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Auditory Evoked Potentials to Verbal Stimuli in Healthy, Aphasic, and Right Hemisphere Damaged Subjects

Pathway Effects and Parallels to Language Processing and Attention*

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Summary. Acoustic evoked potentials to meaningful words were recorded in healthy, aphasic, and right hemisphere-damaged subjects under four conditions: monaural left and right, binaural, and dichotic stimulation. Four major findings emerged. First, healthy and brain-damaged subjects differed in amplitude and latency values of the N1 and P2 components. In healthy subjects N1 was greater and P2 smaller than in aphasics. Both components peaked earlier in patients than in normals. Second, evoked potentials of healthy subjects showed a late sustained component which was decreased in aphasics. Third, the latencies of P1 and N1 as well as the amplitude of N1 showed a "pathway effect", i.e. shorter latency and greater amplitude to contralateral stimulation. Fourth, under the dichotic condition, P1 and N1 peaked earlier over the left hemisphere. The N1 amplitude behaved differently in the three groups depending upon stimulating conditions.

It is suggested that these differences reflect linguistic coding and related attentional processes in patients and normals.

Key words: Auditory evoked potentials – Lateralized brain damage – Language processing – Attention – Auditory pathways – Different stimulating conditions

Zusammenfassung. Es wurden *akustisch evozierte Potentiale* (AEP) *nach verbalen Reizen* bei Gesunden, Aphasikern und rechtshemisphärisch geschädigten Patienten abgeleitet. Sinnvolle Wörter wurden unter vier Bedingungen gegeben: monaural rechts und links, binaural und dichotisch. Es zeigten sich vier Haupteffekte. (1) Gesunde und hirngeschädigte Personen unterschieden sich in Amplituden- und Latenzwerten von N1 und P2. Bei Gesunden war N1

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größer und P2 kleiner als bei Aphasikern. Beide Komponenten gipfelten früher bei Patienten als bei Gesunden. (2) AEP's von gesunden Personen zeigten eine späte langsame Komponente, die bei den Aphasikern erniedrigt war. (3) Die Latenzen von P1 und N1 zeigten ebenso wie die Amplitude von N1 einen „Gehörbahneffekt", d. h. kürzere Latenz und größere Amplitude nach kontralateraler Stimulation. (4) Unter der dichotischen Bedingung gipfelten P1 und N1 früher über der linken Hemisphäre. Die N1-Amplituden der drei Personengruppen verhielten sich unter den verschiedenen Stimulationsbedingungen unterschiedlich. Die Befunde dürften linguistische Verarbeitung und damit verbundene Aufmerksamkeitsprozesse bei Patienten und Gesunden reflektieren.

Introduction

Clinical and behavioral analyses suggested that the left hemisphere in right-handers is primarily responsible for language functions, while the right hemisphere is prefered in the perception of certain nonverbal (musical) stimuli. These views were based on observations including dichotic listening in healthy subjects and studies with split-brain patients (Kimura 1964; Sperry 1973). These data have also been interpreted as reflecting differences in processing strategies between the two hemispheres, the left side being analytical, the right side holistic in nature (Zaidel 1978; Witelson 1977).

Numerous studies with normals have attempted to find electrocortical indices of lateralized processing of speech/nonspeech stimuli, and to correlate some components in evoked cortical potentials (EP). These studies have generally found small EP differences in amplitude or latency favouring the left hemisphere's leading role in speech perception (Wood et al. 1971; Friedman et al. 1975; Galambos et al. 1975).

In electrophysiological studies with brain-damaged patients (aphasics) some authors, using meaningless auditory stimuli (click), reported that the response waveform as a whole changed in appearance due to cortical damage (Liberson 1966; Rappaport et al. 1977) but only few attempts have been made to relate disabilities to specific component changes. Greenberg and Metting (1974) employed verbal stimuli monaurally, but the authors found no amplitude differences between the responses of the two hemispheres, only shorter latencies of certain components were seen over the damaged left side.

Evoked potentials indicated not only functional stages in processing, but the operation of attentional processes as well. Earlier components (particularly N100) reflected the ability to selectively attend to a particular input channel while later components (e.g. P300) appeared to indicate evaluation and related decisions of stimuli from the attended channel (Hillyard and Picton 1979). Therefore EP changes in aphasics should also be evaluated in the light of possible modifications in attentional capabilities of the patients.

Furthermore behavioral investigations with verbal monaural stimulation (Bakker 1970; Frankfurter and Honeck 1973) and simultaneous presentation of different verbal stimuli (dichotic listening paradigm) in normals (Kimura 1967),

split-brain (Milner et al. 1968; Sparks and Geschwind 1968) and other brain-damaged patients (e.g. Schulhoff and Goodglass 1969; Kimura 1961) have suggested (1) a greater efficiency of contralateral auditory pathways, (2) the lateralization of linguistic decoding processes mainly in the left hemisphere, and (3) inhibition of ipsilateral messages during dichotic stimulation.

