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Measurement of the temporal-modulation transfer function for a single listener with cochlear hearing loss and left-hemisphere damage

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Abstract

The modulation depth required for the detection of sinusoidal amplitude-modulation applied to a white noise carrier was measured as a function of modulation frequency, giving temporal modulation transfer functions (TMTFs). Five adult listeners with normal hearing (mean age 52 years), five elderly listeners with moderate cochlear hearing loss (mean age 66 years) and a single elderly listener (aged 73 years) with moderate cochlear hearing loss and left-hemisphere damage were tested in the right ear at 50 dB SL. The five elderly listeners were matched in audiogram with the brain-damaged listener. Modulation detection was systematically poorer than normal in the five elderly listeners with cochlear hearing loss. However, their TMTFs were lowpass in shape, as for the five normal-hearing adult listeners. Modulation detection was much poorer in the elderly listener with cochlear hearing loss and left-hemisphere damage compared to the five normal-hearing adults and the five elderly listeners with cochlear hearing loss. Moreover, modulation detection was poorer at 4, 64 and 128 Hz than at 8, 16 and 32 Hz in the brain-damaged listener, giving his TMTF a bandpass appearance. These results are in agreement with the hypothesis that the main factors limiting the ability to detect changes in the temporal-envelope of sounds are located at a central (retro-cochlear) level of the auditory system rather than at a peripheral (cochlear) level. They also suggest that the TMTF approach may prove useful in distinguishing peripheral and central hearing losses.

Key words: amplitude modulation, auditory cortex, auditory temporal acuity, retro-cochlear hearing loss, temporal modulation transfer time (TMTF)

Introduction

Speech understanding involves supra-threshold auditory processing in the spectral and the temporal domains. Part of the reason some listeners may have difficulty understanding speech in quiet or in the presence of background noise, reverberation or competing talkers may be related to abnormal auditory temporal acuity, that is, poorer-than-normal ability to resolve changes in the temporal envelope of sounds

(Festen and Plomp, 1990). Several methods have been developed to characterize auditory temporal acuity (see Viemeister and Plack, 1993; Moore 1995, 1997 for detailed reviews). The methods most commonly used in audiological and neurological studies are:

- Fusion threshold for a pair of successive clicks.
- Detection threshold for a temporal gap in a narrowband or broadband sound.

These two methods generally give a consistent estimate of temporal acuity of about 1–3 ms in normal-hearing listeners. The same methods

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show that temporal acuity is similar in normal-hearing listeners and listeners with cochlear lesions (Glasberg and Moore, 1992), providing that the stimuli used to measure temporal acuity are clearly audible and stimuli do not show audible intrinsic random fluctuations that could be confused with the temporal event to be detected in the temporal task. On the other hand, fusion or gap thresholds measured in patients with cortical or subcortical lesions (Albert and Bear, 1974; Chocholle et al., 1975; Auerbach et al., 1982; Efron et al., 1985; Tanaka et al., 1987; Yaqub et al., 1988; Divenyi and Robinson, 1989; Robin et al., 1990) are generally larger (sometimes considerably so) than normal (5–300 ms). Taken together, these data suggest that the main factors limiting the ability to detect changes in the temporal envelope of sounds are located at a central (retro-cochlear) level of the auditory system rather than at a peripheral (cochlear) level.

The two methods described above generally give a consistent estimate of auditory temporal acuity in normal-hearing listeners (1–3 ms). However, fusion and gap thresholds cannot be regarded as measures of temporal acuity *per se* because the ability to distinguish two clicks from a single click depends also on the detection of spectral changes above 10 kHz (Leshowitz, 1971) and the ability to detect a silent interval in a tone or noise is also limited by the amplitude resolution of the auditory system (Buunen and van Valkenburg, 1979).* Consequently, the conclusions of previous studies concerning the effects of cortical and subcortical lesions upon temporal acuity and using fusion or gap thresholds measurements should be treated with caution. A general approach developed by Viemeister (1979) circumvents these methodological limitations. This approach involves measuring temporal modulation transfer functions (TMTFs), that is, the ability of listeners to detect sinusoidal amplitude-modulation applied to a broadband noise, as a function of modulation frequency. TMTFs show that, for normal-hearing listeners, modulation sensitivity is relatively independent of modulation frequency up to approximately 16 Hz and decreases progressively at higher modulation frequencies (Viemeister, 1979; Bacon and Viemeis-

