Specialization of left auditory cortex for speech perception in Man depends on temporal coding

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Speech perception requires cortical mechanisms capable of analysing and encoding successive spectral (frequency) changes in the acoustic signal. To study temporal speech processing in the human auditory cortex, we recorded intracerebral evoked potentials to syllables in right and left human auditory cortices including Heschl's gyrus (HG), planum temporale (PT) and the posterior part of superior temporal gyrus (area 22). Natural voiced (/ba/, /da/, /ga/) and voiceless (/pa/, /ta/, /ka/) syllables, spoken by a native French speaker, were used to study the processing of a specific temporally based acoustico-phonetic feature, the voice onset time (VOT). This acoustic feature is present in nearly all languages, and it is the VOT that provides the basis for the perceptual distinction between voiced and voiceless consonants. The present results show a lateralized processing of acoustic elements of syllables. First, processing of voiced and voiceless syllables is distinct in the left, but not in the right HG and PT. Second, only the evoked potentials in the left HG, and to a lesser extent in PT, reflect a sequential processing of the different components of the syllables. Third, we show that this acoustic temporal processing is not limited to speech sounds but applies also to non-verbal sounds mimicking the temporal structure of the syllable. Fourth, there was no difference between responses to voiced and voiceless syllables in either left or right areas 22. Our data suggest that a single mechanism in the auditory cortex, involved in general (not only speech-specific) temporal processing, may underlie the further processing of verbal (and non-verbal) stimuli. This coding, bilaterally localized in auditory cortex in animals, takes place specifically in the *left* HG in man. A defect of this mechanism could account for hearing discrimination impairments associated with language disorders.

Introduction

Language perception is essentially based on subtle differences in the timing of acoustic elements of the speech signal. Many neurological observations in aphasic patients have shown that disorders in auditory language perception are linked to an impaired processing of time-related (temporal) information (Efron, 1963; Robin *et al.*, 1990). It has been postulated that an enhanced capacity to analyse and encode successive temporal changes in the acoustic signal, requiring cortical mechanisms, may underly the left hemisphere's contribution to speech processing (Tallal and Piercy, 1973; Tallal and Newcombe, 1978). These authors demonstrated that a deficit in the processing of the rate of change of acoustic cues, such as the formant transitions, rather than a deficit in the linguistic nature of the stimuli, could explain phonological disorders observed in language-learning-impaired children. This hypothesis seems to be confirmed by the improvement of speech discrimination and language understanding in these children after a period of intensive behavioral training consisting of recognition of brief and fast sequences of speech and non-speech stimuli (Merzenich *et al.*, 1996; Tallal *et al.*, 1996). Since disturbances in temporal processing can create deficits in language-learning-impaired children (Schwartz and Tallal, 1980), this temporal processing is likely to be a fundamental mechanism in sound perception which could be supported by cortical processing involving the auditory cortex.

One of several temporal cues used in perception and discrimination of stop consonants is the voice onset time (VOT). Lisker and Abramson (Lisker and Abramson, 1964) have shown that the voicing and aspiration differences among stop consonants in a wide variety of languages can be characterized by changes in VOT, which, in turn, reflect differences in the timing of glottal activity relative to supralaringeal events. It has been assumed that the perception of the VOT is under the control of the left hemisphere (Liberman *et al.*, 1952; Lane, 1965, Fujisaki and Kawashima, 1971; Studdert-Kennedy, 1976; Ades, 1977; Miller, 1977; Pastore *et al.*, 1977; Stevens, 1981; Kuhl and Padden, 1983; Macmillan, 1987). Several studies, however, have reported slight deficits in VOT discrimination in patients with damage to the left hemisphere (Oscar-Berman *et al.*, 1975; Basso *et al.*, 1977; Blumstein *et al.*, 1977; Miceli *et al.*, 1978; Itoh *et al.*, 1986). Electrophysiological data suggest that the perception of the VOT is controlled by several cortical processes – some of which are restricted to the right hemisphere and others of which are common to both hemispheres (Molfese, 1978). A question which we deal with this study concerns the nature of the VOT cue itself. Is VOT processed by specialized speech mechanisms or by more basic acoustically tuned cortical mechanisms (Pisoni, 1977)?

Recording of cortical auditory evoked potentials (AEP) to speech sounds represents in humans a physiological approach to study the neural activity that underlies the discrimination of speech sounds. AEP studies have actually demonstrated that the auditory cortex of mammals is able to encode precisely acoustic information which changes over time (McGee *et al.*, 1996) and have shown that primary auditory cortex evoked responses reflect encoding of VOT (Steinschneider *et al.*, 1982, 1994, 1995). In prior investigations, we recorded evoked potentials intracerebrally in auditory cortex during presurgical exploration of patients who were candidates for a cortectomy for the relief of intractable epilepsy. Those recordings have shown that those auditory areas reported in several anatomical studies (Brodmann, 1909; Braak, 1978; Galaburda and Sanides, 1980) to be morphologically distinct are also functionally distinct. Studies of the intracortical localization of AEP generators have shown an anatomical segregation of components according to their latencies along Heschl's gyrus (HG), demonstrating that earlier components (from 13 to 50 ms) originated from the dorso-postero-medial part of HG (primary cortex) and later ones (from 60 ms) originate from the lateral part of HG and planum temporale (PT, secondary cortex) (Liégeois-Chauvel *et al.*, 1991, 1994).

In this study, we examine the neural responses to speech and

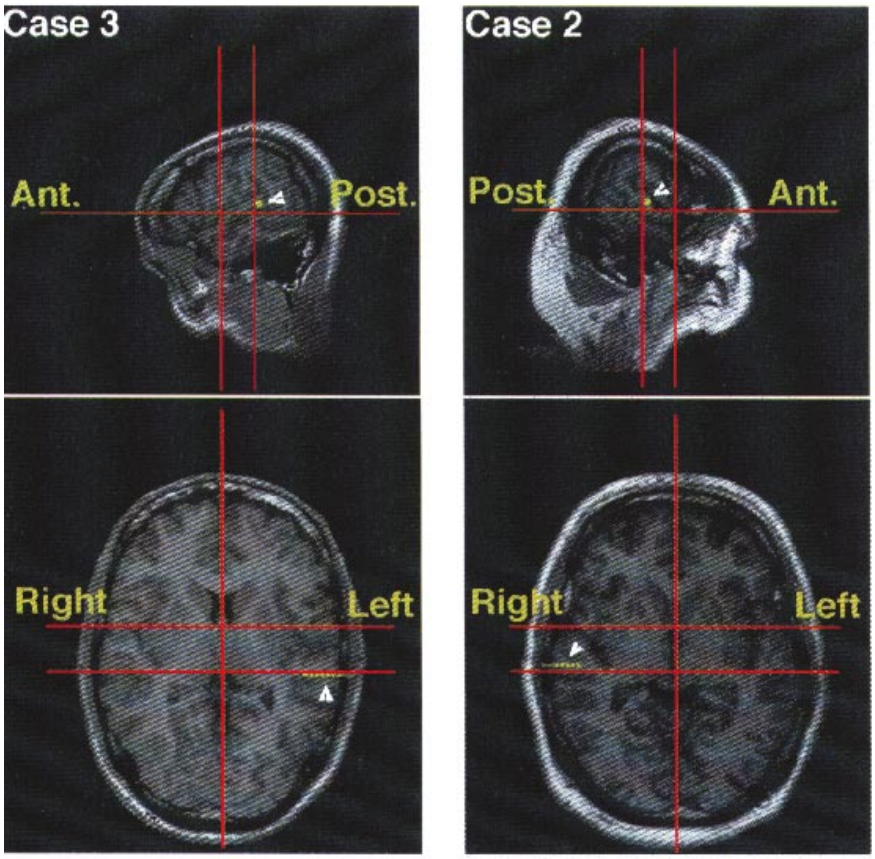

Figure 1. Saggital (upper part) and axial (lower part) MRI slices for cases 2 and 3. The electrode tracks were reconstructed and are indicated by the yellow dots (saggital slices) and yellow-green bars (axial slices) near the white arrowheads.

non speech-sounds within the cortical auditory areas such as HG, PT and the anterior region to HG corresponding to area 22 (Brodmann, 1909). We focus on the stop consonants to investigate the nature of the temporal mechanisms involved in consonant perception. In particular, we seek to assess the degree to which the acoustic versus phonetic nature of the VOT is coded by the different auditory areas, leading to cortical lateralization in the perception of stop consonants.

