Normal Acoustic Reflex Amplitude Growth and the Influence of Cochlear Hearing Loss

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ABSTRACT
The nature of acoustic reflex amplitude (ARA) growth at 1KHz and 2KHz was investigated in normal hearing and cochlear disordered subjects subdivided into Meniere’s Disease and heterogeneous pathology groups. Statistical and graphical analyses revealed significant inter-group variation in ARA growth rate. The normal and Meniere’s groups behaved similarly, while the heterogeneous group demonstrated a faster ARA growth rate. The differential sensitivity of various measurement methods was examined. Explanations were put forward to account for variability in ARA data amongst cochlear disordered subjects. It was concluded that the clinical sensitivity of ARA measurement was questionable.

OPSOMMING
Die toename van die akoestiese refleks amplitude (ARA) by 1KHz en 2KHz is in normaalhorendes en proejpersone met kogliere letsels ondersoek. Laasgenoemde is in Meniere se siekte en groepe met 'n heterogene patologie onderverdeel. 'n Betekenisvolle variasie tussen groepe m.b.t. die ARA-groeiempo van die heterogene groep was. Verder is die sensitieweverskil van verskeie metingsmetodes ondersoek. Ten einde die veranderlikheid in ARA-data tussen proejpersone met kogliere letsels te verantwoord, is verskeie verklarings gebied. Die gevolgtrekking is gemak dat die kliniese sensitiviteit van die ARA-meting bevaarbaar is.

The role of acoustic reflex measurement in determining the nature of sensori-neural hearing loss has been well established. Traditionally the acoustic reflex threshold test, a static measure, and the reflex decay test, a dynamic temporal measure, are employed for this purpose (Jerger, 1975). Measurement of other dynamic properties such as amplitude and latency has not yet achieved the same clinical status. Borg (1976) however, stated that such properties may contribute significantly to clinical diagnosis in impedance audiometry if they are proven to be pathology sensitive.

Acoustic reflex amplitude (ARA) is defined as the change in acoustic impedance between quiescent and reflexive states. ARA is intensity dependent: for pure tone stimulation it has a dynamic range of 20-30 dB, representing the intensity range over which the reflex shows an amplitude growth (Wilson and McBride, 1978). The nature of ARA growth in normal subjects has been variably reported in the literature. Uliel (1980) and Clemis and Sarno (1980) reported a curvilinear function while several other investigators have reported a linear function. Petersen and Liden (1972) and Beadle and Harford (1973) reported a slower growth rate in pathologic ears of variable cochlear etiology, than in normal ears. Both Uliel (1980) and Beadle and Harford (1973) specifically investigated ears with loudness recruitment and thus the discrepancy in their respective findings is particularly notable. A further confusion is that Jerger and Hayes (1983) reported an abnormally slow growth rate to be characteristic of a retrocochlear group. These discrepancies indicate that the influence of cochlear pathology on ARA has not been unequivocally established. Furthermore, since Uliel (1980) did not find an abnormal growth rate to be characteristic of all cochlear disordered subjects, it is logical to suspect that the influence of cochlear disorder on ARA might vary as a function of pathology. The need for further research on ARA in the cochlear population is clear. However, results cannot be classified validly as “faster” or “slower” than normal, until “normal” growth has been unequivocally defined. Further investigation into the shape of the ARA growth function in normal subjects is therefore also necessary.

METHODOLOGY

AIMS

The amplitude growth of the acoustic reflex was investigated at 1KHz and 2KHz, in normal hearing and cochlear disordered subjects with the aim of:

1. describing the configuration of the ARA growth function in normal hearing subjects for various measurement methods, namely: HL, SL, Δ HL, and Δ SL;

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2. investigating the influence of cochlear dysfunction on the ARA growth function. Specifically, to investigate ARA in a group of Meniere’s Disease subjects, and a group of subjects with variable cochlear etiology, excluding Meniere’s Disease;

3. examining the variable sensitivity of different measurement methods in distinguishing between the normal and pathological subject groups.

