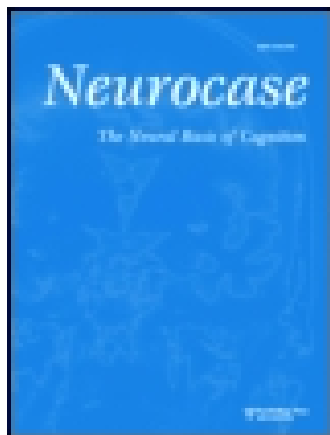


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Publisher: Routledge

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## Neurocase: The Neural Basis of Cognition

Publication details, including instructions for authors and subscription information:

<http://www.tandfonline.com/loi/nncs20>

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Published online: 02 Feb 2010.

To cite this article: Christian Füllgrabe, Didier Mailliet, Christine Moroni, Catherine Belin & Christian Lorenzi (2004) Detection of 1<sup>st</sup>- and 2<sup>nd</sup>-order Temporal-envelope Cues in a Patient With Left Superior Cortical Damage, *Neurocase: The Neural Basis of Cognition*, 10:3, 189-197, DOI: [10.1080/13554790490495258](https://doi.org/10.1080/13554790490495258)

To link to this article: <http://dx.doi.org/10.1080/13554790490495258>

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# Detection of 1<sup>st</sup>- and 2<sup>nd</sup>-order Temporal-envelope Cues in a Patient With Left Superior Cortical Damage

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## Abstract

This psychophysical study explores the extent to which the auditory cortex is necessary for various aspects of temporal-envelope perception, that is, perception of the slow temporal modulations in amplitude known to be crucial for sound identification. The ability to detect 1<sup>st</sup>- and 2<sup>nd</sup>-order sinusoidal amplitude modulation (AM) is evaluated in a single patient showing left-hemisphere damage encroaching the primary and secondary auditory cortices. Here, 1<sup>st</sup>- and 2<sup>nd</sup>-order AM refer to (1) sinusoidal variation in the amplitude of a 2 kHz pure tone, and (2) sinusoidal variation in the *depth* of a 64 Hz AM applied to the 2 kHz pure tone, respectively. The results replicate previous findings by showing that damage to the left auditory cortex results in a selective deficit in auditory sensitivity to the lowest 1<sup>st</sup>-order AM (i.e., 1<sup>st</sup>-order AM frequencies  $\leq 16$  Hz). Moreover, a dissociation is apparent between the ability to detect 1<sup>st</sup>- and 2<sup>nd</sup>-order temporal-envelope cues. The patient shows poorer than normal ability to detect 2<sup>nd</sup>-order AM at low frequencies ranging from 4–23 Hz, but normal ability to detect the high (64 Hz) 1<sup>st</sup>-order AM carrying these 2<sup>nd</sup>-order modulations. This result indicates that damage to the left primary and secondary auditory cortices affects the ability to detect temporal variations in the *local* properties of sounds (such as AM depth). It is also consistent with the idea that, as in vision, central nonlinear mechanisms are involved in the computation of such local (or 2<sup>nd</sup>-order) temporal properties.

## Introduction

In vision, spatial and motion information are carried not only by variations in image intensity (i.e., luminance, a “1<sup>st</sup>-order” characteristic of the visual image) but also can be carried by variations in higher-order image characteristics such as contrast (a “2<sup>nd</sup>-order” characteristic; Smith, 1994). In other words, visual images contain two types of structures: (1) a 1<sup>st</sup>-order structure, corresponding to a description of the way visual intensity varies across space or space-time, and (2) a 2<sup>nd</sup>-order structure, corresponding to a description of the way some other *local* properties of the stimulus (such as contrast) vary across space or space-time.

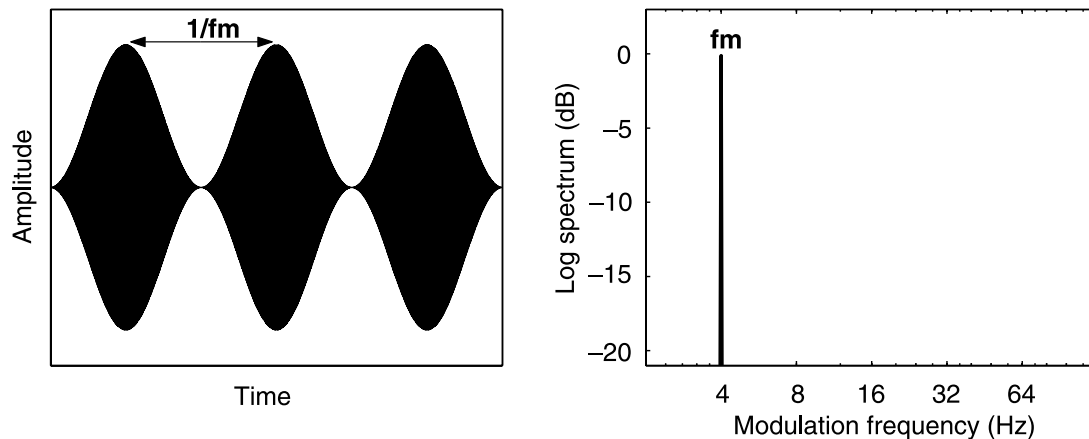
So far, many psychophysical (e.g., Cavanagh and Mather, 1989; Lu and Sperling, 1995; Chubb and Sperling, 1988), brain imaging (Smith *et al.*, 1998) and neuropsychological

studies (Greenlee and Smith, 1997) conducted with human subjects on motion perception have suggested that so-called “2<sup>nd</sup>-order motion” evoked by moving contrast modulation is detected by a different mechanism from that which detects 1<sup>st</sup>-order motion (luminance defined motion). This parallel mechanism or route may involve a central nonlinear transform of the stimulus that has the effect of introducing a low distortion product at the contrast modulation frequency in the sensory space-time representation of visual stimuli (e.g., Burton, 1973; Henning *et al.*, 1975; Wilson *et al.*, 1993). Although some studies indicate that an early compressive nonlinearity at the photoreceptor level introduces 1<sup>st</sup>-order components into the sensory representation of 2<sup>nd</sup>-order stimuli, it is generally assumed that the