ter, 1985). In this modulation detection task, the use of spectral cues is fully precluded since the modulation of broadband noise does not affect its (flat) long-term power spectrum. For low modulation frequencies (below approximately 16 Hz), detection is limited by the amplitude resolution of the auditory system, rather than its temporal resolution. As the modulation frequency increases beyond approximately 16 Hz, temporal resolution starts to have an effect and modulation-detection thresholds increase.

In the present experiment, the effects of a cortical lesion on temporal acuity were measured using clearly audible stimuli. The TMTF approach was used in order to avoid the methodological biases of fusion and gap thresholds measurements. TMTFs were measured for a white noise carrier using a single patient with lesions to the left auditory cortex. As this patient was relatively old and showed cochlear hearing loss prior to brain damage, the two following control groups were used:

- Five adult listeners with normal hearing.
- Five elderly listeners with matched cochlear hearing loss.

The noise carrier was sufficiently wide to avoid audible fluctuations in amplitude. All subjects were tested at identical sensation levels (50 dB SL). Moreover, the five presbycusis listeners were chosen so as to be matched in audiogram with the brain-damaged patient, ensuring that differences between elderly listeners in terms of temporal acuity were not due to differences in the range of audible frequencies in the noise. Matching was based on the listeners' right ear audiometric thresholds, as modulation sensitivity was assessed using the right ear only (that is, the ear contralateral to the cortical lesion in the brain-damaged patient). Psychoacoustical investigation of the brain-damaged patient was finally completed by measuring intensity discrimination thresholds, that is his ability to discriminate between two *steady* white noises presented at different intensities. This task was introduced as a control task of the modulation-detection task. It was performed to:

*This argument has been developed by Viemeister and Plack (1993): a gap is equivalent to a decrement in the signal intensity. Buunen and van Valkenburg (1979) used a task that required listeners to detect brief decrements in the intensity of a broadband noise (rather than a period of complete silence, as in the gap detection task). Their results showed that the gap detection threshold was dependent on the depth of the decrement: the smaller the change in intensity, the larger the gap threshold. This illustrates the important role played by intensity differences and therefore, intensity resolution, in gap detection.

- Evaluate the contribution of potentially confounding non-perceptual effects related to stimulus characteristics (e.g. stimulus duration) and the forced choice procedure (e.g. the sequential mode of presentation of auditory stimuli).
- Evaluate the contribution of amplitude resolution to the modulation-detection thresholds measured at low modulation frequency.
- Dissociate the ability to perceive *steady* amplitude changes from the ability to perceive *dynamic* amplitude changes.

Method

Listeners

JPK is a 73-year-old, right-handed male. He was admitted to the neurological department of the Reims Hospital in February 1994 (at the age of 68 years) after suffering a cerebrovascular accident (CVA) with a sudden right hemiplegia and language disorders. A computed tomography (CT) scan revealed a left-sylvian ischemic lesion. Neurological assessment performed at this time confirmed a total right hemiplegia, and revealed both speech production and comprehension deficits and music perception deficits. JPK

underwent speech therapy for five months. In June 1994, JPK had recovered from his music perception deficit, with persistence of speech production and comprehension deficits. Neurological assessment performed at this time indicated a persistence of right leg and arm weaknesses. JPK was finally classified as a Wernicke aphasic. Figure 1 shows an MRI scan (axial view) of JPK performed three years after the CVA. T_2 -weighted images reveal the presence of an extended necrosis of brain tissues in the left sylvian area (superficial and deep parts) involving the cortical and subcortical parts of the posterior temporal lobe including T1 (superior temporal) gyrus.