Numerous attempts have been made to obtain evoked potential (EP) correlates of these differences. In animals, greater EPs to clicks (Rosenzweig 1951) and multiple unit activity to speech sounds (Walker and Halas 1972) were recorded on monaural contralateral stimulation. In humans, EPs to monaural stimuli were generally found to be larger or to have shorter latency after contralateral stimulation (Majkowski et al. 1971; Price et al. 1966; Butler et al. 1969; Peronnet et al. 1974; Szirtes 1977).

Controversial results have been reported by investigators using dichotic stimulation in normals. Haaland (1974) found no interhemispheric amplitude differences in the N1 component of EPs to speech stimuli, other studies (Neville 1974, 1980; Anderson 1977) have reported a greater N1 component on the left than on the right.

The main objective of the present paper was to describe EP waveforms to verbal stimuli in healthy and brain-damaged subjects and to correlate the observed differences with impairments in coding and attentional processes due to the pathological brain function. A further aspect of this study was, to describe the effect of different presentation forms of speech stimuli on EPs.

Material and Methods

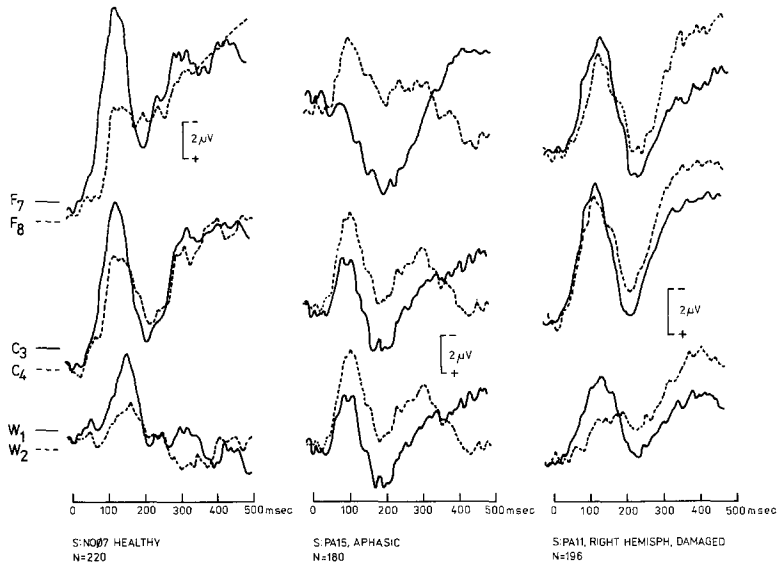
Subjects. We investigated 8 normal adults between 18 and 34 years old ($\bar{x} = 25$ years), 12 aphasic patients (4 Broca and 8 Global, as tested with a German version of the Boston Diagnostic Aphasia Examination) with left hemisphere damage due to vascular accidents (36–77 years old, $\bar{x} = 58$ years), and 6 patients (21–75 years old, $\bar{x} = 55$ years) with right hemisphere damage (RHD) without language disturbances. Their damage was also due to vascular accident. All subjects were right-handed by the usual questionnaires, had no hearing loss greater than 30 dB or asymmetrical thresholds between the ears as established by a pure-tone audiogram in the language-relevant frequencies (250–4000 Hz).

Procedure. The EEG was recorded from electrode positions F7, F8, C3, C4, as well as from points midway between T3 and T5 (W1) and T4 and T6 (W2) of the international 10–20 system. Beckman electrodes were used and fixed with collodion, the electrode impedances were below 4 kΩ. Linked ears served as reference. Eye movements were controlled by electrodes from above and lateral to the eye. Brain activity was recorded monopolarly, amplification was done by an Elema-Mingograph (time constant 1.2 s, upper cut-off 30 Hz). Storage was made on a Sangamo FM tape recorder, and data analysis off-line with a PDP 12 computer.