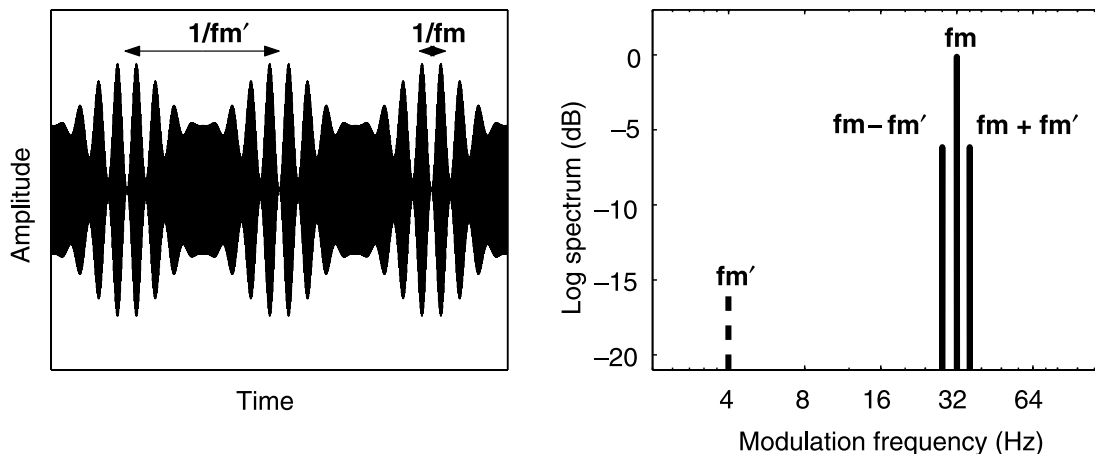
main source of distortion occurs at a more central stage (e.g., Wilson *et al.*, 1993; Scott-Samuel and Georgeson, 1999).

This general approach, which proved to be extremely successful in vision studies, has been recently transposed to the auditory modality by suggesting that dynamic information in acoustic stimuli not only is carried by temporal variations in the sound's amplitude (a 1<sup>st</sup>-order characteristic of the acoustic stimulus, corresponding to the sound's onset and offset, or to the slow amplitude modulations [AM] composing the temporal envelope of sound), but also can be carried by temporal variations in 2<sup>nd</sup>-order acoustic characteristics such as *AM depth* (the auditory analog of visual contrast; Shofner *et al.*, 1996; Sheft and Yost, 1997; Moore *et al.*, 1999; Lorenzi *et al.*, 2001a, b; Ewert *et al.*, 2002; Füllgrabe and Lorenzi, 2003; Millman *et al.*, 2003). First- and 2<sup>nd</sup>-order AM stimuli are illustrated in Figs. 1 and 2, respectively. As shown in Fig. 1

(left panel), the 1<sup>st</sup>-order AM stimulus is obtained by applying sinusoidal modulation to the amplitude of a pure-tone or a noise stimulus (the “audio” carrier). First-order sinusoidal modulation is described by its frequency  $fm$ , and its depth  $m$  (in percent). The right panel in Fig. 1 shows that there is spectral energy in the (physical) modulation spectrum at the (1<sup>st</sup>-order) AM frequency  $fm$ . A 2<sup>nd</sup>-order AM stimulus is illustrated in Fig. 2. Here, the (1<sup>st</sup>-order) AM signal acts as a “carrier” stimulus of frequency  $fm$ , and 2<sup>nd</sup>-order AM is obtained by applying a slow sinusoidal modulation of frequency  $fm'$  to the depth  $m$  of the carrier 1<sup>st</sup>-order AM. This 2<sup>nd</sup>-order AM is thus defined by its frequency  $fm'$  and its depth  $m'$  (in percent). Fig. 2 shows that the sinusoidal modulation of the AM signal's depth generates (1) two additional components (i.e., sidebands) in the stimulus' modulation spectrum at frequencies  $fm - fm'$  and  $fm + fm'$ , and (2) a beat, i.e., a slow cyclic increase and decrease in



**Fig. 1.** Waveform (left panel) and modulation spectrum (right panel) of a first-order AM of frequency  $fm$  (4 Hz). The right panel shows that there is spectral energy in the (physical) modulation spectrum at the envelope frequency  $fm$ .



**Fig. 2.** Waveform (left panel) and modulation spectrum (right panel) of a second-order AM of frequency  $fm'$  (4 Hz) applied to the depth of an AM signal of frequency  $fm = 32$  Hz. The right panel shows that there is no spectral energy in the (physical) modulation spectrum at the envelope beat frequency  $fm'$ ; the dashed line represents the hypothetical distortion product at frequency  $fm'$  (4 Hz) upon which second-order AM perception may partially be based.

fluctuation strength of the stimulus' temporal envelope at a frequency equal to  $fm'$ .