SUBJECTS
The subject sample in this investigation comprised three groups: a control group (A) of 16 normal hearing subjects (mean age 24 years; 38 test ears), an experimental group (B) of six subjects with Meniere’s Disease (mean age 38 years; six test ears), and a further experimental group (C) of four subjects with variable cochlear etiology, excluding Meniere’s Disease, (mean age 40 years; six test ears). This age limit of 20-50 years was imposed since ARA data has been shown to be relatively stable across this age range (Osterhammel and Osterhammel, 1979). Both sexes were represented.

Subjects were selected on the basis of case-history findings and audiologic results obtained by the author from a battery comprising pure tone air and bone conduction audiometry, impedance audiometry, the Metz Recruitment and Rosenberg Tone Decay tests. Table 1 summarises the specific criteria according to which subjects were differentiated into groups A, B and C.

<table>
<thead>
<tr>
<th>GROUP A</th>
<th>GROUP B</th>
<th>GROUP C</th>
</tr>
</thead>
<tbody>
<tr>
<td>Tympanometry</td>
<td>Type-A tympanogram to rule out the presence of middle ear disorders.</td>
<td>Type-A tympanogram to rule out the presence of middle ear disorders.</td>
</tr>
<tr>
<td>Static Compliance</td>
<td>0.55cc-1.5cc.</td>
<td>0.5cc-1.5cc.</td>
</tr>
<tr>
<td>Reflex Thresholds</td>
<td>Between 70-100 dB at all frequencies.</td>
<td>Between 70-100 dB at 1KHz and 2KHz, contralaterally.</td>
</tr>
<tr>
<td>Supra-threshold dynamic range</td>
<td>Minimum of 25 dBHL to allow for sufficient ARA growth.</td>
<td>Minimum of 25 dBHL to allow for sufficient ARA growth.</td>
</tr>
<tr>
<td>Metz Test</td>
<td>Negative.</td>
<td>Positive: ARSL &lt; 60 dB.</td>
</tr>
<tr>
<td>Tone Decay Test</td>
<td>—</td>
<td>Negative.</td>
</tr>
</tbody>
</table>

Table 1 Summary of specific criteria for subject selection and grouping

EQUIPMENT
A Madsen Electro-Acoustic Impedance Audiometer (model Z073A) which delivered a 220 Hz probe tone was used. This was calibrated according to standards set out in IEC publication 318. A Hewlett Packard Moseley X-Y Plotter (model 7035A) was connected to the impedance meter. The X-axis was activated by depression of the pure tone stimulus interrupter switch on the impedance meter, resulting in a time-locked 2 second excursion. Presentation of the 250 msec stimulus followed automatically but not immediately, allowing a baseline to be plotted before reflex elicitation.

The impedance meter was set at sensitivity 2 and the range of the Y-axis was calibrated such that 0.05 cc = 0.04 mv = 1 mm on the chart paper. An acoustic reflex was recorded as a relative deflection from the baseline, representing an increase in input impedance (refer to Figure 1).

EXPERIMENTAL PROCEDURE
Tymanometry was performed on each test ear prior to experimental testing in order to determine the point of maximum compliance. The acoustic reflex testing was conducted by the contralateral stimulus mode at 1KHz and 2KHz. The starting point for stimulus intensity was subject specific: testing began at reflex threshold and...
proceeded in 5 dB increments up to 125 dBHL. In this manner a series of graphical figures (refer to Figure 1) were obtained representing reflex amplitude at consecutive stimulus levels, for each test ear at both frequencies.

ANALYSIS OF RESULTS

The graphical figures represented the raw data. The amplitude of each reflex was established by calculating the millimetre measurement from the lowest point of the positive deflection to the highest point representing the maximal increase in input impedance. Millimetre measurements were converted to mV's according to the scale — 1 mm = 0.04 mV. mV scores used in data analysis were restricted to those at hearing levels (HL) and sensation levels (SL) at which a reflex response was common to all test ears. Data were also tabulated separately for those normal subjects who demonstrated the broadest dynamic range of amplitude growth, thus forming a subgroup of the normal sample.