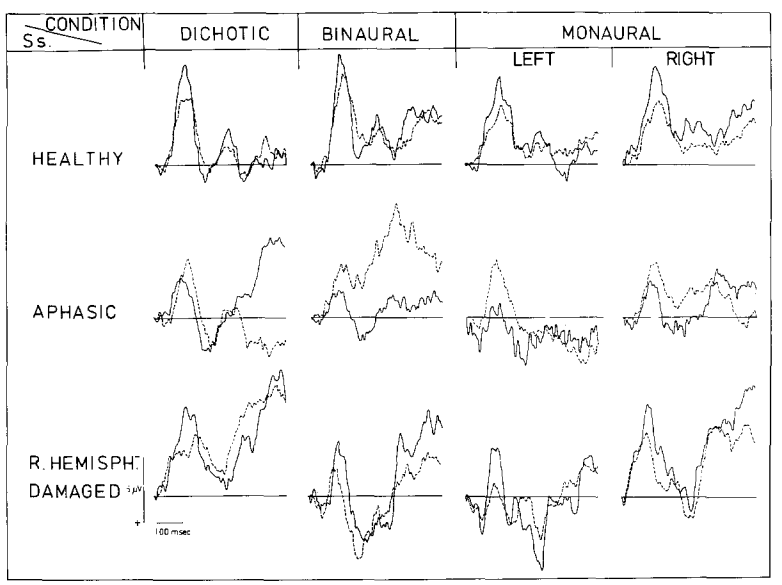
The averaging program related all measurements to a normalization level computed as the mean voltage of EEG activity just preceding the appearance of the stimuli. Averaged evoked potentials, derived from between 30 and 80 artifact free trials, were displayed on an X-Y plotter.

The peaks of the EPs were identified by visual inspection of the different waveforms of each subject. Within a range where usually the different peaks are expected (P1 30–50 (60) ms, N1 100–120 (130) ms, P2 150–200 (230) ms, N2 or sustained negativity 300–550 ms) we selected the maximum of the amplitude.

Stimuli were one- and two-syllabic words with durations between 300–700 ms. All words began with consonants (e.g. Feld/Geld—field/money) to insure that the initial phonetic coding



a



b

Fig. 1. **a** Grand average waveforms across conditions in a healthy, aphasic and RHD subject. Number indicates that of individual sweeps in the average. Note the negative displacement of the whole waveform in frontal and central leads of healthy and RHD subjects. **b** Auditory evoked potentials (AEP) over the central region in a healthy, Broca-aphasic and a RHD subject under different stimulating conditions. Solid line left (C3), dotted line right (C4) hemisphere

processes (CV transitions) would be time-locked across stimuli. They were recorded on an Uher stereo tape recorder as described by Rothenberger and Jürgens (1978). Presentation was made at 60 dB above threshold through Koss PRO/5 LC headphones.

Four stimulating conditions, randomized over subjects, were used: left and right monaural, dichotic, and binaural presentation. For the monaural and binaural condition we used a list of 60

Table 1. Summary of analysis of variance. **1.** Amplitude data

Source	Lead	Mean square	Mean square error	*df*	*F*	*P*<
Component: P1						
A (group)	F7-8	10.32	2.50	2.23	4.12	0.05
	C3-4	12.50	2.99	2.23	4.18	0.05
	W1-2	11.59	2.41	2.23	4.80	0.025
C (condition)	W1-2	3.74	0.73	3.69	5.13	0.005
Component: N1						
A (group)	F7-8	138.35	32.41	2.23	4.27	0.05
	C3-4	207.66	52.05	2.23	3.99	0.05
A × B	F7-8	27.35	5.41	2.23	5.06	0.025
(group × hemisphere)	C3-4	36.58	3.97	2.23	9.21	0.005
	W1-2	23.26	2.53	2.23	9.20	0.005
A × C	F7-8	12.53	4.10	6.69	3.07	0.025
(group × condition)	C3-4	26.02	7.24	6.69	3.60	0.005
	W1-2	7.07	2.84	6.69	2.49	0.05
Component: P2						
A (group)	F7-8	134.44	22.85	2.23	5.89	0.01
	C3-4	141.18	37.23	2.23	3.79	0.05
	W1-2	41.85	12.33	2.23	3.39	0.10

words, for the dichotic condition a list of 30 pairs was available. The lists were given twice for each condition. The interval between words and conditions was variable; free recall was required under the dichotic condition (the subjects could reproduce the two words in any order they liked).

To enhance the subjects' alertness and cooperation, especially in the case of patients with neuropathology, the experimenter periodically interrupted stimulus presentation and encouraged the subject to describe what he or she had just heard particularly when there raised difficulties finding and reproducing the word.

During the experiment, subjects remained supine in a dimly illuminated, sound-attenuated chamber and were instructed to focus on a fixation point provided by a small lamp. They were instructed to repeat the verbal stimuli after sudden brightening of the fixation lamp which occurred 3–4 s later to avoid artifacts of the AEP.