Psychophysical studies assessed auditory sensitivity to 2<sup>nd</sup>-order temporal envelope cues (i.e., slow variations in AM depth) by measuring 2<sup>nd</sup>-order temporal modulation transfer functions (TMTF). These relate detection thresholds for 2<sup>nd</sup>-order sinusoidal AM (i.e.,  $m'$  at threshold) to  $fm'$ , the frequency of the 2<sup>nd</sup>-order sinusoidal modulation (Lorenzi *et al.*, 2001a, b; Füllgrabe and Lorenzi, 2003). Second-order TMTFs measured in normal-hearing listeners for various carrier modulation frequencies  $fm$  display a lowpass shape, indicating that sensitivity to 2<sup>nd</sup>-order AM degrades when the 2<sup>nd</sup>-order frequency increases. Additional psychoacoustical measurements (Lorenzi *et al.*, 2001a) reveal that 2<sup>nd</sup>-order modulation detection thresholds are degraded when masker (1<sup>st</sup>-order) modulations are introduced near the envelope beat frequency  $fm'$ . Together with previous psychoacoustical and electrophysiological data (Shofner *et al.*, 1996; Sheft and Yost, 1997; Moore *et al.*, 1999; Ewert *et al.*, 2002), this result supports the idea that detection of spectral energy at the envelope beat frequency  $fm'$  contributes to the detection of 2<sup>nd</sup>-order modulation. In other words, this result suggests that 2<sup>nd</sup>-order AM is (perceptually) converted into 1<sup>st</sup>-order AM via a given auditory mechanism. Since there is no energy at the envelope beat frequency  $fm'$  in the modulation spectrum of the physical stimulus (as shown in Fig. 2), it is argued that, as in vision, some nonlinear mechanism along the auditory pathway must generate an audible distortion component at the envelope beat frequency  $fm'$  in the internal (i.e., sensory) modulation spectrum of complex temporal envelopes (however, see Füllgrabe and Lorenzi, 2003; Millman *et al.*, 2003 for a different interpretation).

Additional psychoacoustical studies conducted with listeners showing cochlear damage and cochlear implantees (Tandetnik *et al.*, 2001; Füllgrabe *et al.*, 2003; Lorenzi *et al.*, in press) suggest that peripheral nonlinearities such as fast-acting compression produced by outer hair cells within the cochlea do not contribute to the generation of the distortion component. Therefore, the nonlinearities must be located at a post-cochlear level of the auditory system, if the perception of a distortion product is involved in 2<sup>nd</sup>-order AM detection. Consistent with this notion, an electrophysiological study (Shofner *et al.*, 1996) indicates that certain neural units in the brainstem show synchronized responses to 2<sup>nd</sup>-order temporal modulations. Moreover, a brain imaging study using the MEG technique (Draganova *et al.*, 2002) reveals simultaneous coding of 1<sup>st</sup>- and 2<sup>nd</sup>-order modulation frequency information in the human auditory cortex. Source localization indicates that the response patterns elicited by each type of AM originate from overlapping or adjacent areas of the primary auditory cortex.

Given the paucity of available neurophysiological data on complex AM processing, little is known concerning which central (i.e., neural) structures of the auditory system mediate 2<sup>nd</sup>-order envelope perception. The present psychoacoustical study addressed this issue by exploring – for the first time –

auditory sensitivity to 1<sup>st</sup>- and 2<sup>nd</sup>-order AM in a single patient with unilateral damage to the left temporal cortex in order to determine whether or not cortical auditory areas play a role in 2<sup>nd</sup>-order temporal modulation processing, or, in other words, whether or not cortical auditory areas play a role in the ability to detect temporal variations in the *local* properties of sounds (such as AM depth).

## Case report

LA is a 70 year-old, right-handed female. She is a native French speaker. After graduating from junior high school, she was employed as a manager in the town hall until her cerebrovascular accident (CVA). She was admitted to the neurological department of the Avicenne Hospital in August 2001 for sudden speech and language disorders. A CT scan revealed a left-sylvian ischemic lesion. Neurological assessment did not indicate any sensorimotor deficit. The Montreal-Toulouse Aphasia Battery (MT-86; Nespoulous *et al.*, 1986; Béland and Roch Lecours, 1990) was administered to LA at that time (in August 2001; see Table 1, second column). The results obtained with this MT-86 test indicated a moderate naming deficit for oral and written language and a severe deficit in oral comprehension. Moreover, LA showed deficits in oral repetition and writing to dictation, but no deficit was observed in the ability to copy and read. This general profile corresponded to an aphasic disorder with a deficit in auditory analysis for verbal stimuli. LA underwent intensive speech therapy until her second neuroradiological and neuropsychological assessment. Raven matrices and the MT-86 test were administered to LA in June 2002, prior to the psychophysical measurements (see Table 1, third column). Her IQ measured at that time using Raven matrices was normal (116). The

**Table 1.** Neuropsychological assessment of patient LA using the Montreal-Toulouse Aphasia Battery (MT 86). Assessment was performed twice (in August 2001 and June 2002)

Sub tests (MT 86)	August 2001	June 2002
<i>Oral Language</i>		
Spontaneous Language	12/18**	17/18
Oral comprehension (single words)	9/9	9/9
Oral comprehension (sentences)	21/38***	35/38*
Object manipulation (following oral instructions)	1/8***	7/8
Repetition (of single words and sentences)	28/33*	29/33*
Oral naming	20/31**	29/31*
<i>Written Language</i>		
Ability to copy	12/13	12/13
Reading	33/33	33/33
Written Naming	37/39	34/39*
Writing to dictation	2/35***	21/35**
Written comprehension (single words)	5/5	5/5
Written comprehension (sentences)	8/8	8/8

\*Mild deficit.