The following analyses were performed:

1. GRAPHICAL ANALYSIS
   Mean ARA functions for the three subject groups were graphically displayed with the aim of examining the influence of subject group and measurement method variables on the configuration of these functions. Data for the subgroup of normal subjects was graphically displayed so as to examine the effect of dynamic range on function configuration.

2. SLOPE INDEX MEASUREMENT
   Slope index values were calculated for each graphical figure, based either on the whole HL or SL stimulus range (100-125 dBHL or 0-25 dBSL) or based only on the final two HL or SL increments (120-125 dBHL or 20-25 dBSL); thus observed differences in overall or 'tail-end' function configurations could be quantified.

3. STATISTICAL ANALYSIS
   The two-way analysis of variance statistic, with repeated measures on Β (2-ANOVA-RB), was used to establish whether a significant interaction existed between subject group (variable A) and stimulus level (variable B). The simple main effects (SME) and Tukey's honestly significant difference (HSD) statistics were used to quantify further the variation in ARA between subject groups at specific stimulus levels.

RESULTS AND DISCUSSION

NORMALS

The graphical representation of data from 38 test ears at 1KHz and 2KHz, over a 100-125 dBHL range and a 0-25 dBSL range, revealed that the nature of ARA growth was neither mathematically linear or curvilinear (refer to Figure 2). This finding contrasts with the relevant literature which has variably reported ARA configurations in normal subject groups to be exactly linear (Uliel, 1980; Clemis and Sarno, 1980) or curvilinear (Dallos, 1964; Sprague et al., 1981; Wilson and McBride, 1978). It is possible that this discrepancy between present and other research findings is a function of the small sample size and considerable variability in function configurations, that characterised this study.

The differential effect of measurement method on ARA configuration was visually evident at 2KHz but not at 1KHz (refer Figure 2). There was no difference between the HL-SL configurations at 1KHz, whereas at 2KHz an asymptotic function was characteristic of the SL but not the HL function.

The obtained ‘tail-end’ slope index values were as follows:

<table>
<thead>
<tr>
<th>Measure</th>
<th>Value</th>
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<tbody>
<tr>
<td>HL</td>
<td>0.3 mV</td>
</tr>
<tr>
<td>SL</td>
<td>0.3 mV</td>
</tr>
<tr>
<td>HL</td>
<td>0.5 mV</td>
</tr>
<tr>
<td>SL</td>
<td>0.1 mV</td>
</tr>
</tbody>
</table>

These values substantiated the graphical findings: while there was no difference between the HL and SL values at 1KHz, the SL index
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mV

30 dBHL

25 dBSL

100-125 dBHL

Figure 3 A complete ARA function for a sample of 10 normal subjects, with super imposed HL and SL ranges, at 1KHz and 2KHz.

at 2KHz was reduced relative to the HL value, indicating an asymptotic function.

This HL-SL configuration difference is in keeping with the findings of Sprague et al (1981), and requires an explanation particularly in view of its frequency selectivity in this study. Figure 3 illustrates an ARA function for the subgroup of normal subjects who demonstrated a broad dynamic range (85-125 dBHL) at 1KHz and 2KHz. The superimposition of HL and SL ranges on the graphical figures reveals that the differences in function configuration description (such as asymptotic versus non-asymptotic) can result from analyses of different portions of a wide intensity range. This possibly explains discrepancy in descriptions reported in the literature: linear versus curvilinear. Figure 3 illustrates further that the absence of an asymptotic SL function at 1KHz was simply a function of the breadth of dynamic range under investigation, namely 25 dBSL; an asymptotic function would have resulted had a 30 dBHL range been investigated. Considerable variability was found in the rate and pattern of ARA growth amongst normal subjects. This inter-subject variability was pronounced for HL measurement and relatively reduced for SL measurement, in keeping with the findings of Petersen and Linden (1972). A broad range of normal variability might obscure the clinical identification of mild or even moderate pathological deviance, and therefore it is implied that SL measurement offers a better prognosis for clinical sensitivity in ARA testing, than HL measurement, by reducing this range.