Results

EPs were generally composed of four peaks (Fig. 1 a and b): an early small *positive deflection (P1),* the first *negative peak (N1),* a second *positive deflection (P2)* and a later *negative wave (N2).*

I. Analyses of Variance

Separate three-way analyses of variance (Table 1/1 and 1/2) were performed for latency and amplitude values of each EP-component, and also for the corresponding leads in frontal, central and temporal position (subject groups × hemispheres ×

Table 1. Summary of analysis of variance. **2.** Latency data

Source	Lead	Mean square	Mean square error	*df*	*F*	*P*<
Component: P1						
A (group)	C3-4	7,027.81	947.68	2.23	7.42	0.005
	W1-2	3,454.31	968.75	2.23	3.57	0.05
B × C	F7-8	395.31	95.69	3.69	4.13	0.01
(hemisphere × condition)	C3-4	325.75	127.31	3.69	2.48	0.10
	W1-2	452.62	173.30	3.69	2.61	0.10
Component: N1						
A (group)	C3-4	2,850.50	852.15	2.23	3.35	0.10
B × C	F7-8	343.00	123.52	3.69	2.78	0.05
(hemisphere × condition)	C3-4	330.67	132.26	3.69	2.50	0.10

Table 1. 3. Amplitude of N1 component according to group and hemisphere (values in μV). A comparison within group but between hemispheres

Lead	Frontal		Central		Temporal	
Hemisphere	Left	Right	Left	Right	Left	Right
Group						
Normals	−5.63	−5.03	−7.33	−6.64	−3.71	−3.41
Aphasics	−2.28	−3.26	−3.22	−4.62	−1.96	−2.90
RHD patients	−5.34	−3.86	−6.99	−5.73	−4.13	−2.72

stimulating conditions). The program, written by F. Bartlett, also performed a posterior *t*-test between means.

Latency of P1

a) Groups. There was a significant main effect of groups in the central and temporal leads ($F = 7.42$, *df* 2.23; $P < 0.005$ in central, $F = 3.57$, *df* 2.23; $P < 0.05$ in temporal leads). *T*-tests revealed a significant latency difference ($P < 0.01$) between healthy (31.2 ms in central and 48.4 ms in temporal leads) and aphasic subjects (50.3 and 61.6 ms respectively). RHD patients also showed longer P1 latency (in central leads 43.1 ms, in temporal leads 54.1 ms) than healthy subjects but the difference was not significant.

b) Stimulating Conditions. A significant interaction appeared between hemisphere × condition in frontal responses ($F = 4.13$, *df* 3.69, $P < 0.01$) and approached significance in the two other leads as well. This interaction resulted from the very short latency of P1 (37.6 ms) over the left frontal region under the dichotic condition. This value differed from monaural left (48.5 ms) and right (48.1 ms) stimulation at the 0.01 level and from the binaural condition (45.7 ms) at the 0.05 level over the left hemisphere.

Under the dichotic condition, P1 latency was shorter ($P < 0.05$) over left than over right frontal region (37.6 vs 43.7 ms). With respect to pathway efficiency, it was noted that during right ear stimulation the left side's P1 component preceded the right by 7.6 ms ($P < 0.05$). During left ear stimulation this component peaked 6.4 ms earlier over the contralateral right side than over the left ($P < 0.10$). These pathway effects were primarily seen in the aphasics.

Amplitude of P1

a) Groups. There was a significant group effect in all three leads ($F = 4.12$ in frontal, 4.18 in central, and 4.80 in temporal leads, *df* 2.23, $P < 0.05$). *T*-tests revealed a difference at the 0.005 level between healthy and aphasic subjects, P1 being always greater in aphasics. The mean values were 0.54, 0.43, and 0.52 μV in frontal, central and temporal responses of healthy subjects, and 1.26, 1.23, and 1.28 μV in aphasics, respectively. Similar differences were observed in the comparison of healthy vs RHD subjects (1.08, 1.04, and 1.15 μV) but this reached the 0.05 significance level only in the central and temporal responses.

b) Stimulating Conditions. A significant stimulating condition effect appeared in the temporal responses ($F = 5.13$, *df* 3.69, $P < 0.005$). The P1 component showed smaller amplitude in the dichotic (0.66 μV) than in the other three conditions. It differed from P1 in monaural left (1.10 μV) and right responses (1.28 μV) at the 0.10 and 0.005 level respectively.

Latency of N1

a) Groups. The first negative component (N1) showed a general tendency toward shorter latency in both patient groups in each recording, but this approached significance only in the central leads. In this case the average latencies were 116.2 ms in aphasics, 118.7 ms in RHD patients, and 128.3 ms in normals.

b) Stimulating Conditions. A weak hemisphere × condition interaction appeared in frontal ($F = 2.78$, *df* 3.69, $P < 0.05$) and central EPs ($F = 2.50$, *df* 3.69, $P < 0.10$). In the frontal responses it resulted from the shortest latency of N1 over the left side (109.2 ms) in the dichotic condition. It differed from the right side response (117.7 ms) in the same condition ($P < 0.025$). Over the left hemisphere the N1 peak under the dichotic condition was earlier (109.2 ms) than under the monaural left condition (121.7 ms, $P < 0.025$) and just missed significance when compared to N1 under the binaural condition (117.6 ms).

