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Binaural interaction in auditory evoked potentials: Brainstem, middle- and long-latency components

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Binaural interaction occurs in the auditory evoked potentials when the sum of the monaural auditory evoked potentials are not equivalent to the binaural evoked auditory potentials. Binaural interaction of the early- (0–10 ms), middle- (10–50 ms) and long-latency (50–200 ms) auditory evoked potentials was studied in 17 normal young adults. For the early components, binaural interaction was maximal at 7.35 ms accounting for a reduction of 21% of the amplitude of the binaural evoked potentials. For the middle latency auditory evoked potentials, binaural interaction was maximal at 39.6 ms accounting for a reduction of 48% of the binaural evoked potential. For the long-latency auditory evoked potentials, binaural interaction was maximal at 145 ms accounting for a reduction of 38% of the binaural evoked potential. In all of the auditory evoked potentials binaural interaction was long lasting around the maxima. The binaural interaction component extends for several milliseconds in the brainstem to tens of milliseconds in the middle- and long-latency components. Binaural interaction takes the form of a reduction of amplitude of the binaural evoked potential relative to the sum of the monaural responses, suggests that inhibitory processes are represented in binaural interaction using evoked potentials. Binaural processing in the auditory pathway is maximal in the time domain of the middle-latency components reflecting activity in the thalamo-cortical portions of the auditory pathways.

Auditory evoked potential; Brainstem; Middle-latency; Long-latency; Binaural interaction; Evoked potential; Adults

Introduction

Binaural interaction in auditory evoked potentials is observed when the sum of the monaural responses is not equal to the binaural response (Kemp and Robinson, 1937; Rosenzweig and Amon, 1955; Wernick and Starr, 1968; Kelly-Ballweber and Dobie, 1984; McPherson et al., 1989). Binaural interaction is observed in the human brainstem auditory evoked potentials between 5 and 8 ms and accounts for approximately 14–23% of the expected amplitude of the binaural evoked potential (Dobie and Berlin, 1979; Wrege and Starr, 1981; McPherson et al., 1989). Binaural interaction in the middle-latency auditory evoked potentials is observed between 20–40 ms and approaches about 50% of the expected amplitude of the auditory evoked potentials (Dobie and Norton, 1980; Berlin et al., 1984; McPherson et al., 1989). Berlin et al. (1984) has reported the presence of binaural interaction in the long-latency auditory evoked potentials in the human without detailing its extent.

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The purpose of this study was to examine at the same recording session auditory evoked potentials extending over a time domain of 250 ms encompassing brainstem, middle- and long-latency components.

Methods

Seventeen subjects between the ages of 19 and 28 years were used in this study. Each subject had normal pure tone hearing thresholds and a negative history of ear disease within the past twelve months. Middle ear acoustic impedance measurements were normal. Informed consent was obtained from each subject.

Auditory evoked potentials were recorded between electrodes at Cz (positive) and a non-cephalic site overlying the seventh cervical vertebra (Cvii) on the skin. A ground electrode was placed at Fpz. Brain potentials were amplified and filtered between 10 and 3000 Hz (3 dB down, 6 dB/octave) for both the brainstem auditory evoked potentials and the middle-latency auditory evoked potentials, and between 1 and 500 Hz for the long-latency auditory evoked potentials (3 dB down, 6 dB/octave). Rarefaction acoustic clicks using a 100 microsecond duration were presented at 11.1/s for both the brainstem auditory evoked potentials and the middle-latency auditory evoked potentials, and at

1.7/s for the long-latency auditory evoked potentials. The stimulus was presented at 60 dB above monaural threshold for wave V of the brainstem auditory evoked potentials across all conditions (McPherson et al., 1989). In addition, the threshold for wave V did not exceed 15 dBnHL, was equal for both ears and wave V demonstrated latencies within 0.25 ms between each ear. Peak equivalent sound pressure levels ranged from 91 dB peSPL to 101 dB peSPL. A 10 ms sample was obtained for the brainstem auditory evoked potentials, a 100 ms sample for the middle-latency auditory evoked potentials, and 300 ms for the long-latency auditory evoked potentials. Two samples consisting of 2000 trials each for the brainstem auditory evoked potentials, 1000 trials each for the middle-latency auditory evoked potentials and 500 trials each for the long-latency auditory evoked potentials were obtained for right monaural, left monaural and binaural stimulus presentations. The stimulus sequence required approximately 2 h of test time per subject.