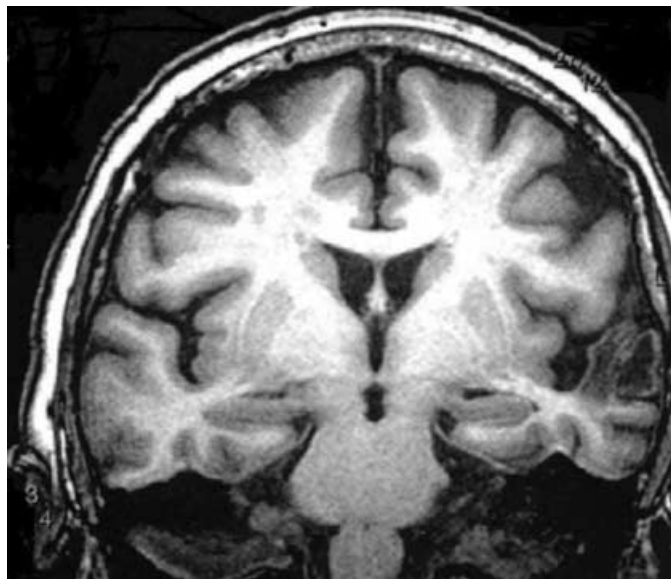
\*\*Moderate deficit.

\*\*\*Severe deficit.

results obtained on this second MT-86 test showed a similar pattern of deficits, but indicated that LA had substantially recovered from her initial severe aphasia. However, an additional neurolinguistic (unpublished) test based on a list of 80 French nonwords revealed that LA still showed clear deficits in her ability to repeat nonwords presented orally (she scored 49/80 compared to 76/80 in control subjects [standard deviation (SD) = 2.9]). This lower-than-normal performance confirmed LA's deficit in auditory analysis for verbal stimuli.

Figs. 3 and 4 show MRI scans (coronal and axial views) of LA performed in May 2002, that is, 10 months after CVA. T<sub>2</sub>-weighted images reveal the presence of an extended necrosis of brain tissues in the left sylvian area involving cortical and subcortical parts of the temporal lobe. The volume of the ischemic lesion (measured using the "Tissue\_Volume" software developed by the Neurological Dpt [HIA, Val de Grâce, Paris]) is 2.7 cc. The lesion includes the transverse temporal gyrus (Talairach *et al.*, 1967), in which most neuroanatomists locate the primary auditory cortex (Penhune *et al.*, 1996; the lesion includes the various localisations of the primary auditory cortex [e.g.,  $x \in (-25; -50)$ ;  $y \in (-15; -35)$ ;  $z \approx 12$ ] according to probabilistic maps [Penhune *et al.*, 1996]). The lesion also includes the planum temporal and the superior temporal gyrus, but spares the middle temporal gyrus and insula. Thus, the ischemic lesion affects both the primary and secondary auditory cortices.

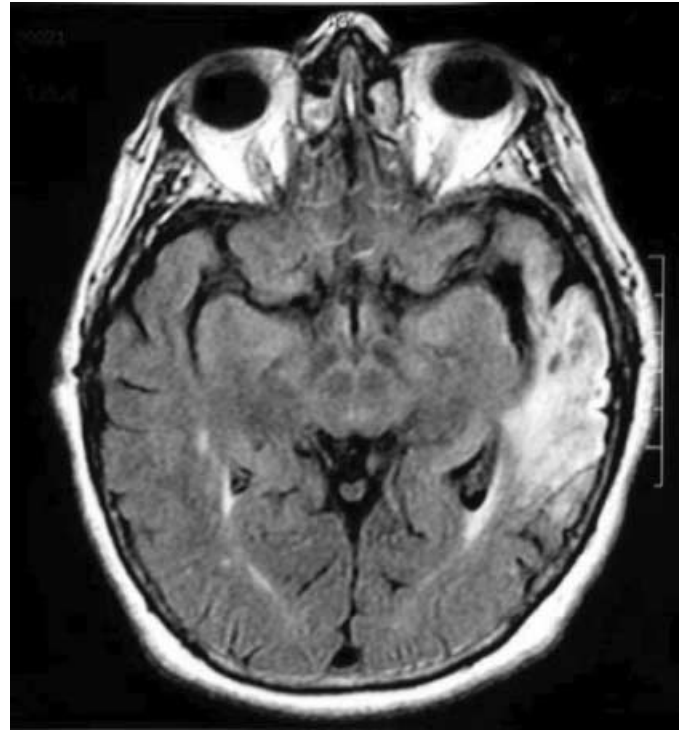
All psychophysical experiments were performed 1 year after the CVA. LA was neurologically stable during this period. Audiological assessment conducted at this time revealed that LA showed a bilateral, symmetrical high-



Right

Left

**Fig. 3.** Coronal MRI view of patient LA, indicating that the left primary and secondary auditory areas have been severely damaged due to the sylvian infarct.



Right

Left

**Fig. 4.** Horizontal MRI view of patient LA.

frequency cochlear hearing loss (see Table 2), presumably caused by presbycusis. It is important to note that LA's audiometric thresholds were within normal limits (i.e.,  $\leq 25$  dB HL) at 2 kHz in each ear. Three elderly normal-control listeners, JM (male, 66 years), RD (male, 71 years), and JB (female, 69 years; mean age: 69 years; SD: 3 years) also participated in the study. As for LA, audiometric thresholds at 2 kHz (RD [10 dB HL], JM [15 dB HL], and JB [25 dB HL]) were within normal limits for these control listeners. All listeners were fully informed about the goal of the present study, and provided written consent before their participation (all experiments were conducted in accordance with the Declaration of Helsinki).