With respect to pathway efficiency, one comparison achieved significance in the hemisphere × condition interaction of central responses. The left N1 peak (116.0 ms) preceded the right (125.7 ms) during monaural right stimulation ($P < 0.01$).

Amplitude of N1

a) Groups. The amplitude of the N1 component showed significant group effects in frontal ($F = 4.27$, *df* 2.23, $P < 0.05$) and central leads ($F = 3.99$, *df* 2.23, $P < 0.05$). In healthy subjects the N1 peak in frontal (5.3 μV) and central (7.0 μV) recordings was

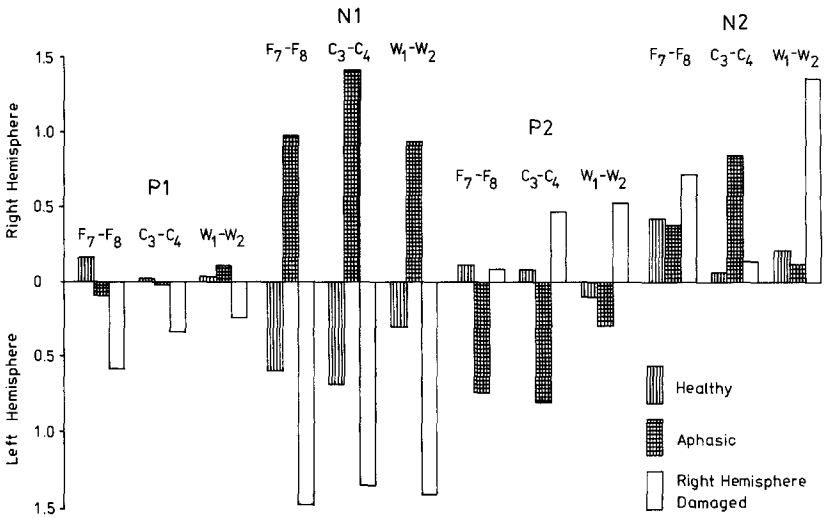


Fig. 2. Interhemispheric amplitude differences (μV) for each component, averaged across conditions

greater than in aphasics (2.8 and 3.9 μV, respectively, $P < 0.01$). While the overall magnitude of N1 in healthy and RHD subjects (4.6 and 6.4 μV, respectively) was similar, the difference between aphasics and RHD patients just missed the 0.05 significance level.

Significant group × hemisphere interactions were found for N1 amplitude ($F = 5.06$, *df* 2.23, $P < 0.025$ in frontal; $F = 9.21$, *df* 2.23 in central and 9.20 in temporal leads respectively, $P < 0.005$). Comparison of N1 amplitude between groups over the *same hemisphere* revealed further sources of the group × hemisphere interaction. Over the *left* side in frontal and central responses, healthy subjects differed strikingly ($P < 0.005$) from aphasics; there was no difference between healthy and RHD subjects while the latter group showed again significantly ($P < 0.01$) greater N1 amplitude than did the aphasics. Over the *right* hemisphere the difference in N1 amplitude in frontal and central responses between healthy and aphasic subjects fell just short of significance.

A comparison within groups but *between hemispheres* revealed the following patterns (Fig. 2, Table 1/3). There was no significant asymmetry in healthy subjects with only a slight tendency for a greater left amplitude of N1. In aphasics the N1 peak tended to be greater over the non-damaged right hemisphere ($P < 0.05$ in frontal and central leads, $P < 0.10$ in temporal leads). In RHD patients the N1 component was greater over the left side ($P < 0.05$ in all three comparisons).

b) Stimulating Conditions. A significant group × condition interaction appeared for the N1 amplitude ($F = 3.07$, *df* 6.69, $P < 0.025$ in frontal; $F = 3.60$, *df* 6.69, $P < 0.005$ in central; and $F = 2.49$, *df* 6.69, $P < 0.05$ in temporal leads, respectively). This was due to the fact that N1 amplitudes were greatest under the dichotic condition in healthy subjects, during monaural right ear stimulation in RHD patients (smallest under monaural left), and there was no significant difference between conditions in any leads of the aphasics.

Latency of P2

a) Groups. The latency of the second positive component (P2) showed an overall tendency to peak earlier in patients. Only in temporal responses did the difference approach the significance level. Average values were 229.3 ms in normals, 210.4 ms in aphasics, and 212.6 ms in RHD patients. A weak group × hemisphere interaction could be observed in central EPs which reflected shorter latencies of P2 over the respectively damaged hemisphere in patients.

b) Stimulating Conditions. There was no hemisphere × condition or group × condition interaction.