Materials and Methods

Subjects

Seventeen epileptic patients (eight male, nine female, aged 17–38 years) participated to this study. It is important to note that these patients have been *a posteriori* selected in such a way that none of them had their epileptogenic zone including the auditory areas, and no patient showed atypical language representation during specific examinations. Recordings of brainstem evoked potentials carried out before the stereoelectroencephalography (SEEG) confirmed that all the patients had normal afferent pathway conduction.

All patients were informed about the research protocol during the SEEG and gave consent (Ethical Committee, Rennes, November 15, 1993).

Presurgical Exploration

The presurgical SEEG (Bancaud *et al.*, 1965; 1992; Chauvel *et al.*, 1996) of patients with temporal, temporo-parietal or temporo-frontal epilepsy requires implantation of multiple depth electrodes in the temporal lobe and neighbouring areas of adjacent lobes; its purpose is to determine the anatomical structures involved in the initiation and propagation of seizures and the accurate limits of future cortical excision (Talairach *et al.*, 1974). In addition to the cases where the posterior areas of the superior temporal gyrus (STG) are suspected of constituting the whole or part of the epileptogenic zone, this region is frequently explored because of its strategic position as a pathway for ictal discharge propagation towards inferior parietal and opercular regions (Bancaud and Chauvel, 1987), as well as for the necessity for functional mapping before surgery.

Anatomical Definition of Depth Electrode Position

The multilead electrodes (0.8 mm diameter, 5–15 contacts, 2 mm length, 1.5 mm apart) are introduced orthogonally through the usual double grid system fastened to the Talairach stereotaxic frame. Anatomical localization of each lead of the electrode is based on a stereotaxic method which has been described elsewhere (Szikla *et al.*, 1977; Talairach and Tournoux, 1988; Liégeois-Chauvel *et al.*, 1991). Owing to the oblique orientation of HG, a single electrode can explore different auditory areas (medial and /or lateral part of HG, and /or PT and/or area 22; see Table 1).

We could verify that the selected anatomical structures had indeed been explored by visualization of the electrode tracks through stereotactic magnetic resonance imaging (MRI) (Fig. 1).

Figure 2 shows a typical brain (from an MRI) in a sagittal (Fig. 2*A*,*B*) and frontal view (Fig. 2*C*). Figure 2*A*,*B* shows the entrance points of the electrodes used to explore the left and right STG; the numbers on the points correspond to the cases listed in Table 1. Figure 2*C* shows the points at which the leads of the electrodes were most responsive, in selected cases, during the exploration of the posterior portion of the STG.

Stimuli

The auditory stimuli used were speech sounds and speech *analogue* sounds.

Speech Sounds

We used stop consonants (/ba/, /da/, /ga/, /pa/, /ta/, /ka/) pronounced and recorded by a native French speaker in a sound-attenuated room. It is

Table 1

Case	Sex	Age	Handedness	MRI	Anatomical location of leads
1	M	31	R	–	L HG: medial part 1 lead; lateral part 3 leads
2	M	32	L[a]	normal	R HG: medial part 1 lead; lateral part 3 leads
3	M	23	L[a]	normal	L HG: medial part 1 lead; lateral part 5 leads
4	F	38	R	normal	R HG: lateral part 3 leads
5	M	21	R	left inferior frontal gyrus lesion	L HG: medial part 2 leads; lateral part 1 lead L PT: 3 leads:
6	F	31	R	N	R PT: 4 leads
7	M	38	R	–	L area 22: 3 leads
8	M	38	R	right parietal lesion	R area 22: 3 leads
9	F	23	R	normal	L HG: medial part 1 lead; lateral part 3 leads
10	F	30	R	right hippocampal sclerosis	R HG: medial part 2 leads
11	F	37	L[a]	normal	L HG: Lateral part 5 leads
12	M	17	R	left lateral occipito-temporal gyrus lesion	L HG: lateral part 3 leads
13	F	27	L[a]	–	L HG: lateral part 1 lead L area 22: 3 leads
14	F	18	L[a]	left medial temporal gyrus lesion	L HG: lateral part 2 leads L area 22: 2 leads
15	M	34	R	right post.cingulate lesion	R area 22: 3 leads
16	F	32	R	right temporo-sylvian lesion	R HG: lateral part 3 leads R area 22: 3 leads
17	F	21	R	left para-hippocampal gyrus lesion	L PT: 4 leads

L, left; R, right; HG, Heschl's gyrus; PT, planum temporale.

[a]An intracarotidal amytal test (Wada test) has been performed in the left-handed patients. All of them had a left hemisphere dominance for language.

possible to describe stop voiced consonants in terms of a sequence of acoustic segments. These segments appear on the spectrogram as a series of relatively homogeneous stretches of the signal demarcated by rather abrupt changes in spectral form, marking the boundary between segments (Fant, 1973). In French, the VOT (i.e. the temporal relationship between the release burst in a stop consonant and onset of glottal pulsing) of the voiced consonants (/b/, /d/, /g/) is negative, preceding by ~110 ms the release burst, whereas for voiceless consonants (/p/, /t/, /k/), the VOT is positive, following the release burst (20 ms) (left side of Fig. 3). Note that there are timing differences in production and perception of VOT across different languages; for English voiced stops, voicing precedes or is nearly coincidental with release (Lisker and Abramson, 1964).

The three voiced syllables (VS) (/ba/, /da/, /ga/) had a VOT of ~110 ms and a total length of ~360 ms. The three voiceless syllables (VLS) (/pa/, /ta/, /ka/) had a length of ~200 ms. The syllables were presented to all the patients over headphones in a pseudo-random order. The stimulus intensity was adjusted to 70 dB SL (sensation level, relative to the threshold at 1000 Hz).

Acoustic Speech Analogues

The four acoustic speech analogues used are displayed at the right side of Figure 3. *VOTa*, the sound mimicking the voicing, lasts 110 ms and contains three low frequencies (200, 400, 600 Hz). *VLa-short*, the complex-sound analogue to a vowel, reflects the vowel's spectral content (200, 400, 600, 800, 1500, 2700, 4100 Hz) and duration (223 ms). *Va* mimicks the syllable's temporal course: it has 110 ms of near silence followed by the seven stronger frequencies present in the two previous complex sounds for a total duration of 320 ms. *VLa-long* has the same spectral components as Va but without the near silence (pre-voicing) at the beginning; its total duration is 400 ms.

All the patients had heard VOTa and VLa-short sounds and either Va or VLa-long sounds, delivered in a pseudo-random order in the same series.

Recordings

We recorded from 65 different sites of recording in auditory areas: 14 and 23 leads in the right and left HG respectively, 4 and 7 leads in the right and left PT, and 9 and 8 leads in right and left area 22, located anteriorly to HG.