**Table 2.** Audiometric thresholds for patient LA (i.e., air conduction thresholds in dB HL for the right and left ear). Bold characters highlight audiometric thresholds obtained at 2 kHz

Frequency (kHz)	Left ear (dB HL)	Right ear (dB HL)
0.2	10	15
0.5	15	15
1	10	15
<b>2</b>	<b>15</b>	<b>20</b>
3	20	20
4	30	40
6	30	40
8	55	50

## Stimuli and methods

The experiments were controlled by a PC. Stimuli were generated using a 16-bit D/A converter at a sampling frequency of 44.1 kHz, and were delivered diotically via Sennheiser HD 565 earphones at 75 dB SPL (rms). Listeners were tested individually in a quiet room. In both AM detection tasks, AM was applied over the full stimulus duration of 2 s, including 50 ms rise/fall times shaped using a raised-cosine function. The inter-stimulus interval was 1 s. To mask possible distortion products in the audio-frequency domain, all stimuli were presented in a low-pass filtered noise (cutoff frequency: 500 Hz; rolloff: 36 dB/oct).

### First-order TMTFs

Listeners had to detect the presence of sinusoidal AM applied to a 2 kHz pure-tone carrier. On each trial, a standard and a target stimulus were presented successively in random order. The standard,  $S(t)$ , was an unmodulated 2 kHz pure tone. The target,  $T(t)$ , was a 2 kHz pure-tone carrier which was sinusoidally amplitude-modulated at a given AM frequency. The expression describing the target's temporal envelope was:

$$T(t) = c[1 + m \sin(2\pi fm t + \phi)] \quad (1)$$

where  $m$  is the modulation depth ( $0 \leq m \leq 1$ ),  $fm$  is the modulation frequency ( $fm$  was 4, 16, 64, and 87 Hz), and  $\phi$  is the starting phase of the modulation, randomized on each interval. The term  $c$  is a multiplicative compensation term set such that the overall power was the same in both intervals. AM-detection thresholds were obtained using an adaptive two-interval, two-alternative forced-choice (2I, 2AFC) procedure with a two-down, one-up stepping rule that estimates the modulation depth  $m$  necessary for 70.7% correct detection. Listeners received visual feedback concerning the correct interval after each trial. The step size of  $m$  variation corresponded initially to a factor of 1.59, and was reduced to 1.26 after the first two reversals. The arithmetic mean of the value of  $m$  at the last 10 reversals in a block of 16 reversals was taken as the threshold estimate for that block (in percent). For each listener and for each modulation frequency, thresholds presented here are based upon three estimates.

### Second-order TMTFs

Listeners had to detect the presence of sinusoidal modulation applied to the depth of a sinusoidally amplitude-modulated pure-tone carrier. On each trial, a standard and a target stimulus were presented successively in random order to the listener. The standard,  $S(t)$ , consisted of a 2 kHz pure-tone carrier sinusoidally amplitude-modulated at a given AM frequency  $fm$ , with a fixed modulation depth  $m$  of 50% ( $m = 0.5$ ). The expression describing the standard's temporal envelope was:

$$S(t) = [1 + m \sin(2\pi fm t + \phi)] \quad (2)$$

where  $\phi$  represents the starting phase of the modulation, randomized on each interval. The target,  $T(t)$ , also consisted of a 2 kHz carrier. It was amplitude-modulated at a given modulation frequency  $fm$ , and its modulation depth was sinusoidally modulated at a given (2<sup>nd</sup>-order) AM frequency  $fm'$ . The expression describing the target's temporal envelope was:

$$T(t) = [1 + [m + m' \sin(2\pi fm' t + \phi')] \sin(2\pi fm t + \phi)] \quad (3)$$

where  $m'$  is the depth of modulation-depth variation ( $0 \leq m' \leq 0.5$ ),  $m$  is the "carrier" modulation depth ( $m = 0.5$ ),  $fm'$  is the frequency of modulation depth variation, and  $\phi'$  represents the starting phase of 1<sup>st</sup>- and 2<sup>nd</sup>-order modulations, randomized on each interval. The overall power was the same in both intervals. The AM carrier frequency  $fm$  was fixed at 64 Hz, and the 2<sup>nd</sup>-order AM frequency  $fm'$  was 4, 11 or 23 Hz. Second-order AM detection thresholds (i.e.,  $m'$  at threshold) were obtained using the same 2I, 2AFC psychophysical procedure with feedback as used to determine 1<sup>st</sup>-order AM detection thresholds.

### Intensity discrimination thresholds

Psychophysical investigation was completed by measuring intensity discrimination thresholds, that is, the ability of listeners to discriminate between two steady (i.e., unmodulated) 2 kHz pure tones presented at different intensities. This task was introduced as a control task for the 1<sup>st</sup>- and 2<sup>nd</sup>-order SAM-detection tasks because it allowed: (1) to evaluate the contribution of potentially confounding non-perceptual effects related to stimulus characteristics (e.g., stimulus duration) and the 2I, 2AFC procedure (e.g., the sequential mode of presentation of auditory stimuli), (2) to assess the contribution of amplitude resolution to the AM-detection thresholds measured at low modulation frequency, and (3) to dissociate the ability to perceive *steady* changes in amplitude from the ability to perceive *dynamic* changes in amplitude [see Lorenzi *et al.* (2000) for a similar use of intensity discrimination threshold]. On each trial, a standard and a target stimulus were presented successively in random order to the listener. The standard consisted of a 2 kHz pure tone presented at a reference intensity  $I$  giving a SPL of 75 dB. The target consisted of another 2 kHz pure tone presented at a greater intensity,  $I + \Delta I$ . Intensity difference limens were obtained using a 2I, 2AFC procedure that estimates the intensity difference  $\Delta I$  necessary for 70.7% correct responses. The listener's task was to determine the interval containing the louder sound. Listeners received visual feedback concerning the correct interval after each trial. The step sizes and thresholds ( $\Delta L$ ) were calculated in decibels ( $\Delta L = 10 \log[(I + \Delta I)/I]$ ). The step size of variation was initially 4 dB and was reduced to 0.25 dB after the first two reversals. The arithmetic mean of the last 10 reversals in a block of 16 reversals was taken as the threshold estimate for that block. Thresholds presented here are based upon three estimates.