Insert receivers (Viennatone ME22-21) were switched between ears according to a random number assigned each subject. In addition, calibration for intensity and phase were completed before, during and following this study to minimize the role of systematic differences in stimuli applied to the ears in binaural interaction.

The monaural waveforms were digitally added to obtain the sum of the monaural responses (e.g. right ear + left ear). The binaural interaction component was determined by subtracting the binaural evoked waveforms from the sum of the monaural waveforms. Grand average waveforms were constructed by averaging across all subjects for each condition.

The latencies of the brainstem auditory evoked potentials were obtained for waves I through V for each subject. The latencies for both the middle-latency auditory evoked potentials and the long-latency auditory evoked potentials were labeled according to their polarities and approximate latencies. Peak-to-following trough amplitudes were measured for waves I, II, III, IV and V. Baseline-to-peak measurements were made for the amplitudes of N10, N20, P30, N40, N90, P140 and N200. Since the negative and positive peaks did not show a sustained slow potential shift the baseline-to-peak measurement was used to provide information about each component An upward deflection in the binaural interaction waveform occurred when the sum of the monaural evoked potentials was in greater amplitude than the binaural evoked potentials. The converse was true for downward deflections.

Left-right ear differences in monaural evoked potentials were evaluated by subtracting the left monaural evoked potentials from the right monaural evoked

TABLE I

Means and standard deviations of the latencies (ms) of the auditory evoked potentials

	AER Component	Right Ear	Left Ear	Sum of RE+LE	Binaural	BIC
I	Mean	1.64	1.57	1.56	1.61	
	SD	(0.10)	(0.07)	(0.08)	(0.09)	
II	Mean	2.88	2.84	2.84	2.86	
	SD	(0.21)	(0.13)	(0.17)	(0.18)	
III	Mean	3.73	3.76	3.73	3.76	
	SD	(0.21)	(0.12)	(0.14)	(0.13)	
IV	Mean	4.95	4.89	4.95	5.02	
	SD	(0.28)	(0.17)	(0.28)	(0.18)	
V	Mean	5.72	5.73	5.70	5.76	
	SD	(0.22)	(0.23)	(0.18)	(0.19)	
VI	Mean	7.29	7.33	7.49	7.46	7.34
	SD	(0.24)	(0.20)	(0.23)	(0.19)	(0.58)
N10	Mean	10.63	10.58	11.14	10.21	
	SD	(0.79)	(0.68)	(0.97)	(0.27)	
N20	Mean	21.47	16.84	17.47	16.86	16.27
	SD	(1.52)	(1.64)	(1.69)	(0.90)	(2.69)
P30	Mean	29.95	28.75	29.32	30.19	27.12
	SD	(1.79)	(2.70)	(2.27)	(1.87)	(3.08)
N40	Mean	47.81	48.09	50.62	50.05	39.38
	SD	(0.06)	(2.30)	(7.35)	(1.98)	(3.80)
N90	Mean	85.46	85.63	83.20	79.82	
	SD	(10.03)	(8.27)	(19.16)	(7.43)	
P140	Mean	147.27	145.78	141.87	147.72	152.15
	SD	(18.97)	(14.98)	(21.13)	(14.67)	(10.92)
N200	Mean	220.72	234.26	222.46	234.45	
	SD	(32.91)	(22.07)	(32.24)	(22.61)	

potentials. Means and standard deviations were used to describe the results of this study. Prior to performing iterative t-tests, a multi-analysis of variance (MANOVA) was used to establish the validity of using iterative t-tests. Multiple t-test ($N = 144$) were used to evaluate the difference between the binaural interaction of various components of the auditory evoked potentials. Both the Geisser and Greenhouse statistic (1958) and the Bonferroni t-statistic (Bailey, 1977) were used to evaluate a priori iterative t-tests. A conservative significance level of $P < 0.0001$ is used throughout the results to qualify as statistically significant. Probability levels less than our criteria are also listed in Table III.

Results

Latency and amplitude measurements (Tables I and II) of the auditory evoked potentials are in good agreement with other studies of auditory evoked potentials (Picton et al, 1974; Mendel and Wolf, 1983). In addition, the description of binaural interaction in the brainstem auditory evoked potentials and the middle-latency auditory evoked potentials are similar to those described in our previous studies (Wrege and Starr, 1981; McPherson et al., 1989). The grand average waveforms are shown in Figs. 1–3 and illustrate the time domain of the binaural interaction component relative to the auditory evoked potentials.