The recordings of intracerebral AEPs were monopolar, with each lead

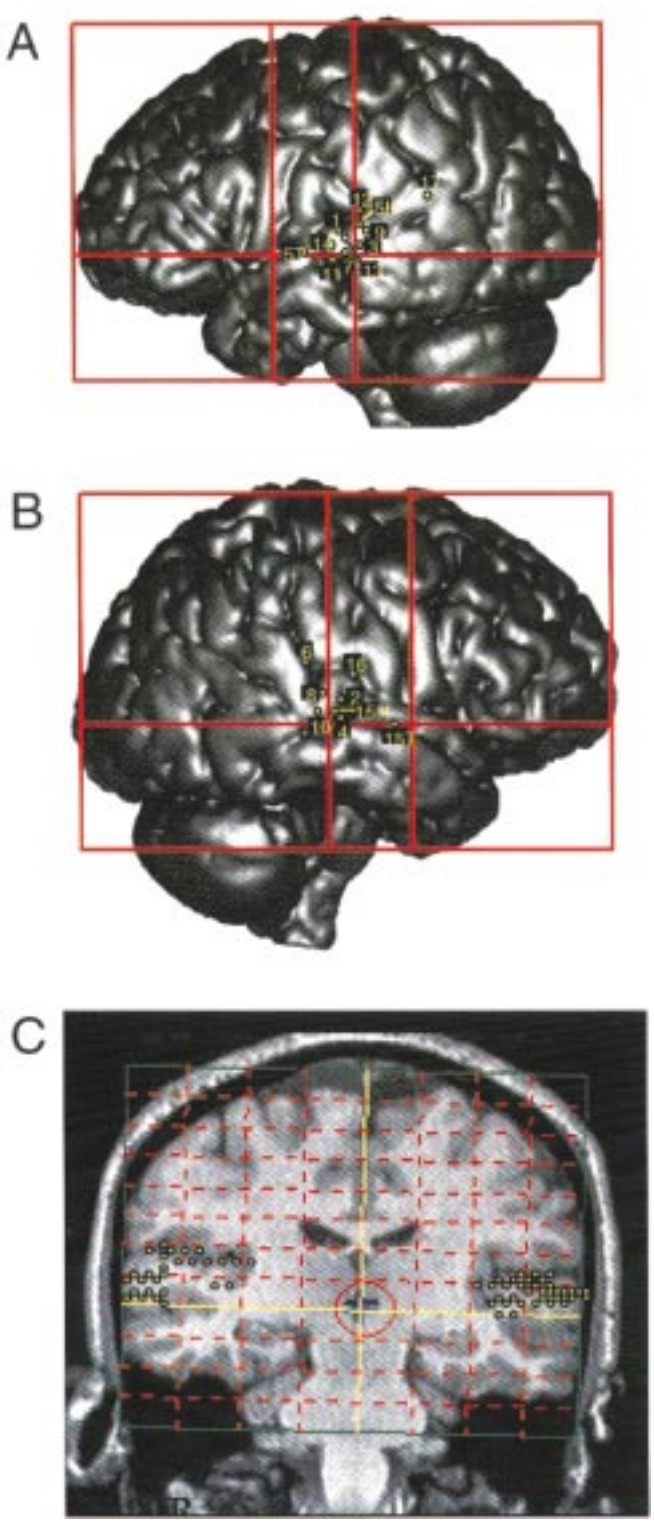

Figure 2. Anatomical location of depth electrodes in the left (*A*) and right (*B*) superior temporal gyrus. The distribution of some leads exploring HG is displayed in (*C*) (see comments in the text and Table 1).

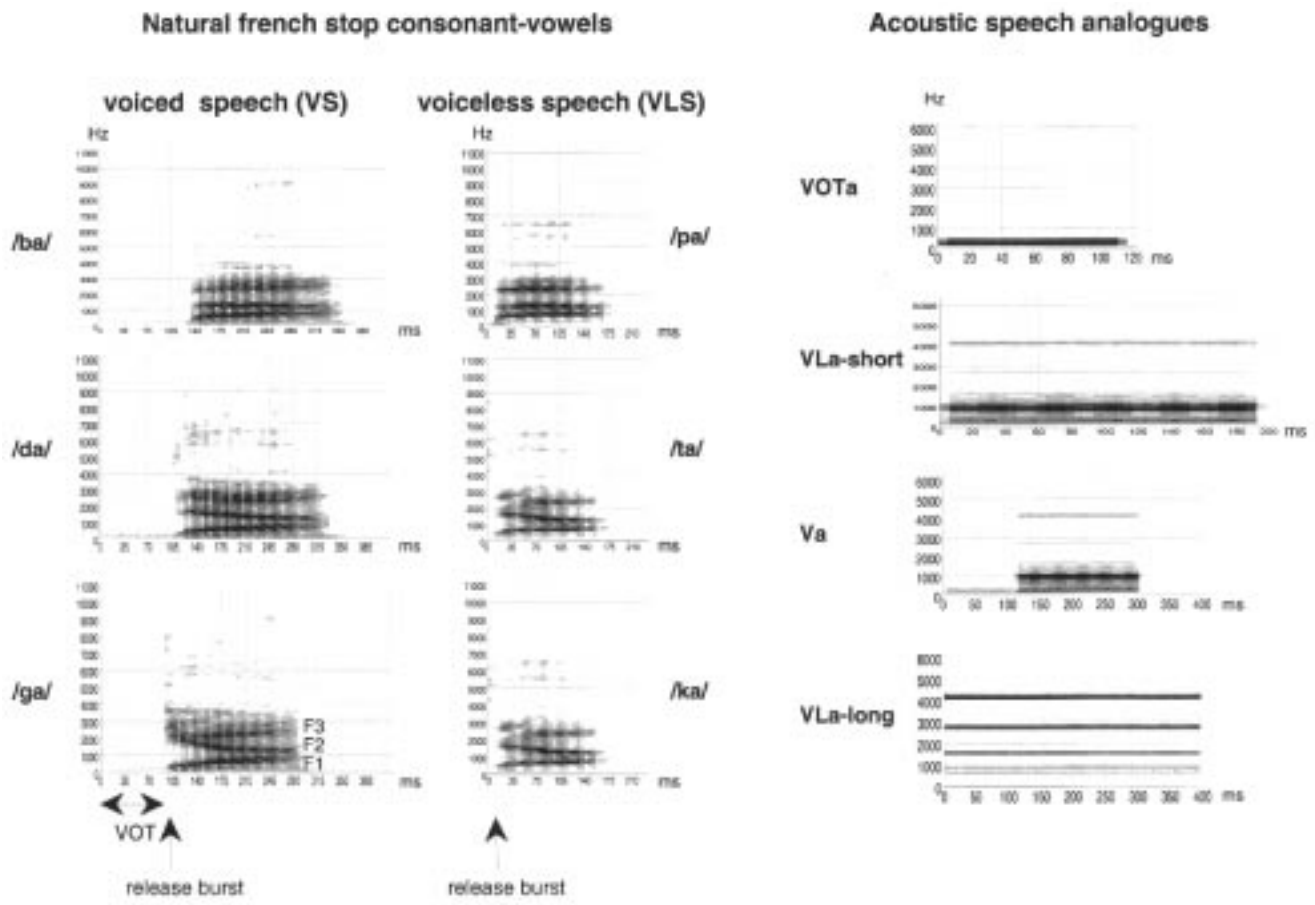

Figure 3. (Left) Spectrogram of natural syllables pronouced by a French female. *y*-axis represents frequency (Hz), *x*-axis time (ms), and the darkness shows the formants of vowel /a/. The VOT is measured from the release burst. It is negative when voice precedes the release burst and positive when it follows it. For French voiced consonants, VOT is always negative, varying from 100 to 120 ms according to the consonant. Conversely, VOT is positive with a value of ~20 ms for voiceless consonants. (Right) Spectrogram of acoustic speech analogues (see comments in the text).

of each depth electrode referenced to an extra-dural lead. All the signals were amplified and filtered simultaneously (bandpass 1 Hz–1.5 kHz). Data acquisition started 82 ms before the presentation of the sound and lasted for 819 ms. Average waveforms (100 trials) were digitally low-pass filtered using a Butterworth simulation (cutoff frequency 175 Hz, slope 48 dB/octave).

During a recording session, each patient laid comfortably in a chair in a sound-attenuated room and listened passively to the sounds.

Results

Recordings in the Left HG

No difference was found in AEPs among the three VS nor among the three VLS. Therefore, we present the grand average of AEPs in response to voiced versus voiceless stimuli at different recording sites.