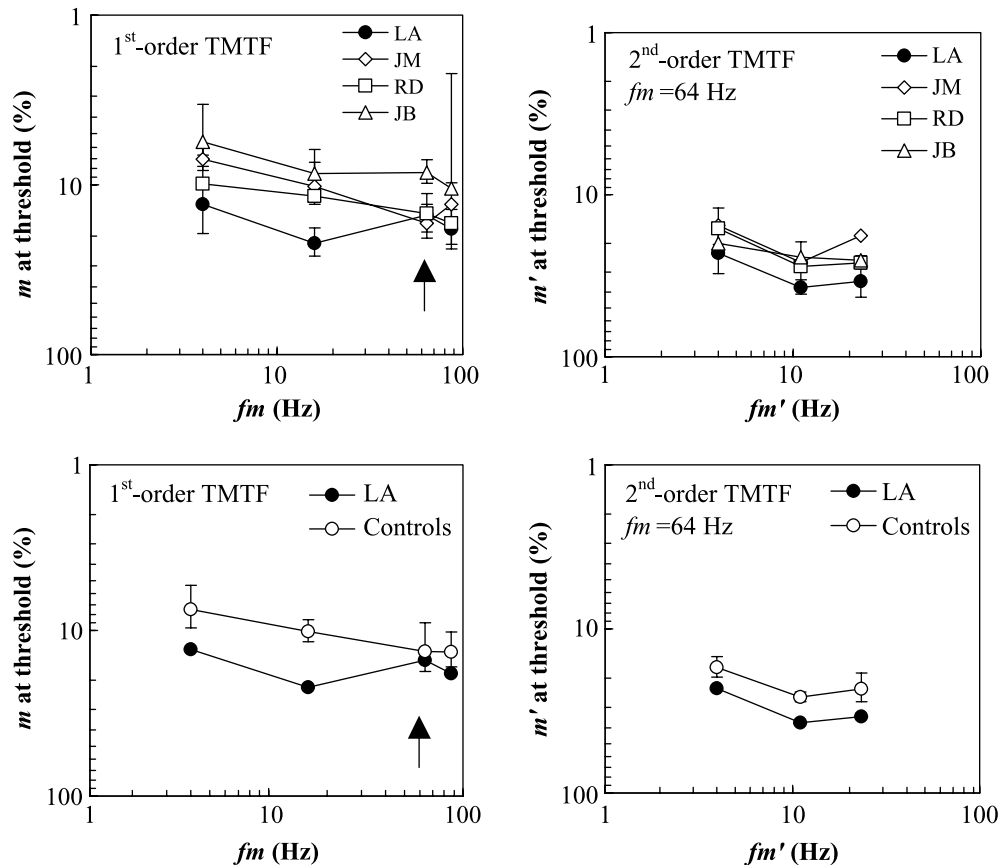
## Results

First- (left panels) and 2<sup>nd</sup>-order (right panels) AM detection thresholds for LA (filled circles) and the 3 control listeners (open symbols) are shown in Fig. 5. Results are plotted with worst thresholds towards the bottom of the ordinate, that is, 100 and 50% for 1<sup>st</sup>- and 2<sup>nd</sup>-order AM detection thresholds, respectively. Top panels present the individual data, and bottom panels show thresholds measured in LA plotted along with the average control data.

First-order TMTFs for controls are roughly low-pass in shape. On average, detection thresholds are smallest at the lowest AM frequency ( $fm = 4$  Hz); they worsen progressively when modulation frequency is increased up to 64 Hz and remain constant beyond this value. By comparison, the 1<sup>st</sup>-order TMTF measured in LA is rather flat in shape (although detection thresholds increase above 4 Hz).

Overall, detection thresholds for controls are higher than those obtained previously with similar pure-tone carrier and AM frequencies in young adults (Kohrausch *et al.*, 2000; Moore and Glasberg, 2001; Lorenzi *et al.*, 2001a; Füllgrabe

and Lorenzi, 2003). This probably reflects an effect of ageing on temporal envelope sensitivity, as shown previously in a study using noise carriers (Takahashi and Bacon, 1992). More precisely, the present control data are consistent (within a 2 dB range) with Takahashi and Bacon's (1992) detection data collected on 10 healthy listeners belonging to the (70–76 years) age group (mean age: 72 years). Fig. 5 also indicates that LA shows worse-than-normal 1<sup>st</sup>-order detection thresholds at the two lowest modulation frequencies of 4 and 16 Hz, where thresholds measured in LA are significantly increased by a factor of 2 compared to controls. At higher modulation frequencies (e.g.,  $fm = 64$  Hz), however, detection thresholds are very similar in all listeners (see Tables 3 and 4 for a detailed presentation of LA's 1<sup>st</sup>- and 2<sup>nd</sup>-order AM thresholds at each modulation frequency and control values, expressed as mean thresholds for the three age-matched control listeners with the 95% confidence interval). This is consistent with the results of two previous studies showing increased (1<sup>st</sup>-order) AM detection thresholds at low AM frequencies (<16 Hz) in an elderly patient (73 years) with