Figure 2 shows a coronal MRI view of JPK, indicating that the left primary auditory area (Heschl's gyrus) has been severely damaged due to the sylvian infarct. All psychophysical experiments were performed five years after the CVA, and JPK was medically stable during this period. JPK was also diagnosed with bilateral, symmetrical high-frequency cochlear hearing loss four months before his CVA, and was fitted with a behind-the-ear hearing aid on the right ear at this time. Audiometric thresholds measured before

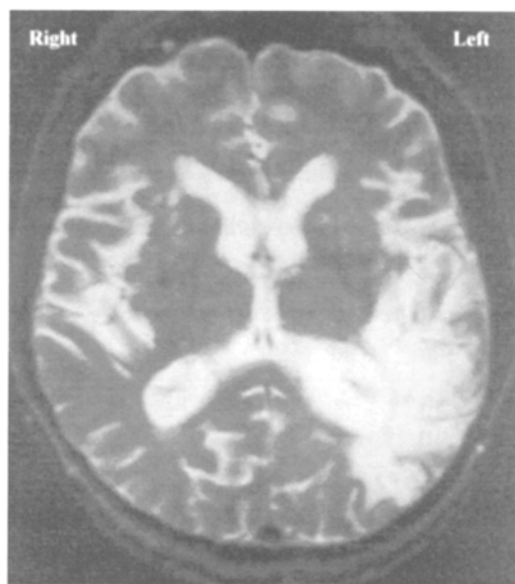


Fig. 1. MRI scan of patient JPK carried out three years after CVA. The Figure comprises a T_2 -weighted axial section revealing the presence of an extended necrosis of brain tissues in the left sylvian area (superficial and deep parts) involving the cortical and subcortical parts of the posterior temporal lobe including T1 (superior temporal) gyrus.

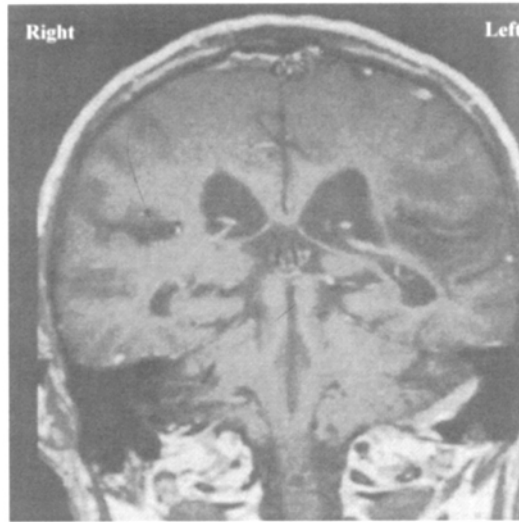


Fig. 2. Coronal MRI view of patient JPK (carried out three years after CVA as for Figure 1), indicating that the left primary auditory area (Heschl's gyrus) has been severely damaged due to the sylvian infarct.

and after the CVA were identical, indicating that the hearing loss had a purely cochlear origin. Hearing loss was classified as moderate in the right ear according to JPK's pure tone average (PTA) hearing loss at 250, 500, 1000, 2000 and 4000 Hz (PTA = 43 dB HL).

Table 1 gives JPK's audiometric thresholds measured in the right ear three months before the psychophysical experiments.

Two control groups of listeners without brain damage were also tested: five adult listeners (PH,

FH, MCH, JEM, JOM; mean age 52 years; standard deviation (SD): 4 years) with normal hearing (pure tone thresholds >20 dB HL between 250 Hz and 4000 Hz) and no history of hearing disorders; five elderly listeners (GA, LG, MDE, BL, MDO; mean age 66 years; SD 3 years) with bilateral, symmetrical high-frequency cochlear hearing loss. As for JPK, the absence of conductive component to their hearing loss was indicated by normal tympanograms and air-bone differences in absolute threshold <15 dB; hearing

Table 1. Characteristics of listeners: age (years) audiometric thresholds for the right ear (dB HL) and diagnosis for hearing-impaired listeners