Figure 4*A–H* illustrates the pattern of AEPs recorded from eight selected leads at various sites in the left HG to the VS (solid curves) and VLS (dashed curves). The basic sequence of cortical activation in response to both sets of stimuli was similar up to 170 ms mean latency. However, two main features of the intracortical responses are modified by VOT. First, the response seemed to be more complex for VS than for VLS. Except for the earliest components recorded exclusively in the primary cortex, six main successive components (48, 93, 168, 207, 262 and 353 ms mean latency) are recorded in response to VS and only five to VLS (45, 88, 163, 216, 292 ms).

Second, the processing of the VS in the left HG is sequential in nature. There are prominent evoked responses (N/P48, N/P93) at the onset of the voiced consonants. The next response (N/P170) is time-locked with the release burst of the consonant, followed by the responses N/P207 and N/P262, which correspond to the processing of the last part of the syllable. The last component, a slow-wave positive component peaking around 353 ms, marks the end of the response. Note that the interval separating the early responses and the N/P170 equals the duration of VOT.

Occasionally, the offset of the sound evoked a response that peaked at 277 ms for VLS and at 420 ms for VS.

Interestingly, multiple low-voltage components superimposed over the main components were seen often for VS and occasionally for VLS. These oscillatory components appeared during the formant transitions after the burst and lasted almost the duration of the vowel (Fig. 4*A*,*F*). The power spectra of these responses peak at a frequency of ~80 Hz.

Recordings in the Right HG

In the right HG (Fig. 5), VS and VLS elicited response patterns that were similar in the order and duration of response components. Components always present for both sets of stimuli were at 46, 84, 173 and 283 ms (mean latency). The shape of responses depended on neither the duration nor the nature of the stimulus. Thus, the sequential processing of the different

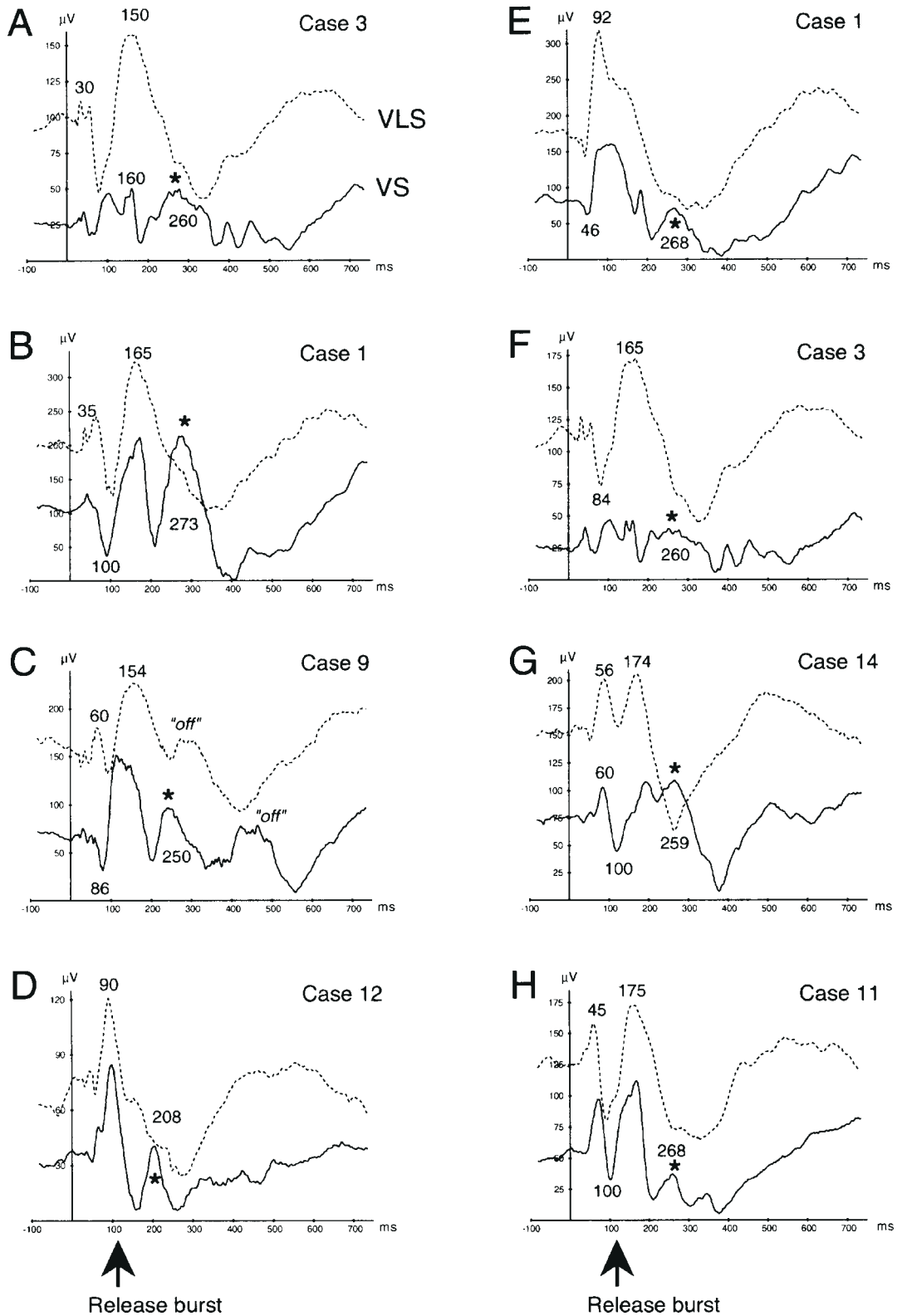

Figure 4. Auditory evoked potentials (AEPs) recorded at differents sites within the left HG in response to voiced (VS, full curve) and voiceless (VLS, dashed curve) syllables. The asterix indicates the additional component of the evoked sequence that is specifically recorded in response to VS. It peaks at 262 ms mean latency (see Table 1 and comments in the text). The arrow indicates the latency of the release burst. Negative polarity upwards.

parts of VS observed in the left HG is not found in the right HG. Morever, there are no oscillatory responses