**Fig. 5.** First- (left panels) and 2<sup>nd</sup>-order (right panels) AM detection thresholds for LA (filled circles) and the 3 control listeners (open symbols). Top panels present the individual data, and bottom panels show thresholds measured in LA plotted along with the mean of the control data. In the case of 1<sup>st</sup>-order TMTFs, the ordinate indicates 1<sup>st</sup>-order modulation depth at threshold  $m$ , and the abscissa represents  $fm$ . In the case of 2<sup>nd</sup>-order TMTFs, the ordinate indicates 2<sup>nd</sup>-order modulation depth at threshold  $m'$ , and the abscissa represents  $fm'$ . In both cases, modulation depth at threshold is expressed in linear units, and plotted on a logarithmic scale. In the top panels showing individual data, error bars show one standard deviation about the mean of three threshold estimates. In the bottom panels showing the mean control data, error bars show one standard deviation about the mean across listeners. The 64 Hz 1<sup>st</sup>-order AM frequency is indicated by the upward-pointing arrow.

**Table 3.** First-order AM detection thresholds (in percent) at each 1<sup>st</sup>-order AM frequency ( $fm$ ; first column) for (1) patient LA (second column), and (2) control listeners (third column); control values are expressed as mean thresholds for the three age-matched control listeners with the 95% confidence interval

$fm$ (Hz)	LA mean thresholds (%)	Normal control listeners mean thresholds (95% confidence interval)
4	13	7.5% (5; 9.9)
16	22.1	10.1% (8.4; 11.9)
64	15.1	13.3% (8.4; 18.3)
87	18.2	15.5% (11.9; 19.1)

**Table 4.** Second-order AM detection thresholds (in percent) at each 2<sup>nd</sup>-order AM frequency ( $fm'$ ; first column) for (1) patient LA (second column), and (2) control listeners (third column); control values are expressed as mean thresholds for the three age-matched control listeners with the 95% confidence interval

$fm'$ (Hz)	LA mean thresholds (%)	Normal control listeners mean thresholds (95% confidence interval)
4	23	17.2% (14.4; 20)
11	37.4	26% (24; 28)
23	34.3	23.2% (18; 28.4)

Wernicke aphasia following left-hemisphere damage encroaching Heschl's gyrus (Hescot *et al.*, 2000) or in a patient showing bilateral hemispheric damage including the left Heschl's gyrus and the right frontal lobe (Griffiths *et al.*, 2000).

The right panels in Fig. 5 show that 2<sup>nd</sup>-order TMTFs for LA and control listeners are very similar in shape. Overall, detection thresholds are smallest at the lowest 2<sup>nd</sup>-order AM frequency ( $fm' = 4$  Hz); they worsen at  $fm' = 11$  Hz and remain constant beyond this value. Again, thresholds for control listeners are higher than those obtained previously with similar pure-tone carriers and 2<sup>nd</sup>-order AM frequencies in young adults (Lorenzi *et al.*, 2001a). As for 1<sup>st</sup>-order detection data, this probably reflects an effect of ageing on temporal envelope sensitivity. Although equal detection thresholds are observed in LA and controls for the 64 Hz 1<sup>st</sup>-order carrier AM, 2<sup>nd</sup>-order AM detection thresholds measured for LA are significantly increased by a factor of 1.3–1.5 compared to controls at all 2<sup>nd</sup>-order AM frequencies (see Table 4). When  $fm' = 11$  and 23 Hz, 2<sup>nd</sup>-order AM detection thresholds measured in LA get close to the maximum (i.e., worse) threshold (50%).

Intensity discrimination threshold measured in LA ( $\Delta L = 1.15$  dB) is within the normal range (95% confidence interval: [1–1.5 dB]; mean  $\Delta L = 1.26$  dB). This result indicates that: (1) nonperceptual effects related to the stimulus characteristics or the 2I, 2AFC procedure used in this study cannot account for the worse-than-normal 1<sup>st</sup>- and 2<sup>nd</sup>-order AM-detection thresholds measured in patient LA, and (2)

these worse-than-normal thresholds were caused by a deficit in temporal-envelope processing per se rather than a deficit in amplitude resolution.

## Discussion

The present single-case psychophysical study reveals that left-hemisphere damage affects the auditory ability to process slow simple (1<sup>st</sup>-order) and complex (2<sup>nd</sup>-order) temporal envelopes.

First, the present data confirm previous findings by showing that damage to the left primary and secondary auditory cortices may result in a *selective* deficit in auditory sensitivity to slow temporal modulations (i.e., AM frequencies below 16 Hz). This is consistent with previous electrophysiological and brain imaging studies demonstrating that, in mammals and humans, neurones in primary and secondary auditory cortical areas respond preferentially to low (<30–40 Hz) AM frequencies (e.g., Schreiner and Urbas, 1986, 1988; Eggermont, 1994; Eggermont and Smith, 1995; Kilgard and Merzenich, 1999; Giraud *et al.*, 2000). For instance, the f-MRI study conducted by Giraud *et al.* (2000) with human subjects shows strong responses to low AM frequencies (4–8 Hz) in Heschl's gyrus (BA41), and in regions lateral and posterior to Heschl's gyrus, that is, in secondary auditory areas situated in the superior temporal sulcus (BA22) and externally on the lateral surface of the superior temporal gyrus (BA42). This is also compatible with the results of a similar single-case report showing impaired modulation detection at low modulation frequencies (<16 Hz) in a Wernicke aphasic patient with lesions involving the left primary and secondary auditory cortical areas (Hescot *et al.*, 2000). Of course, it is important to note that the present single-case study does not allow to draw any firm conclusion about the lateralization of the mechanisms involved in temporal-envelope processing; a cohort of patients with right and left hemisphere damage will have to be tested in a future study in order to address this issue. Moreover, it is worth noting that this result does not imply that there are neurons tuned primarily for the detection of 2<sup>nd</sup>-order AM. Neurons in the auditory cortex may have complex receptive fields that happen to be sensitive to complex patterns of modulation, but are not specifically tuned for their detection (e.g., deCharms *et al.*, 1998).