Listener	Age	Frequency (Hz)					Diagnosis
		250	500	1000	2000	4000	
PH	51	20	10	15	15	15	Normal
FH	48	10	10	10	15	15	Normal
MCH	58	20	20	20	10	20	Normal
JEM	53	10	10	15	10	20	Normal
JOM	51	15	10	10	10	15	Normal
GA	67	35	25	40	65	80	Presbycusis
LG	70	25	25	25	50	70	Presbycusis
MDE	65	20	30	35	45	75	Presbycusis
BL	67	5	15	35	65	85	Presbycusis + noise exposure
MDO	61	35	30	50	60	105	Presbycusis
JPK	73	30	15	25	65	80	Presbycusis + cortical lesions

loss in the right ear was classified as moderate according to PTA hearing loss ($39 \leq \text{PTA} \leq 56$ dB HL). Table 1 gives ages and audiometric thresholds of these normal-hearing and hearing-impaired listeners in the right ear.

Before experimentation, (unaided) speech intelligibility was assessed with the phonetic test of Lafon (Lafon, 1972; lists of 17 French words; each word including three phonemes and all lists being phonetically balanced) in listener JPK and the five elderly listeners with cochlear hearing loss. For each listener, speech intelligibility was assessed in the right ear in the presence of a contralateral white noise presented at 30 dB. All stimuli were delivered via Sennheiser HD 265 earphones. Figure 3 shows individual psychometric functions for these six listeners, that is, the percentage of phonemes correctly identified as a function of speech level. In the phonetic test of Lafon (1972), speech-identification threshold refers to the speech level needed for 50% of phonemes to be correctly identified. In agreement with their diagnosis of cochlear hearing

loss, the speech-identification thresholds for the five presbycusis control subjects ranged from 35 dB to 55 dB, and identification performance was nearly perfect at 80 dB. In comparison, JPK's performance reached a maximum of 30% correct identification at 80 dB, supporting the diagnosis of central auditory disorder in addition to cochlear hearing loss.

TMTFs

Apparatus and stimuli

All stimuli were generated by use of a 16-bit D/A converter at a sampling frequency of 44.1 kHz, and were delivered monaurally via a Sennheiser HD 265 earphone. The white noise carrier was independently generated for every stimulus. Listeners had to detect the presence of sinusoidal amplitude modulation applied to the white noise carrier. On each trial, a standard and a target stimulus were successively presented in random order to the listener. The standard was unmodulated white noise $n(t)$. In the target, the white noise carrier was sinusoidally amplitude-modulated

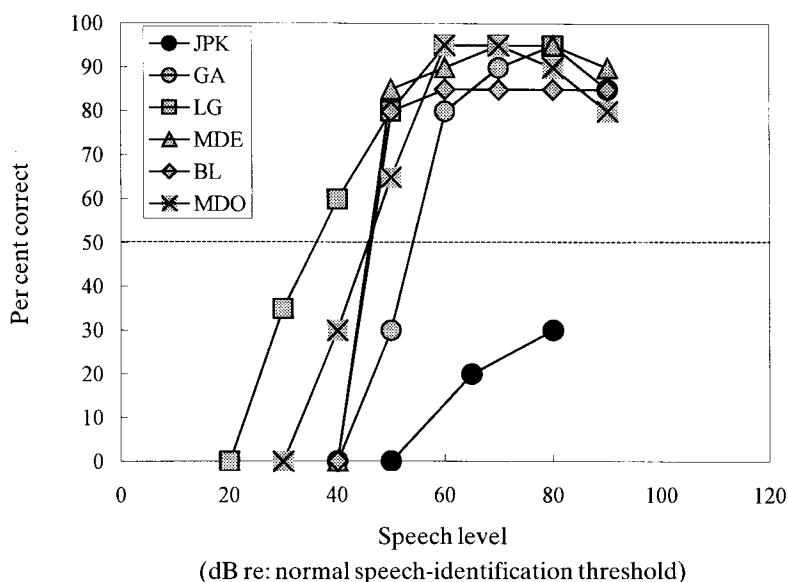


Fig. 3. Unaided speech intelligibility assessed with the phonetic test of J.C. Lafon (Lafon, 1972) in listener JPK (black circles) and the five elderly listeners with cochlear hearing loss (grey symbols). Speech intelligibility was assessed in the right ear, in the presence of a contralateral masking white noise. For each listener, the percentage of phonemes correctly identified is plotted as a function of speech level (in dB re: speech-identification threshold for normal-hearing listeners). The performance level corresponding to 50% of phonemes correctly identified is indicated by the dotted line.