Amplitude of P2

a) Groups. The amplitude of the P2 component showed only a main effect of groups in frontal ($F = 5.89$, df 2.23, $P < 0.01$) and central leads ($F = 3.79$, df 2.23, $P < 0.05$). In healthy subjects the P2 component of frontal (0.06 μV) and central (0.2 μV) responses differed significantly ($P < 0.01$) from the P2 of aphasics (3.3 and 2.9 μV respectively). The magnitude of this component in RHD patients ranged between 2.4 and 1.6 μV respectively but the differences fell short of significance. It should be noted that similar tendencies were observed in temporal leads as well.

b) Stimulating Conditions. There was no hemisphere × condition or group × condition interaction.

Latency and Amplitude of N2

a) Groups. In the late component (N2 or sustained negativity) no significant differences were observed in either the latency or amplitude values. There was, however, a relatively consistent tendency in each group; the right hemisphere showed a greater late negative component (see Fig. 2). This asymmetry was especially clear in aphasic's central (C3 2.8 vs C4 3.6 μV) and RHD patients' temporal responses (W1 2.1 vs W2 3.5 μV).

b) Stimulating Conditions. There was no hemisphere × condition or group × condition interaction.

Table 2. Latency (ms) and amplitude (μV) values of P1 and N1 of the frontal responses over the two hemispheres (L = left, R = right) under different stimulating conditions

Condition	Component							
	P1				N1			
	Latency		Amplitude		Latency		Amplitude	
	L	R	L	R	L	R	L	R
Dichotic	37.6	43.7	0.9	0.8	109.2	117.7	4.8	3.9
Binaural	45.7	41.7	1.3	1.0	117.6	118.4	3.8	3.8
Monaural R	48.1	55.7	0.9	1.0	116.0	125.7	4.7	3.7
Monaural L	48.5	42.1	1.0	0.9	121.7	118.6	4.4	4.7

Table 3.1. Latency (ms) and amplitude (μV) values of the contralateral and ipsilateral auditory pathways under the monaural condition (after Tanguay et al. 1977)

Group	Lead	Component	Latency			Amplitude		
			Contra	Ipsi	*P*	Contra	Ipsi	*P*
Aphasic	Central	P1	50.6	61.1	0.05	1.34	1.31	n.s.
Aphasic	Temporal	P1	59.6	73.1	0.01	1.70	1.36	n.s.
Normal	Central	N1	127.0	135.4	0.10	6.70	6.20	0.01
RHD	Temporal	N1	133.5	126.5	n.s.	3.60	3.30	n.s.

Table 3.2. Latency (ms) and amplitude (μV) values of the two hemispheres (L = left, R = right) under the monaural condition (after Tanguay et al. 1977)

Group	Lead	Component	Latency			Amplitude		
			L	R	*P*	L	R	*P*
Aphasic	C3 vs C4	N1	117.9	124.8	n.s.	3.1	4.5	0.05
Aphasic	W1 vs W2	N1	131.5	125.6	n.s.	1.9	2.9	n.s.
Normal	C3 vs C4	N1	130.5	131.9	n.s.	6.9	6.0	0.05
RHD	W1 vs W2	N1	125.6	134.3	n.s.	4.2	2.6	0.05

II. Monaural Stimuli

The efficiency of ipsilateral and contralateral pathways was also assessed by separate analysis of *monaural responses* using the same data. For this purpose we followed the procedure of Tanguay et al. (1977) and subtracted the sum of the two hemispheres ipsilateral responses (left hemisphere/left ear + right hemisphere/right ear) from the sum of values representing contralateral stimulation (left hemisphere/right ear + right hemisphere/left ear). Hemisphere effects were studied by subtracting EP values representing the right side of the scalp (right hemisphere/right ear + right hemisphere/left ear) from those representing the left side of the scalp (left hemisphere/left ear + left hemisphere/right ear). This type of analysis revealed (Table 3) that the earlier appearence of the *P1 peak* in contralateral responses was due primarily to the responses in the aphasic group. Thus, in central and temporal responses, this component was leading the ipsilateral peak by about 12 ms (50.6 vs 61.1 ms, and 59.6 vs 73.1 ms respectively). The *N1 component* in the healthy group also demonstrated a shorter latency from contralateral stimulation in central leads (127.0 vs 135.4 ms). A similar analysis of amplitude also yielded a significant contralateral advantage in the central N1 peak of healthy subjects (6.7 vs 6.2 μV), and the two patient groups revealed the same tendency as well.

III. Dichotic and Binaural Conditions

An additional analysis was performed on *N1 amplitude* values under *dichotic and binaural* conditions. Binaural values were subtracted from dichotic ones and *t*-tests

for paired values were again calculated for each lead and group. In healthy subjects, left hemisphere values increased significantly in frontal ($\bar{x} = 1.7\,\mu V$, $P < 0.05$) and in temporal ($\bar{x} = 1.3\,\mu V$, $P < 0.02$) leads, but the same tendency, though insignificant, was apparent over the right hemisphere. In aphasics there was no significant difference between the two conditions, the trend being rather a decreased amplitude under the dichotic condition. In RHD patients N1 increased significantly in the left frontal ($\bar{x} = 1.8\,\mu V$, $P < 0.05$) and central ($\bar{x} = 1.3\,\mu V$, $P < 0.05$) responses.