Many psychoacoustical and behavioural studies have suggested that temporal modulations carry important information and have a communicative meaning (e.g., Shannon *et al.*, 1995; Smith *et al.*, 2002). Moreover, a number of psychoacoustical studies have demonstrated that the AM frequencies crucial for speech intelligibility range from 4–16 Hz (e.g., Houtgast and Steeneken, 1985; Shannon *et al.*, 1995). The reduced ability of patient LA to perceive temporal-envelope cues below about 30 Hz is therefore compatible with her aphasic profile (and especially her deficit in auditory analysis for verbal stimuli).

It is possible to simulate the effects of a loss of 1<sup>st</sup>-order AM sensitivity on speech identification performance. Consistent with the present findings, previous studies have clearly



shown that degrading speech by filtering out low AM frequencies (<16 Hz) leads to poorer speech identification performance in normal-hearing listeners (e.g., Drullman *et al.*, 1994a, b). However, whether such an approach based on envelope filtering could be used to simulate the effects of a loss of 2<sup>nd</sup>-order AM sensitivity on speech identification performance has not yet been demonstrated.

The present data reveal an interesting dissociation between the ability to detect 1<sup>st</sup>- and 2<sup>nd</sup>-order temporal envelope cues: LA shows poorer-than-normal ability to detect 2<sup>nd</sup>-order AM at frequencies ranging from 4–23 Hz, but normal ability to detect the 64 Hz (1<sup>st</sup>-order) AM carrying these 2<sup>nd</sup>-order AMs. This result reveals that damage to the left primary and secondary auditory cortices affects the ability to detect temporal variations in the *local* properties of sounds (such as AM depth). By comparison, peripheral (i.e., cochlear) damage does not affect such an ability (Tandetnik *et al.*, 2001; Füllgrabe *et al.*, 2003).

This result is also consistent with the notion that, as in vision, nonlinear mechanisms along the auditory pathway generate an audible distortion component at the 2<sup>nd</sup>-order AM frequency  $fm'$  in the internal (sensory) modulation spectrum of the stimuli. In agreement with the present data, similar deficits are expected for the detection of 1<sup>st</sup>- and 2<sup>nd</sup>-order AM components at common frequencies below about 30 Hz if: (1) as suggested above, cortical lesion affects the central mechanisms involved in the detection of low 1<sup>st</sup>-order AM components (below about 30 Hz); and (2) a nonlinearity introduces a 1<sup>st</sup>-order component into the sensory representation of 2<sup>nd</sup>-order stimuli. The present results are also consistent with the notion that the main source of distortion occurs at a post-cochlear stage. However, it is not possible to conclude from the current data whether this distortion occurs before or at the auditory cortex level.

Two recent psychoacoustical studies conducted with young normal-hearing listeners (Füllgrabe and Lorenzi, 2003; Millman *et al.*, 2003) argued against the existence of distortion products in the temporal-envelope domain by suggesting that the detection of 2<sup>nd</sup>-order AM is based on the detection of a slow envelope beat cue appearing at the output of hypothetical “modulation filters” (e.g., Dau *et al.*, 1997) tuned to or near the carrier frequency  $fm$  (these slow envelope beat cues would be caused by the *linear* interaction of the carrier and sideband AM components in these broadly tuned filters). The present results appear inconsistent with this alternative approach because normal ability to detect the 64 Hz (1<sup>st</sup>-order) AM carrying the 2<sup>nd</sup>-order AMs should have led to normal ability to detect the slow envelope beat cue appearing at the output of the modulation filter tuned to  $fm = 64$  Hz. Of course, it cannot be excluded that LA showed parallel deficits for detecting 1<sup>st</sup>- and 2<sup>nd</sup>-order AM components at common frequencies below 30 Hz because the cortical lesion affected both: (1) the central mechanisms involved in the detection of low 1<sup>st</sup>-order AM components (below about 30 Hz), and (2) the central mechanisms involved in the detection of the slow envelope beat cues appearing at the output of modulation

filters. This alternative hypothesis – although less parsimonious than the initial one based on nonlinear processes – might explain why the increase in 2<sup>nd</sup>-order AM detection threshold (by a factor of 1.3–1.5) caused by cortical lesion was smaller than that observed for 1<sup>st</sup>-order AM detection (increase in thresholds by a factor of 2). On the other hand, the fact that 2<sup>nd</sup>-order AM detection was not degraded more severely by cortical lesion is consistent with the notion that perception of a distortion product at the envelope beat frequency contributes only partially to 2<sup>nd</sup>-order AM detection.

## Acknowledgements

C Füllgrabe is supported by a MENRT grant. C Lorenzi is supported by the Institut Universitaire de France. The authors are indebted to C Kervella for developing the psychoacoustical software used in the present study, and to C Nioche and YS Cordoliani (Radiology Dpt, HIA Val de Grâce, Paris) for their help in MRI data analysis.

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