at a given modulation frequency. The expression describing the target was:

$$s(t) = c [1 + m \sin(2\pi f_m t)] n(t) \quad (1)$$

where m is the modulation depth ($0 \leq m \leq 1$); f_m is the modulation frequency (f_m was 4, 8, 16, 32, 64 or 128 Hz). The term c is a multiplicative compensation term (Viemeister, 1979) set so that the overall power was the same in all intervals. The expression for c is:

$$c = [1 + m^2/2]^{-0.5} \quad (2)$$

The stimuli were presented at a sensation level (SL) of 50 dB. To further discourage the use of intensity cues, random variations of ± 1 dB in 1-dB steps were applied independently to the standard and target intensities. Both the standard and target had a 2 s duration, including 25 ms rise/fall times, shaped using a raised-cosine function. The inter-stimulus interval was 1 s. Stimuli were low-pass filtered at 20 kHz and delivered monaurally to the listeners' right ears. Listeners were tested individually in a soundproof booth.

Procedure

Modulation-detection thresholds were obtained using an adaptive two-interval, two-alternative forced-choice (2I, 2AFC) procedure that estimates the modulation depth, m , necessary for 70.7% correct detection. The listener's task was to identify the interval containing the modulation. Control subjects entered their response using a response box. Because of his aphasic disorder and right hemiplegia, JPK was unable to use the response box and to describe verbally what he perceived. He therefore responded by humming and gesturing what he perceived (stationary, fluctuating or buzzing sounds). Then, an experimenter entered JPK's responses with the response box. Visual feedback about the correct interval was given after each trial. The step sizes and thresholds were based on the modulation depth in decibels ($20 \log m$). The step size of m was initially 4 dB and was reduced to 2 dB after the first two reversals. The mean value of $20 \log m$ at the last 10 reversals in a block of 14 reversals was taken as the threshold estimate for that block. Thresholds presented here are based upon three estimates for each control subject (i.e. normal-hearing and hearing-impaired listeners without brain damage), and upon two estimates for JPK (data collection was extremely slow with

JPK (one threshold estimate/week) and JPK complained increasingly about the difficulty of the task). The worst threshold that could be measured is 0 dB, which corresponds to a modulation depth of 1 (100% modulated noise). The more negative the value of $20 \log m$, the better the detection threshold.

Intensity discrimination thresholds

Apparatus and stimuli

Again, all stimuli were generated by use of a 16-bit D/A converter at a sampling frequency of 44.1 kHz, and were delivered monaurally via a Sennheiser HD 265 earphone. The white noise carrier was independently generated for every stimulus. On each trial, a standard and a target stimulus were successively presented in random order to JPK. The standard consisted of a white noise presented at a reference intensity (I) giving a SL of 50 dB. The target consisted of another white noise presented at a greater intensity ($I + \Delta I$). Both the standard and target had a 2 s duration, including 25 ms rise/fall times, shaped using a raised-cosine function. The inter-stimulus interval was 1 s. Stimuli were low-pass filtered at 20 kHz and delivered monaurally to JPK's right ear.

Procedure

Intensity difference limens were obtained using a 2I, 2AFC procedure that estimates the intensity difference (ΔI) necessary for 70.7% correct detection. JPK's task was to identify the loudest sound interval. As for the modulation-detection task, JPK responded by gesturing what he perceived. Then, an experimenter entered JPK's responses with a response box. Visual feedback concerning the correct interval was given after each trial. The step sizes and thresholds (ΔL) were calculated in decibels ($\Delta L = 10 \log \{(I + \Delta I)/I\}$). The step size of variation was initially 2 dB and was reduced to 0.25 dB after the first two reversals. The mean of the last 10 reversals in a block of 14 reversals was taken as the threshold estimate for that block. Two estimates could be collected on JPK's right ear.