INTER-GROUP COMPARISONS

Graphical representation of the function configurations for groups A, B, and C, for SL data at 1KHz and 2KHz revealed a marked trend; group C demonstrated a visually steeper ARA function (faster ARA growth rate), while groups A and B were similar regardless of stimulus parameters (refer to Figures 4 and 5). This trend was also characteristic of HL data at 1 and 2KHz, although less pronounced.

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Whole slope index values quantified and confirmed these findings; the highest values were consistently obtained for group C, indicating a faster ARA growth rate in this group than in either groups A or B (refer to Table 2).

Figure 4 ARA functions at 1KHz, for SL data, showing inter-group growth rate trends.

Figure 5 ARA functions at 2KHz, for SL data, showing inter-group growth rate trends.
in this study.

...a normal growth rate as shown in the population have also yielded a slower than normal ARA growth rate (Petersen and Liden, 1972) and a normal growth rate. Clearly, a faster than normal ARA growth rate cannot be interpreted as being indicative of cochlear pathology since subjects in group C yielded the faster ARA growth rate.

The Tukey’s t-test results quantified inter-group variability at the specific stimulus levels identified by the SME statistic. The finding of a faster than normal ARA growth rate in the heterogeneous pathology group (C) agrees with the research of Clemis and Sarno (1980) in association with a faster than normal ARA growth rate. The use of this term in this context does not agree with other research. Beedle and Harford (1973) specifically investigated cochlear subjects with concomitant loudness recruitment and found a slower ARA growth rate. Furthermore, in this study all experimental subjects demonstrated loudness recruitment on the Metz Test, however only those subjects in group C yielded the faster ARA growth rate.

The dissimilarity in ARA growth rate between groups B and C was unexpected since Clemis and Sarno (1980) repeatedly observed a faster than normal ARA growth rate in Meniere’s Disease subjects, as did Sprague et al. (1981) in a single case of Meniere’s Disease.

The dissimilarity in ARA growth rate between groups B and C was surprising in view of their common lineage — both groups belonged to the cochlear population. This dissimilarity raises two issues for discussion: firstly, the variability in ARA data within the cochlear population requires an explanation, and secondly, the interpretation or meaning of a faster than normal ARA growth rate must be questioned. Clearly, a faster than normal ARA growth rate cannot be interpreted as being indicative of cochlear pathology since subjects in the population have also yielded a slower than normal ARA growth rate (Petersen and Liden, 1972) and a normal growth rate as shown in this study.

The 2-ANOVA-RB statistic for HL and SL measurement at 1KHz and 2KHz, yielded significant F-ratio values (p<0.01, and p<0.005 for SL data at 2KHz). The SME statistic indicated that significant variation within all subjects (variable A collapsed), existed at specific stimulus levels for 1KHz and 2KHz; these levels being more numerous for SL than HL data, and for 1 KHz and 2 KHz data. These findings suggest that the 1 KHz stimulus and the SL measurement method were more sensitive to inter-subject variation, and therefore have greater potential clinical value.

The finding of a faster than normal ARA growth rate in the heterogeneous pathology group (C) agrees with the research of Clemis and Sarno (1980) who found this to be characteristic of cochlear disordered subjects, but is contrary to the findings of Petersen and Liden (1972) and Beedle and Harford (1973) who found a slower growth rate in cochlear disordered subjects. The similarity in growth rate between groups A and B was unexpected since Clemis and Sarno (1980) repeatedly observed a faster than normal ARA growth rate in Meniere’s Disease subjects, as did Sprague et al. (1981) in a single case of Meniere’s Disease.