Recordings in PT

We analyzed the evoked responses to VS and VLS in the left and

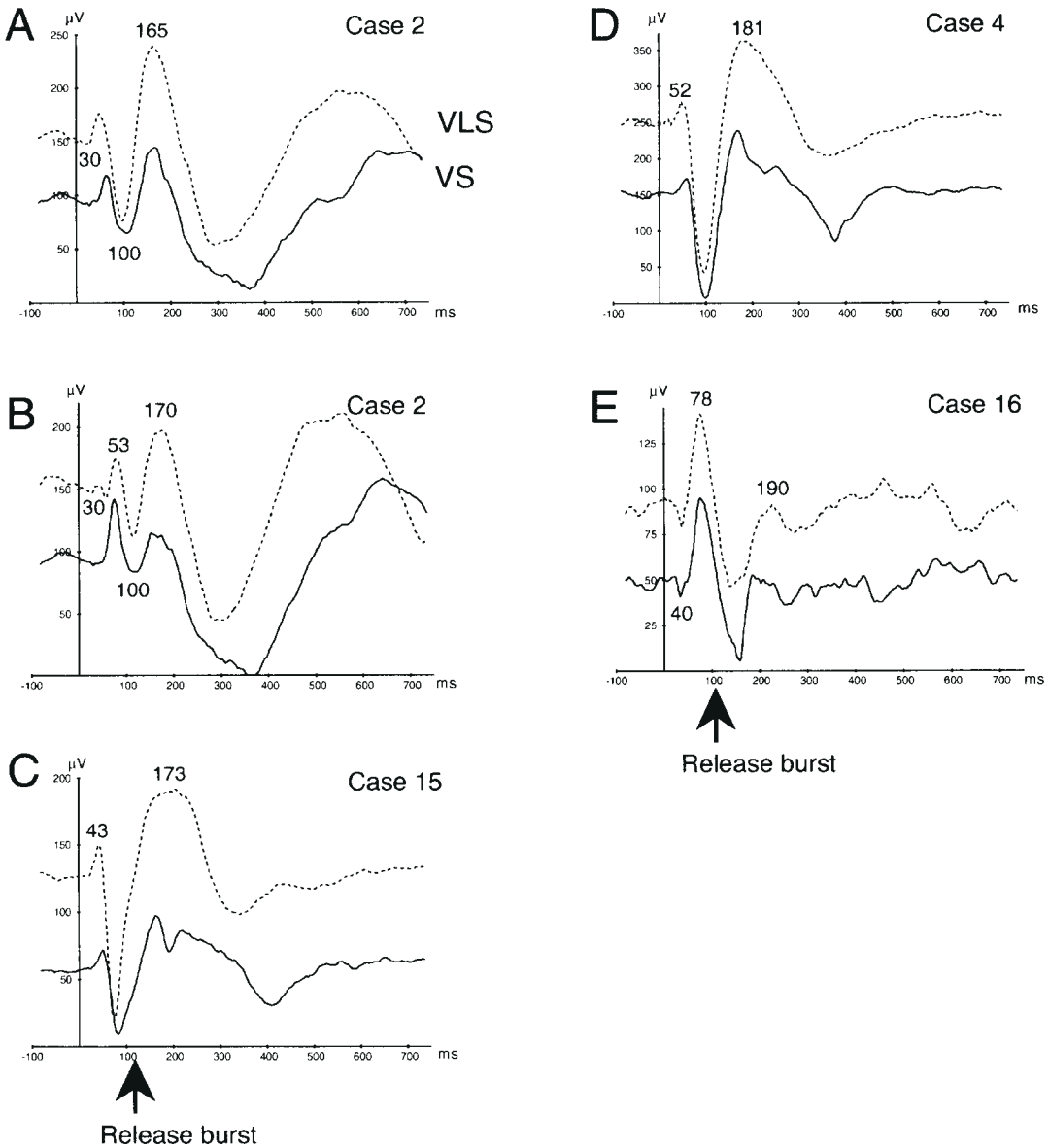

Figure 5. AEPs recorded in the right HG. See the legend of Figure 4 and comments in the text.

right PT and area 22 in order to see if the asymmetry of processing is preserved in these regions.

In the left PT (Fig. 6*A*,*B*), evoked responses to VS (full curves) were similar to those recorded in HG, and the same successive components were observed (70, 96, 161, 221, 252, 337 ms mean latency). The first component peaked later than the first one found in HG. The temporal processing of VOT and the vowel seemed to be preserved. However, the difference in the morphology of evoked reponses between VS and VLS was less clear. In some cases (Fig. 6*A*), a large slow wave culminating between 500 and 600 ms after stimulus onset could be recorded.

Figure 6*C* shows the responses recorded in the right PT. No difference is observed in waveforms of evoked responses to both stimuli.

Recordings in Area 22

The results of recordings in left and right area 22 are displayed in Figure 7*A–F*. For both VS and VLS, responses had a very similar waveform, a triphasic potential in which the latencies of different components varied as a function of the precise position of the electrode. Any sequential processing did not emerge from these data, as similar AEPs were recorded for both types of syllables

Tables 2 and 3 sum up data obtained from recordings in all the patients. For clinical considerations and owing to individual anatomical variations, the number of leads and the placement of the electrodes differed across patients. Therefore, a grand average of responses recorded at different leads, as is often done with scalp recordings, would be of little significance. Nonetheless, we have labelled the peak latencies of the main components evoked by both types of syllables on the 65 responsive leads in order to check the validity of this temporal coding. The mean latencies and SDs of peaks are summarized in Table 2 for left and in Table 3 for right auditory areas. The reproducibility of the AEPs is manifested in the relatively small SDs, which varied from 5% for the short latencies to 20% for the long latencies.

As we could observe in selected recordings displayed in

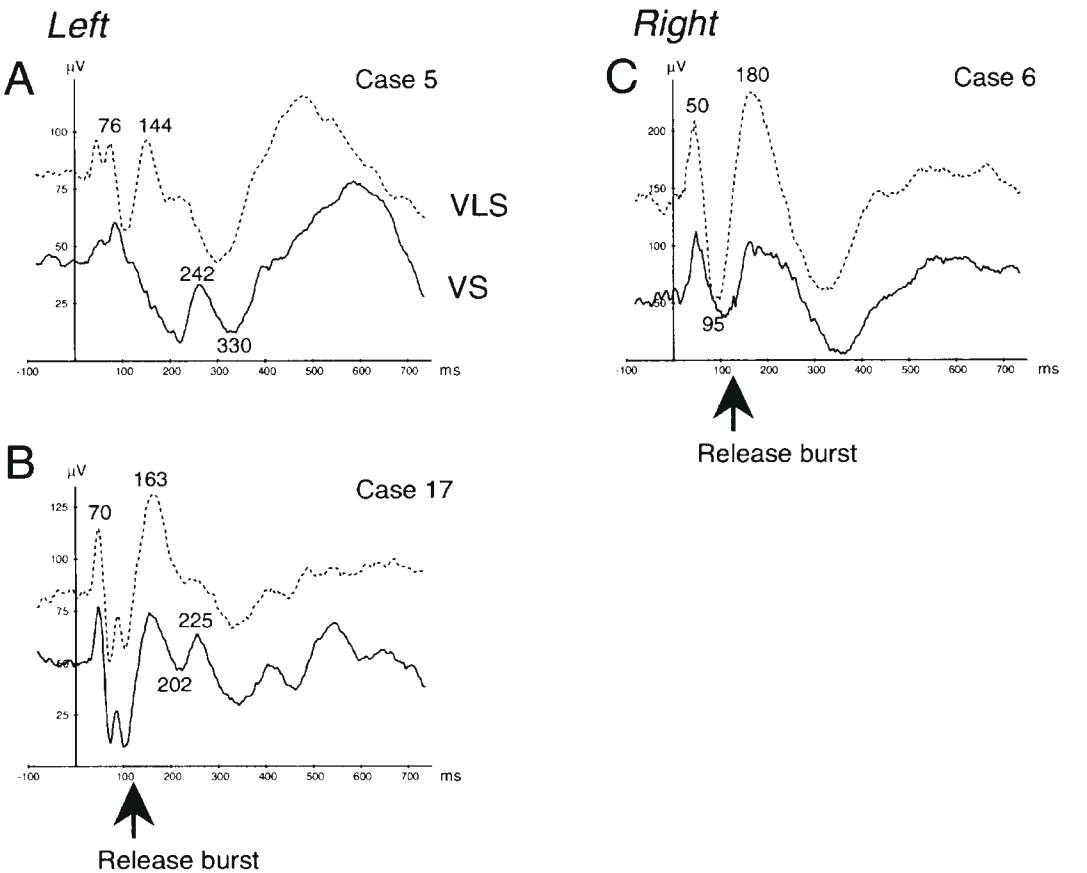

Figure 6. AEPs recorded at differents sites within left and right PT in response to VS (full curve) and VLS (dashed curve). See legend of Figure 4 and comments in the text.