Results

Figure 4 (A) presents the individual TMTFs. Figure 4 (B) presents the mean TMTFs for normal-hearing listeners, for hearing-impaired listeners and for patient JPK. The TMTFs of the five normal-hearing adults and five hearing-impaired

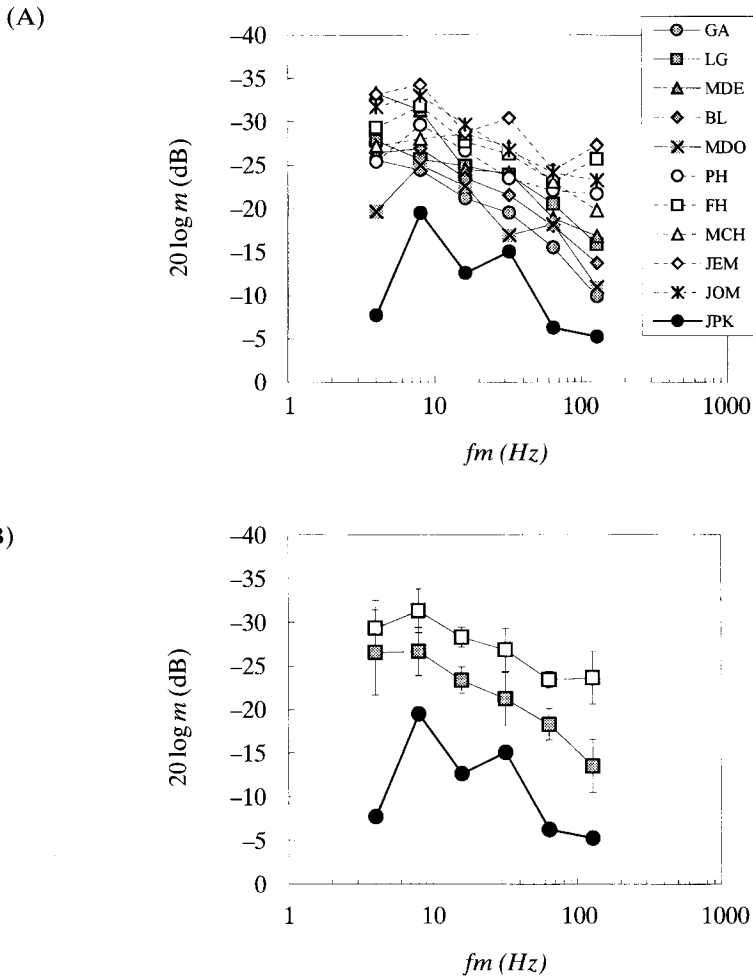


Figure 4. Temporal modulation transfer function (TMTFs) obtained with a gated white noise carrier. Thresholds for detecting sinusoidal amplitude modulation of the noise carrier are expressed as $20 \log m$, where m is the modulation depth, and plotted as a function of modulation frequency f_m . Note that the y-axis is upside down (i.e. negative values of $20 \log m$ are at the top). (A): Individual TMTFs for normal hearing adults (unfilled symbols with dotted lines); elderly listeners with cochlear hearing loss (grey symbols with continuous lines); and patient JPK (black circles with thick continuous lines). (B): Mean TMTFs for normal-hearing adults (unfilled squares with thin lines); and elderly listeners with cochlear hearing loss (grey squares with thin lines). Error bars indicate the across-listener standard error of the mean. Mean TMTFs are plotted along with the individual TMTF of patient JPK (black circles with thick lines).