Discussion

All three components within the first 250 ms differed significantly between healthy and aphasic subjects. The early cortical component (P1) showed longer latency and greater amplitude in both patient groups. The longer latency of P1 in aphasics as opposed to healthy subjects might simply be due to the fact that the first negative component (N1) had a steeper rise in healthy subjects and therefore masked the initial positivity in their response. The difference in P1 component among groups, therefore does not seem to indicate a pathological event of primary importance in aphasia. This suggestion was supported by our results of monaural responses in these aphasics which had revealed similar "pathway effects" (i.e. shorter and/or greater responses to contralateral stimulation) as generally reported in healthy subjects (e.g. Tanguay et al. 1977; Seitz et al. 1980). Whether the shorter latencies of P1 (pathway data) as well as N1 and P2 peaks in aphasics indicated an increased excitability (disinhibition?) resulting from the damage to cortical and subcortical tissue remains a matter of debate (Spink et al. 1979).

In general, according to the review of Klorman et al. (1978) the age difference between normals and patients in our study ($\bar{x} = 25$ years vs $\bar{x} = 57$ years) is unlikely to account for the differences in AEPs reported in our data.

The N1 peak had a markedly greater amplitude in healthy subjects than in aphasics. We did not find significant asymmetry in N1 amplitude in healthy subjects, although the mean left responses were slightly greater for every lead. This is in accord with other reports of amplitude differences between hemispheres in normal subjects (for an overview see the special issue of Brain and Language, edited by D. L. Molfese 1980).

Studies dealing with the effects of aphasia on EPs have reported unequivocal changes in EPs to pure tones or clicks (Liberson 1966; Morley and Liedtke 1976; Kolman and Shimizu 1972). Greenberg and Metting (1974) observed no amplitude asymmetry in EPs to words and matched noise stimuli. In the present study with verbal material the N1 amplitude was reduced over both hemispheres, but there was a significantly greater N1 peak over the nondamaged right side. This lack of agreement may be attributed to their use of bipolar recording which tends to obscure amplitude asymmetries.

Neville (1980) also reported a decrease of the N1 amplitude to words over the damaged as well as over the undamaged hemisphere. The three patients with alexia without agraphia had a lesion of the left occipital lobe and presumably the splenium of the corpus callosum. On the other hand, one patient with a left

occipital lesion without alexia had normal distribution (a decreased or missing N 1 only over the damaged side), suggesting that in the three patients with alexia as well as in our aphasics, the corpus callosum might have been involved in the damage.

Our findings parallel the observation of both a general (symmetrical) and an asymmetrical disturbance in behavioral performance of aphasic subjects during dichotic perception (Schulhoff and Goodglass 1969) and point to an impairment of both specific (coding) and nonspecific (attentional) processes. Greenberg and Metting also reported shorter latency components over the damaged hemisphere. In our study no clear interhemispheric differences in latency have been observed in aphasics.

There were slight latency differences, however, among subject groups: P1 peaked later, while N1 and P2 tended to peak earlier in patients than in normals.

The N1 component (and in part the P2 also) were demonstrated to be responsive to acoustic properties of stimuli (Butler 1972; Lindsey 1971), and to reflect also the operations of phonetic feature extraction in healthy subjects (Wood et al. 1971; Dorman 1974). Furthermore, Molfese (1979, 1980) has demonstrated several factors in EPs to language stimuli which reflect processing of semantic aspects of the stimuli.

While our results do not establish specific relations between EP components and coding operations, our observations point to the impairment of both acoustic and phonetic/phonematic coding in aphasic patients, a well known fact in behavioral data (Efron 1962; Swisher 1967; Carpenter and Rutherford 1973; Blumstein et al. 1977; Keller et al. 1980). In particular, the smaller N 1 peak and the increase in P2, may indicate a reduction of processing capacity in aphasics, which would also explain why their responses did not differ between dichotic and binaural conditions.

