potentials and post-auricular myogenic potentials in normals and patients with known auditory defects. In *Evoked Potentials*. (Barber, C. ed.), MTP Press, England, pp. 337–344.

Keidel, W. D. (1976) The physiological background of the electric response audiometry. In *Handbook of Sensory Physiology*. (Neff, W. D., Keidel, W. D., eds.), Springer-Verlag, Berlin, pp. 105–231.

Martin, F. N. (1972) Differential diagnostic evaluation: non-organic loss and other special procedures. In *Handbook of Clinical Audiology*. (Katz, J., ed.), Williams and Wilkins, Baltimore, pp. 357–373.

Naatanen, R., Picton, T. (1987) The N1 wave of the human electric and magnetic response to sound: a review and analysis of the component structure. *Psychophysiology* **24 (4):** 375–425.

Parsuraman, R., Richer, F., Beatty, J. (1982) Detection and recognition: concurrent processes in perception. *Perception and Psychophysics* **31:** 1–12.

Picton, T. W., Woods, D. L., Baribeau-Braun, J., Healey, T. M. G. (1977) Evoked potential audiometry. *Journal of Otolaryngology* **6:** 91–119.

Picton, T. W., Durieux-Smith, A. (1988) Auditory evoked potentials in the assessment of hearing. *Neurologic Clinics* **6:** 791–808.

Rose, D. E., Keating, L. W., Hedgecock, L. D., Schreuers, K. K., Miller, K. E. (1971) Aspects of acoustically evoked responses. *Archives of Otolaryngology* **94:** 347–350.

Stappels, D., Picton, T. W., Perez-Abalo, M. *et al.* (1985) Frequency specificity in evoked potential audiometry. In *The Auditory Brain Stem Response*. (Jacobson, J. T., ed.), College Hill Press, San Diego, pp. 147–168.

Starr, A., Don, M. (1988) Brain potentials evoked by acoustic stimuli. In *Human Event-Related Potentials*. (Picton, T. W., ed.), Elsevier, New York, pp. 97–105.

Squires, K. C., Squires, N. K., Hillyard, S. A. (1975) Decision related cortical potentials during an auditory signal detection task with cued observation intervals. *Journal of Experimental Psychology: Human perception and Performance* **1:** 268–279.

Van der Drift, J. F. C., Brocaar, M. P., van Zanten, G. A. (1987) The relationship between the pure-tone audiogram and the click auditory brain stem response threshold in cochlear hearing loss. *Audiology* **26:** 1–10.

Address for correspondence:
Dr Deepak Prasher,
Institute of Laryngology and Otology,
The Royal National Throat, Nose and Ear Hospital,
330 Gray's Inn Road,
London WC1X 8EE.