elderly listeners without brain damage are similar to those reported by previous studies for normal-hearing adults (Robenburg, 1977; Viemeister, 1979; Takahashi and Bacon, 1992; Eddins, 1993; Akeroyd and Patterson, 1997),

adults with moderate to severe sensorineural hearing loss (Bacon and Viemeister, 1985; Moore et al., 1992) and elderly people with mild sensorineural hearing loss (Takahashi and Bacon, 1992). Overall, modulation-detection thresholds

are relatively constant up to approximately 16 Hz and increase beyond that frequency, giving the TMTFs their typical lowpass shape. In agreement with the data of Bacon and Viemeister (1985) and Takahashi and Bacon (1992), the five elderly listeners with cochlear hearing loss (mean age 66 years) have poorer modulation-detection thresholds than adult listeners with normal hearing (mean age 52 years), especially at high modulation frequencies (here, at 128 Hz). The mean difference in detection thresholds between normal-hearing adults and elderly listeners with cochlear hearing loss ranges from 3 dB to 6 dB for modulation frequencies between 4 Hz and 64 Hz and reaches a maximum of 10 dB at 128 Hz.

Figure 4 also reveals that the TMTF of patient JPK differed strongly from those for the two control groups. First, modulation detection is much poorer for JPK than for the five normal-hearing adults and the five elderly listeners with cochlear hearing loss. The deviation in sensitivity from the five elderly listeners with cochlear hearing loss reached a maximum of approximately 19 dB at 4 Hz. At this modulation frequency, JPK's first and second estimates of modulation detection threshold are -10 dB and -5 dB, respectively. Second, JPK's modulation thresholds were 6–12 dB poorer at 4, 64 and 128 Hz than at 8, 16 and 32 Hz, giving his TMTF a bandpass shape. Modulation detection was very poor at the lowest and highest modulation frequencies for JPK. Nevertheless, the relatively low modulation-detection threshold measured at 8 Hz (-20 dB) indicates that JPK was able to perform the task.

An analysis of variance (ANOVA) was used to estimate the significance of the differences in modulation detection thresholds between normal hearing adults, elderly listeners with cochlear damage and JPK. This analysis, conducted with factors of group (three levels) and modulation frequency (six levels), showed significant main effects of group [$F(2,8)=23.61$; $p<0.0005$] and modulation frequency [$F(5,40)=40.41$; $p<0.000001$]. The analysis also showed a significant interaction between group and modulation frequency [$F(10,40)=5.26$; $p<0.00001$]. Post-hoc comparisons (Tukey HSD) showed that modulation detection thresholds measured in elderly listeners with cochlear hearing loss were significantly higher than those measured in normal-hearing adults at modulation frequencies between 8 Hz and 128 Hz ($p<0.01$). However, no significant difference was observed at a modulation

frequency of 4 Hz ($p=0.46$). Modulation detection thresholds measured in JPK were significantly higher than those measured in normal hearing adults ($p<0.0005$). They were also significantly higher than those measured in elderly listeners with cochlear hearing loss at all modulation frequencies ($p<0.05$) except 32 Hz where no significant difference was observed ($p=0.13$).

Two estimates of intensity discrimination threshold could be obtained in JPK: $\Delta L_1=1$ dB and $\Delta L_2=1.7$ dB. The first estimate of intensity discrimination threshold (ΔL_1) is within the normal range (0.5–1 dB) for wideband noise stimuli (Miller, 1947). The second estimate (ΔL_2) is slightly poorer than normal (by 0.7 dB). Nevertheless, the nearly normal performance of JPK in this intensity discrimination task indicates that non-perceptual effects related to the stimulus characteristics (stimulus duration, attention for duration of task) or the 2I, 2AFC procedure used in this study (e.g. the sequential mode of presentation of auditory stimuli; response problems) did not contribute substantially to the performance measured in the modulation-detection task. It also suggests that the worse-than-normal modulation-detection threshold measured in JPK at 4 Hz was mainly caused by a deficit in temporal-envelope processing *per se* rather than a deficit in amplitude resolution.

Discussion

Modulation detection thresholds and speech identification performance were measured in an elderly patient with cochlear hearing loss and left-hemisphere damage. The results show that both modulation sensitivity and speech intelligibility were much poorer in this elderly patient with cochlear hearing loss and left-hemisphere damage when compared to two groups of control subjects (i.e. normal-hearing adult listeners and elderly listeners with matched cochlear hearing loss).