Differences in *attentional processes* might also contribute to the observed results. By clinical impression the aphasic patients had the strongest problems with attention. Also our design (first a verbal stimulus, then after 3–4 s a light after which verbal reproduction was required) suggests a CNV-like condition. We mentioned that a further difference between healthy and aphasic subjects was a more pronounced negative displacement of the EP in healthy subjects. This displacement may account in part for the increased N1 and the decreased P2 amplitude values in healthy subjects. Hillyard et al. (1973) found such a negative-going modulation of EP correlated with selective attention and Näätänen and Mitchie (1979) reported this displacement beginning as early as 50 ms. They called this shift "processing negativity" and suggested it could reflect a detection of and orienting to the stimulus and/or subsequent processing of the stimulus features. Whether this early negative displacement is a different process from the sustained potential which appears as a slow negative component (N2) around 400–500 ms in our records, remains an open question. Auditory sustained potentials were reported as having a frontocentral maxium (Picton et al. 1978) and were found to be symmetrically distributed during both linguistic and tone stimulation (Hillyard and Woods 1978). In our study we found that the late sustained component showed somewhat smaller magnitude in aphasics than in other subjects and its amplitude was greater over the right than over the left hemisphere in all groups in all leads. Dimond (1979) has demonstrated that split-brain subjects on vigilance tasks

performed better with stimuli projected to the right hemisphere and arrived to the conclusion that sustained attention seems to be a function of the right hemisphere. It is tempting to suppose that in aphasics the reduction of N1 amplitude over both sides reflects impairment of task-related selective attention while the right hemisphere plays a significant role in maintaining sustained attention during the task. Our suggestion is in accord with observations that sustained potentials indicate not only the acoustic attributes of the stimulus (Picton et al. 1978; Keidel 1976) but they can be observed after cessation of an acoustic stimulus as well (Jarvilehto and Fruhstorfer 1973).

In a study by Kutas and Hillyard (1980) with normals the late EP components elicited by the words during reading a sentence were highly variable across subjects, positive and negative components were seen around 300 ms. They suggest that "this variation may be related to different processing strategies during reading". Their N309 by their warning stimulus (a slide containing /xxxxx/ as a word-like pattern) "may be associated only with the complex or linguistic variety of the task used here".

If our results would reflect primarily attentional deficits, further experiments with statistical use of a principal components analysis (PCA) should show the above described effects as a single factor. We suggest that impairments in psycholinguistic and attentional processing capacities are responsible for the observed EP changes in aphasics.

Pathway effects (Table 2) have been generally reported for the N1 component (Majkowski et al. 1971; Price et al. 1966), and we also found that the N1 peak appears earlier in the left than in the right central responses during right ear stimulation in healthy subjects. Furthermore, amplitude values showed a similar tendency, i.e. larger contralateral N1 peaks in all groups (normals and patients). In addition, we observed that the N1 component in healthy subjects showed an increase over *both* hemispheres as the stimulation became linguistically more complex (from binaural to dichotic). This observation may suggest that the increased coding requirements of the left hemisphere lead to an increased activity of the right hemisphere as well. The complex dichotic stimulus mode may provide a more difficult processing task for the left hemisphere, thus lessening its linguistic dominance of the right. As a result, the right hemisphere is relatively free to develop processing within its own linguistic capabilities (Kelly and Orton 1979). This may also explain why we found less interhemispheric asymmetry in N1 during dichotic than monaural right stimulation in healthy subjects, and the opposite relations in RHD patients. When the right hemisphere is disturbed it is handicapped in the development of its own capabilities and may be easily dominated by the left hemisphere. At the same time, some of our results point to the leading role of the left hemisphere in language processing: e.g. in the frontal responses during dichotic stimulation both the P1 and N1 components appeared earlier over the left hemisphere than over the right. This may show that complex linguistic tasks "facilitate" the pathways to the left hemisphere.

The attention-model of Kinsbourne (1970, 1973) could explain this facilitation. A hemisphere specialized for the processing of verbal material would become activated by foreknowledge of having to evaluate verbal material. Our subjects knew exactly the type of task they had to perform. So they had high attention to verbal stimuli and the corresponding left hemisphere. In addition, the dichotic

word-pair seems to require more analytical processing than the other conditions while the left hemisphere is working in a logical-analytical mode (Witelson 1977).

The N1 amplitude seems to be a good indicator for the linguistic capabilities of the left hemisphere. In normals and RHD patients the N1 amplitude increased with the processing of complex linguistic material. In the aphasic group there was no such effect, with the trend being a rather decreased amplitude. The insufficiency of the damaged left hemisphere seems to be evident when it has to process complex verbal material, although easier tasks may be solved. This supports the position of Keller et al. (1980) who suggested that the effect of a brain lesion on the linguistic receptive capacity was comparable to some form of interference with processing in normal subjects, be it the addition of background noise or increased memory requirements. Under such circumstances, easy tasks continued to pose no problem while complex materials did.

As we have shown hemispheric asymmetries of auditory EPs are not only determined by the task (e.g. priming) or the stimuli (verbal/nonverbal), they may also be influenced by the different stimulating conditions (dichotic, binaural, monaural). This is in accordance with behavioral data (Pohl 1979; Sparks and Geschwind 1968; Sparks et al. 1970).

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