Table II

Mean latency (± SD) of the successive positive and negative main components evoked by voiced (normal type) and voiceless (bold type) syllables recorded in different anatomical structures in the left auditory cortex

Left side	Primary components						*	Last component
HGm	25.4 ± 2.7	33.7 ± 2.5	44 ± 2.9	89.2 ± 7.7	166 ± 6.3	204.5 ± 13.5	262.3 ± 15.9	347.8 ± 32.3
	25.4 ± 2.7	**33.7 ± 2.5**	**42.7 ± 2**	**87.7 ± 8.50**	**157.6 ± 6.5**			**288 ± 36**
HGl			51.7 ± 8.7	97.4 ± 8.8	170.6 ± 19.3	209.5 ± 16.8	262.4 ± 7.4	359.7 ± 20.2
			47.3 ± 6.7	**88.6 ± 7.5**	**168.3 ± 2.9**			**296 ± 25**
PT			71.3 ± 4.1	96.3 ± 11.8	161 ± 9.5	221.1 ± 4.5	252.4 ± 7	337.4 ± 5.3
			86.4 ± 11.3	**118 ± 17.7**	**171 ± 30.7**			**296 ± 42**
Area 22			65.6 ± 9.4	110.8 ± 6.5	176.9 ± 7.9			227.9 ± 13.3
			64 ± 5.4	**110.1 ± 8.4**	**171 ± 16**			**286 ± 28.7**

*Indicates the supplementary negative component found for the voiced syllables in HG and PT (see Figs 4 and 6). Corresponding to earlier results (Liegeois-Chauvel *et al.*, 1991), the primary components were only recorded in the medial part of HG. Notice the difference in latency of the last component recorded in HG between voiced and voiceless syllables.

Table III

Mean latency (± SD) of the successive positive and negative main components evoked by voiced (normal type) and voiceless (bold type) syllables recorded in different anatomical structures in the right auditory cortex: contrary to left HG, only a very small difference in latency of the last component between voiced and voiceless syllables was found for right HG.

Right side	Primary components					Last component
HGm	23.3 ± 1.5	35 ± 7	55 ± 11.1	97 ± 12.8	163.7 ± 14.6	226.3 ± 55.4
	23.3 ± 1.5	**35 ± 7**	**47.3 ± 2.3**	**101.7 ± 2.9**	**199 ± 42**	**244.7 ± 39.**
HGl			48.3 ± 7.9	83.1 ± 8.7	165 ± 5	256 ± 46.4
			46.9 ± 5	**84.1 ± 7**	**173.2 ± 19.9**	**283 ± 42.3**
PT				105.8 ± 5.6	188.5 ± 21.2	340.4 ± 27.
				88 ± 7.36	**171.4 ± 9**	**328.8 ± 9.1**
Area 22			60 ± 3.6	94.7 ± 16	164.9 ± 6.7	223.8 ± 28.2
			55 ± 4.76	**90.8 ± 10.2**	**171.4 ± 12.6**	**300 ± 19.8**

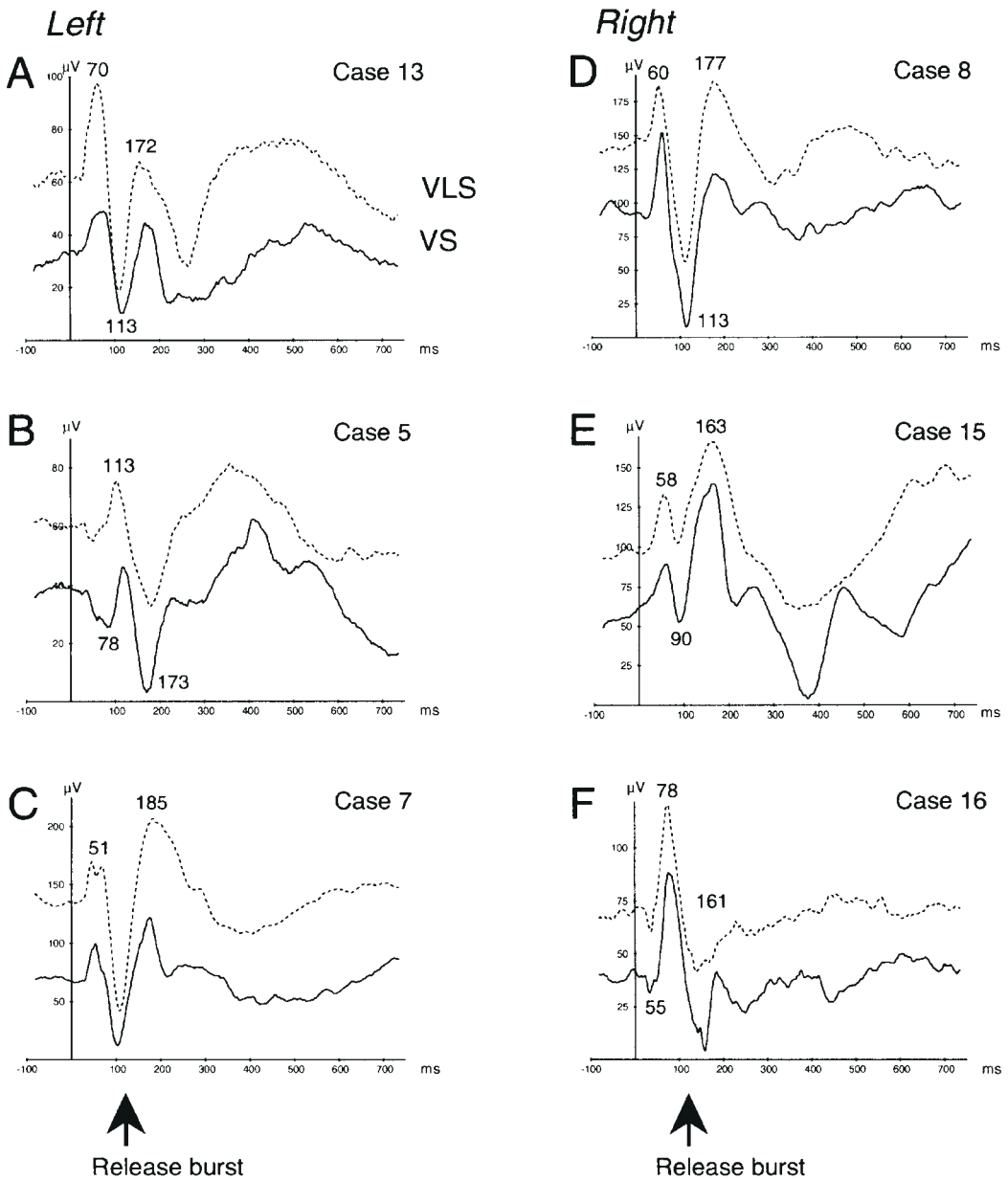

Figure 7. AEPs recorded in right and left area 22. See the legend of Figure 4 and comments in the text.

Figures 4 and 6, the processing of voiced and voiceless syllables is different in the left HG and PT. This is confirmed by the omnipresence of the negative component peaking at 252–262 ms preceded by positivity (204–221 ms) for VS. The last component latency is longer for VS than for VLS, suggesting that the syllable duration is involved in the temporal processing. This processing did not take place in the right auditory areas, so AEP components in response to VS or VLS were quite similar.

The present results demonstrate that the temporal coding of speech sound is performed in the left HG and PT and not in the right homologous cortical structures. This striking event highlights the functional difference between the area 22, on the one hand, and HG and PT, on the other, since this temporal coding is not observed in the left area 22.

Evoked Responses to Acoustic Speech Analogues

To investigate whether this temporal processing is related to the acoustic or to the linguistic features of the syllable, we analysed evoked responses to acoustic speech analogues of different durations and spectral composition (see Materials and Methods).

Figure 8 displays evoked responses to complex tones within the different auditory cortical areas. The first phenomenon is the waveform similarities of potentials from the right side whatever the duration and the spectral complexity of sound. In contrast, evoked responses from the left side were modulated by the stimulus duration. A striking exemple was seen in Figure 8*A*,*E*. An off-response occurred in all three recordings in the left HG (Fig. 8*A*) and in none of the recordings in the right HG (Fig. 8*E*). The off-response was correlated with the duration of the sound, beginning at 400, 270 and 180 ms for VLa-long (solid curve), VLa-short (dashed curve) and VOTa (dotted curve) respectively.