The SME statistic indicated that significant variation within all subjects (variable A collapsed), existed at specific stimulus levels for 1KHz and 2KHz; these levels being more numerous for SL than HL data, and for 1 KHz and 2 KHz data. The SME statistic is characteristic of Meniere’s disease patients (Brackman, Selters and Don, 1982) may be of significance when contrasted with the profile of subjects in group C, all of whom reported a minimum 10 year history of bilateral hearing loss with no fluctuating symptomatology. It would be of interest in future research to specifically compare a group of subjects with known hair cell damage, with a group of ‘early’ stage Meniere’s Disease patients who had shown symptom-reversibility on Glycerol testing (thus suggesting no permanent hair cell damage). Audiogram configuration is another variable which may have contributed to the dissimilarity between groups B and C. Five of the six Meniere’s subjects showed predominantly low frequency hearing loss whereas this was not characteristic of subjects in group C. Future research may reveal that lower test frequencies (below 1KHz) are more sensitive to Meniere’s Disease and therefore elicit a faster ARA growth rate in these subjects.

The term ‘recruitment’ has been used by Ulil (1980) and Clemis and Sarno (1980) in association with a faster than normal ARA growth rate. The use of this term in this context does not agree with other research. Beedle and Harford (1973) specifically investigated cochlear subjects with concomitant loudness recruitment and found a slower ARA growth rate. Furthermore, in this study all experimental subjects demonstrated loudness recruitment on the Metz Test, however only those subjects in group C yielded the faster ARA growth rate.

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An alternative interpretation of intra-cochlear variability and a faster ARA growth rate is based on the relationship between cochlear pathology and the neurological basis of the ARA response system. Evans (1982) states that damage to a cochlear fibre causes a broadening of its frequency threshold curve and a consequent increase in the rate at which adjacent cochlear fibres become activated as a function of increasing stimulus intensity. If this is an acceptable explanation of a faster ARA growth rate, then it is implied that cochlear fibre functioning in the Meniere’s Disease group was normal, since this group yielded an ARA growth rate mapping that of normal subjects. No definitive statements can be made regarding the differential effect of cochlear pathology on hair cell morphology in groups B and C; however, the variability in rate and stage of disease that is characteristic of Meniere’s disease patients (Brackman, Selters and Don, 1982) may be of significance when contrasted with the profile of subjects in group C, all of whom reported a minimum 10 year history of bilateral hearing loss with no fluctuating symptomatology. It would be of interest in future research to specifically compare a group of subjects with known hair cell damage, with a group of ‘early’ stage Meniere’s Disease patients who had shown symptom-reversibility on Glycerol testing (thus suggesting no permanent hair cell damage).

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CONCLUSIONS

The configuration of ARA growth functions obtained for normal hearing subjects were shown to be influenced by the method of ARA measurement, namely HL versus SL measurement. This variable determined which area of dynamic range of amplitude growth was analysed, and consequently determined the asymptotic versus non-asymptotic configuration difference. The same variable may also account for discrepancies in ARA configuration description in the literature. If this hypothesis is correct then the implication is that a standardised methodological basis for ARA is required, because without reliable normative data the relative influence of hearing impairment cannot be successfully determined.

On the basis of HL and SL measurement methods, the Meniere's Disease group yielded an ARA growth rate similar to that of normal subjects, while the heterogeneous pathology group showed a faster than normal ARA growth rate. This intra-cochlear variability has also been found in other studies and suggests that ARA measurement has a poor prognosis for clinical sensitivity. This is implied since faster than normal ARA growth rate (positive result) might indicate cochlear disorder but a negative test result would not contraindicate cochlear disorder.

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REFERENCES


In close cooperation with the Electro-Acoustic Department of Siemens, one of the world’s leading electrical and electronic engineering companies,

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