The TMTF approach used in the present study enabled us to measure auditory temporal-envelope processing *per se* without any confounding effects related to auditory spectral and intensity resolution. Consistent with previous studies of the effects of cochlear damage and ageing on temporal acuity (Bacon and Viemeister, 1985; Moore et al., 1992; Takahashi and Bacon, 1992), our results show that the TMTFs of normal hearing adult listeners and elderly listeners with

cochlear hearing loss are similar and lowpass in shape. In comparison, the TMTF of our single elderly listener with cochlear hearing loss and left-hemisphere damage is more bandpass than lowpass in shape, revealing important modulation-detection deficits at low (4 Hz) and high (64 Hz and 128 Hz) modulation frequencies. More precisely, the main difference in shape appears to be in the one point at 4 Hz. The presence of a more salient deficit at 4 Hz is consistent with previous electrophysiological studies of the temporal responses of cortical neurones showing that, in mammals, neurones in primary and secondary auditory cortical areas respond preferentially to low (<30 Hz) amplitude-modulation frequencies (Schreiner and Urbas, 1986, 1988; Eggermont, 1994). Without demonstrating it, these results are also in agreement with the hypothesis that structures within the left hemisphere are critically involved in temporal-envelope detection. This is in general agreement with the data of Robin et al. (1990) showing that gap detection thresholds are poorer than normal in patients with left-hemisphere lesions, but within the normal range in patients with right-hemisphere lesions. This is also consistent with the results of a similar single-case report showing impaired modulation detection and speech identification performance in an aphasic patient with lesions involving the left inferior-frontal and superior-temporal gyri (Lorenzi et al., 2000). Previous studies conducted with normal-hearing listeners have demonstrated that degrading speech by filtering out low modulation frequencies (between 4 Hz and 16 Hz) leads to poorer speech intelligibility (Drullman, 1994a, b; Horst et al., 1999). The important deficit in speech identification revealed by the clinical evaluation of JPK appears therefore consistent with the patient's impairment in detecting 4-Hz amplitude modulation.

A traditional model used to account for human sensitivity to amplitude modulation assumes that the temporal envelope of the stimuli is smoothed by a single temporal integrator operating at a post-cochlear level (Viemeister, 1979). However, the results of psychophysical adaptation and masking experiments (Tansley and Suffied, 1983; Houtgast, 1989; Dau et al., 1997) performed in humans with modulated sounds have suggested an alternative model in which perceptual channels, each tuned to a given low modulation frequency, decompose the temporal envelope of sounds at a central level. This alternative hypothesis is supported by electrophysiological record-

ings performed in cats at the level of the auditory cortex (Schreiner and Urbas, 1986, 1988). Despite the correspondence between psychophysical and electrophysiological data, the controversy regarding the nature of the temporal processor persists. The bandpass shape of JPK's TMTF, and especially the poorer detection thresholds at 4 Hz than at 8, 16 and 32 Hz, are not readily predicted by a single temporal integrator preserving the lowest amplitude fluctuations and smoothing the fastest ones. At first sight, this would therefore provide support for the modulation filterbank approach (JPK showing an impaired 'modulation-channel' selectively tuned to 4 Hz). However, JPK's impairment in modulation detection at 4 Hz may reflect a mere temporal integration deficit (that is a limited ability to summate the detection cues over time), or abnormal adaptation (so that slow changes in amplitude are effectively reduced in modulation depth). These two alternative explanations in terms of abnormal temporal integration and adaptation would not preclude the existence of a single temporal integrator.

It is important to realize that auditory abilities in patients with various types of central deafness are generally not known because of the paucity of extensive psychoacoustical testing of these patients and the absence of adequate evaluation of the hearing deficits (Musiek et al., 1994). The present study suggests that amplitude-modulation detection is potentially sensitive to central lesions. On the other hand, previous studies have shown that neither cochlear damage (Moore et al., 1992) nor ageing (up to 70–76 years) (Takahashi and Bacon, 1992) affect the shape of the TMTF when stimuli are clearly audible. This procedure appears therefore well-suited for the in-depth clinical assessment of the effects of brain damage on temporal-envelope processing in young and elderly patients, as cochlear damage and age effects are controlled for.

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