The solid curves in Figure 8*B–D* show that Va evoked a double response in the left HG (just as did the natural VS): an initial response to sound onset and a second to burst onset. The

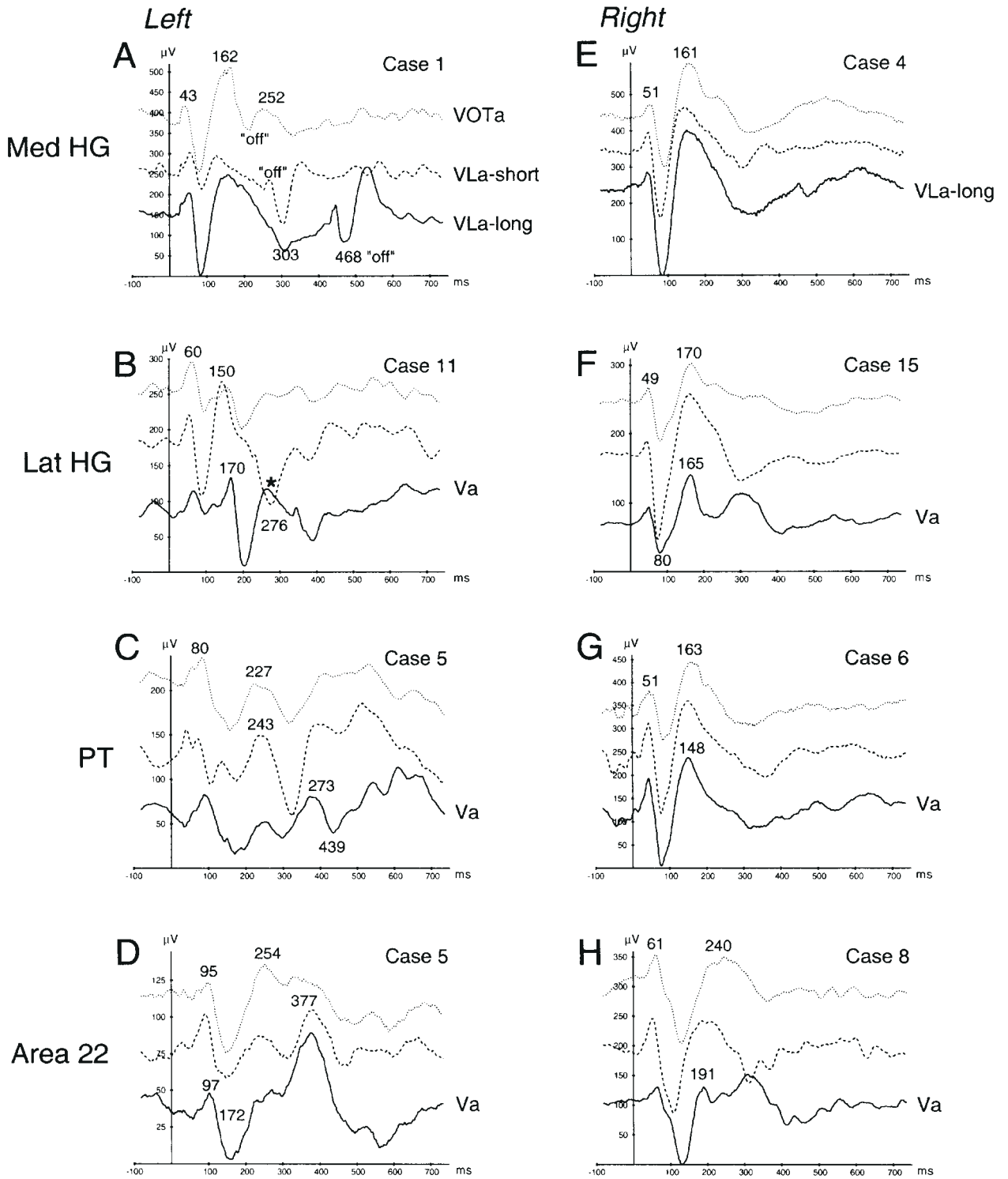

Figure 8. AEPs recorded in different auditory areas: left (*A*,*B*) and right (*E*,*F*) HG; left (*C*) and right (*G*) PT; and left (*D*) and right (*H*) area 22. The stimuli were the acoustic speech analogues VOTa (dotted curve), Vla-short (dashed curve), Va (full curves in *B*, *C*, *D*, *F*, *G*, H), and VLa-long (in *A*, *E*). For details of sounds, see Figure 3 and comments in the text.

response to the burst, however, was different for speech and non-speech sounds; the oscillatory responses superimposed on the potentials evoked by a syllable were never evoked by a acoustic speech analogues.

Sequential processing of the complex sound was predominantly observed in the left HG, to a lesser degree in the left PT, and not in area 22.

Discussion

The results of this study suggest that, in humans, evoked responses of left auditory cortex are related to the temporal structure of sounds. Indeed, we found that voiced and voiceless consonants were differently processed in left HG, suggesting a specific temporal processing of sounds which was not present in the right homotopic structures. It appears that in *left* HG,

neurons responsible for representing the different acoustic elements in syllables were activated in succession. In contrast, in the right cortical auditory areas, speech sounds evoked a similar pattern of response whatever the nature of the syllable, as if the syllable was processed as an entity.

A differential coding of voiced and voiceless syllables is preserved in the left PT. In contrast, this specificity has not been found for area 22, located anteriorly to HG, which does not show any asymmetry between both hemispheres in the processing of syllables. These results raise the question of the functional role of the different cortical auditory areas.

Time-locked responses are evoked to the consonant onset as well as to the release burst of the French VS in the left HG. Our data support a temporally based physiological mechanism for the differential perception of VS and VLS and agree with suggestions that this distinction is partially based upon detection of the non-simultaneous onset of the VOT and the release burst (Stevens, 1980). Furthermore, Dwyer *et al.* (Dwyer *et al.*, 1982) have shown that the presence of abrupt onset for stop consonants crucially determined lateralized processing. This mechanism is specifically involved in *the left auditory cortex*. Similar time-locked responses to speech sounds are also seen in scalp recordings (Kaukoranta *et al.*, 1987). But these AEPs were bilaterally distributed. This absence of asymetry could be explained by the fact that HG is deeply buried inside the sylvian fissure and that the specific activity of primary area, localized in the dorso-postero-medial part of HG (Liégeois-Chauvel *et al.*, 1991) is difficult to record from surface-recording techniques (EEG as well as MEG). At the level of the scalp surface, most of the recordable evoked responses presumably originate in cortical regions near the recording electrodes. However, relatively large signals originating from PT and area 22 might often make a significant contribution to the activity observed at a given scalp recording site.

This processing seems to be related to the acoustic rather than to the phonetic composition of the sound since identical time-locked responses were recorded to speech sound and non-speech sound with the same time course. Indeed, similar time-locked responses as those for speech are seen for the speech analogue sounds, implying that these responses are not specific detectors for acoustic speech components. This had already been observed in Kaukoranta's study (Kaukoranta, 1987). Right ear advantage (REA) has been found for auditory sequences suggesting left-hemisphere specialization for processing stimuli characterized by temporal acoustic features (Divenyi and Efron, 1979; Leek and Brandt, 1983). Schwartz and Tallal (Schwartz and Tallal, 1980) showed a significantly greater REA for discriminating stop consonants that contain transient formant information than for those in which they extended the duration of formant transition or for steady state vowels.

The basic functional organization of the auditory cortex in man (Celesia, 1976; Seldon, 1981; Liégeois-Chauvel *et al.*, 1991, 1994) is close to that of non-human primates. As a matter of fact, studies in monkeys have demonstrated the presence of neurons in the primary auditory cortex that are capable of representing the timing of phonetically important speech components. These cells show spike discharges phase-locked to stimulus periodicities. The (best) neurons could have a temporal resolution close to the millisecond (Brugge and Reale, 1985; Steinschneider *et al.*, 1992). In particular, they show responses time-locked to the onset and the release burst of syllables. However, contrary to the results of the present study, these neurons, expressing the *temporal* content of a sound, are equally present in *both* hemispheres in monkey (Steinschneider *et al.*, 1980, 1982, 1994, 1995). Nevertheless, Glass and Wollberg (Glass and Wollberg, 1983) have found a significantly higher percentage of responding units to natural vocalizations in the primary compared to the secondary left auditory cortex of squirrel monkeys, and this difference was not significant for the right hemisphere. Several behavioural studies with Japanese monkeys reported a significant REA for discriminating their own calls (Petersen *et al.*, 1978, 1984; May *et al.*, 1989). It is possible that the asymmetry found in monkey cortex is already indicative of some differentiation in the left auditory cortex, associated with the analysis of the meaning of the auditory input. However, similar results have been demonstrated in the rats which showed significantly better discrimination of tone sequences with the right ear than with the left ear (Fitch *et al.*, 1993). These data suggest that auditory temporal processing could not be related to linguistic processing.

A second interesting result of the present study is the existence of high-frequency, low-amplitude responses superimposed over the AEPs recorded in left HG and PT to verbal sounds. These oscillations, absent during the voicing, appear at the burst and last almost the duration of the vowel. The power spectrum is around 80 Hz. Different works have studied the auditory cortex with periodic amplitude modulations, and there is a widespread agreement that cortical neurons can fire phase- locked as long as the periodicities in the signal are below 100 Hz (Creutzfeldt *et al.*, 1980; Muller-Preuss, 1986; Schreiner and Urbas, 1988; Eggermont, 1991). The frequency of these responses might be in linear temporal relationship with the fundamental frequency (Fo) of the speaker's voice (near 180 Hz in our female speaker). But, although neural encoding of Fo is apparent at peripheral levels of the auditory pathway, including the VIIIth nerve and brainstem (Delgutte and Kiang, 1984; Blackburn and Sachs, 1990), it is not strongly represented at the level of auditory cortex (Steinschneider *et al.*, 1982; Phillips, 1993a,b). So, it seems more likely that our high-frequency superimposed responses may be one of the phenomena indicative of population coding with neurons whose activities are temporally correlated with each other.

The fact that we observed these oscillations for the speech sounds and not for the speech analogue sounds could be interpreted in two ways. First, it is possible that the formantic transitions within the speech stimuli, that were not incorporated into the non-speech analogue stimuli, might have driven the lateralized oscillatory responses. Eggermont (Eggermont, 1994) showed that the neural synchronization coefficient increased more during stimulus presentation as compared with spontaneous activity, especially when the stimulus was an amplitude- and/or frequency-modulated burst. Second, since the high-frequency superimposed responses are observed only in response to speech sounds and recorded in the left auditory cortex, this oscillatory activity could represent binding functions among the different acoustic features, each of which is detected by different individual neurons leading to a unified meaningful percept. Similar oscillations have been extensively studied since the work of Galambos *et al.* (Galambos *et al.*, 1981) as gamma-band activity supporting general perception mechanisms in the central auditory system (Mäkelä and Hari, 1987; Pantev, 1995). However, DeCharms and Merzenich (DeCharms and Merzenich, 1996) showed that neurons in the primary auditory cortex can coordinate the relative timing of their action potentials during the entire duration of continuous stimuli. They demonstrated that the relative timing of cortical action poten-

tials can signal stimulus acoustic features themselves, a function even more basic that perceptual feature grouping.

The present work has yielded direct evidence that high-frequency responses are generated in the human auditory cortex [confirming our previous data (Liégeois-Chauvel *et al.*, 1994)]. Future studies using speech analogues with other acoustic features may shed light on our understanding of the mechanisms underlying the high-frequency responses.

Relevance to Human Anatomy

This functional asymmetry between the left and right HG may be related to anatomical differences. It is well known that the morphological asymmetry observed in the posterior temporo-sylvian region in man [especially in the associative auditory areas, such as the PT (Braak, 1978; Galaburda and Sanides, 1980; Steinmetz *et al.*, 1989; Rademacher *et al.*, 1993; Kulynych *et al.*, 1994)] is related to the left hemisphere specialization for speech. Seldon (Seldon, 1981) found that, although the total area of primary cortex is the same on both sides, neurons in the left HG generally have a larger tangential dendritic arborization extent in the cell columnal organization than those in the right HG. Recently, morphological imagery studies showed an anatomical asymmetry that arises from a difference in the volume of white matter, which is larger in left than in right HG, which could be due to the presence of thicker or a larger number of myelinated fibers (Penhune *et al.*, 1996). The larger volume of cortical white matter of *left* HG, involving a greater number of connecting fibres and/or more myelinated fibres, could be responsible for the observed enhanced temporal processing capability in left HG.

Impairment in Temporal Processing Observed in Language Disorders

Left-hemisphere or bilateral lesions to auditory cortices lead to word deafness. Not all aspects of speech discrimination are disrupted to the same extent in these pathological situations. There is a general agreement that the discrimination of steady-state vowels is preserved, while discrimination of consonants, especially stops, is not (Saffran *et al.*, 1976; Auerbach *et al.*, 1982; Mendez and Geehan, 1988; Yaqub *et al.*, 1988). Recently, evidence has been provided suggesting that pure word deafness may be a manifestation of a basic temporal processing disorder. Two studies have questioned the ability of word-deaf patients to discriminate VOT. Auerbach *et al.* (Auerbach *et al.*, 1982) reported a patient with bilateral lesions who showed a normal labelling function but an impaired discrimination of consonant–vowel syllables when VOT differed by only 20 ms, even when the syllables had VOT values close to those defining the phonetic boundary between the relevant voiced and voiceless stop consonants. This suggests an underlying deficit in the temporal processing of successive acoustic events. Miceli (Miceli, 1982) presented comparable data for a patient with a more generalized cortical auditory disorder whose deficit in the discrimination of verbal and non-verbal sounds could be at least partly explained by confusions in the temporal processing of successive acoustic events [for review see Phillips and Farmer (Phillips and Farmer, 1990)]. These clinical data are in agreement with our results demonstrating that a non-verbal sound is also temporally coded in the left auditory cortex.

As already mentioned above, since speech signals have a complex time course, a strong performance in auditory temporal processing is necessary in order to process speech information. The level of temporal representation used by the nervous system in the processing of speech signals is an open question. Indeed, the discrimination of steady-state vowels is maintained after bilateral lesions of the primary auditory cortex (Auerbach *et al.*, 1982; Von Stockert, 1982; Mendez *et al.*, 1988). The waveforms of these phonemes are quite different. Vowels have no time structure above the level of the periodicities in their waveforms, and the fact that they are perceived after bilateral lesions presumably reflects the activation of other cortical auditory fields unaffected by the pathology. In contrast, stop consonants contain many transient acoustic events that are closely spaced in time (range around tens milliseconds). Perhaps, the currently most valid theory of speech recognition is the acoustic one: it is based on the fact that each phoneme has a unique acoustic signature (short-term spectrum and temporal variations) (Blumstein and Stevens, 1979, 1980) and that the perceptual mechanisms use a sliding temporal window to sample the stream of sound. The resulting samples then are matched against internal templates for phonetic tagging (Moore *et al.*, 1988; Viemeister and Wakefield, 1991; Phillips, 1993b).

In conclusion, this enhanced sensitivity to temporal acoustic characteristics of sounds of left human auditory cortex probably reflects information processing necessary for *further* speech processing. The task of the left primary and secondary auditory cortex would be to provide the cerebral language processor with a representation of the acoustic signal that has sufficient spectral and temporal pertinence to permit the phonetic tagging. That could explain why the perceptual elaboration of sounds with the relevant time frame of auditory content should be so dependent on the auditory cortex as is shown in language disorders.

A remaining question is why the auditory temporal processing in man should be more developed in the left auditory cortex whereas this mechanism is bilateral in monkey. Given the fact that the processing of syllables in the left hemisphere is already present in 3 month old infants (Dehaene-Lambertz and Dehaene, 1994), further research is needed to reveal to what extent the innate brain mechanisms of speech perception can be modulated by learning. This will give important knowledge about cortical plasticity.

Notes

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