Descriptive statistics are seen in Tables I and II and were obtained from individual recordings for each subject. The values presented in the result section were obtained from the group grand average waveforms (Figs. 1–3).

Monaural responses

The monaural evoked brainstem potentials are asymmetrical at 3.4 ms (right ear response > left ear response) at the time of the trough of wave II, at 3.3 ms at the time of wave IV, and between 5.4 and 6.3 ms (right response ear > left ear response) corresponding to the time of wave V and the following trough, and at 7.1 ms (left ear response > right ear response) corresponding to wave VI. Such amplitude asymmetries cannot be seen in the grand average since they were quite small amounting to less than 4% of the amplitude of the averaged waveforms. The extent of these asymmetries are similar to the differences encountered as residual baseline variability in the absence of auditory stimulation. The baseline variability was determined by repeat testing of one subject in the absence of the acoustic stimulation. Differences between the 'averages' measured peak-to-peak were approximately

TABLE II

Means and standard deviations of the amplitudes (μV) and percent binaural interaction of the auditory evoked potentials

	AER Component	Right Ear	Left Ear	Sum of RE + LE	Binaural	Percent BIC
I	Mean	0.194	0.196	0.338	0.312	8.50
	SD	(0.08)	(0.052)	(0.107)	(0.109)	(6.36)
II	Mean	0.131	0.094	0.136	0.127	5.23
	SD	(0.23)	(0.068)	(0.086)	(0.078)	(4.57)
III	Mean	0.242	0.228	0.389	0.362	7.65
	SD	(0.136)	(0.154)	(0.193)	(0.185)	(8.97)
IV	Mean	0.608	0.611	0.842	0.633	19.51
	SD	(0.483)	(0.48)	(0.667)	(0.456)	(16.42)
V	Mean	0.586	0.631	0.984	0.812	16.23
	SD	(0.312)	(0.443)	(0.538)	(0.397)	(8.32)
VI	Mean	0.469	0.47	0.977	0.759	21.01
	SD	(0.122)	(0.209)	(0.324)	(0.211)	(7.09)
N10	Mean	0.416	0.408	0.995	0.727	28.66
	SD	(0.154)	(0.405)	(0.692)	(0.717)	(10.98)
N20	Mean	0.458	0.396	0.713	0.455	35.71
	SD	(0.156)	(0.156)	(0.295)	(0.186)	(6.99)
P30	Mean	0.323	0.351	0.795	0.443	43.56
	SD	(0.149)	(0.158)	(0.39)	(0.256)	(18.5)
N40	Mean	0.426	0.360	0.758	0.389	48.15
	SD	(0.266)	(0.169)	(0.393)	(0.243)	(9.12)
N90	Mean	4.035	3.693	5.219	3.042	36.22
	SD	(1.881)	(1.562)	(2.545)	(1.821)	(20.38)
P140	Mean	3.757	4.259	7.791	4.416	38.42
	SD	(1.547)	(1.329)	(3.268)	(2.174)	(17.36)
N200	Mean	4.514	4.457	8.841	4.976	31.61
	SD	(2.947)	(2.678)	(7.692)	3.329)	(18.08)

0.2 μV consistent with the differences encountered between responses to left and right ear stimulation to click stimuli.

Monaural responses represented as left-right ear differences in the amplitude of the P30 and N40 components of the middle-latency auditory evoked potential were not statistically significant.

Monaural ear difference in the amplitude of the long-latency auditory evoked potentials occurred at about 140 ms (left ear response > right ear response) but the difference was not statistically significant.

Binaural responses

At the times of waves I, II and III binaural interaction is slight accounting for up to 8% of the sum of the monaural responses (Table II). We consider this measure to represent the 'noise' level for those measurements and not true binaural interaction. In contrast, at the times of waves IV, V and VI binaural interaction could amount up to 21% of the sum of the monaural responses (Table II). The percent binaural interaction peaking at the time of wave VI is significantly greater than the binaural interaction occurring at waves I, II and III (Table III). The peak of the binaural interaction component can be variable due to the relatively gradual change in the amplitude of the binaural interaction over the time domain between 5 and 9 ms. The binaural interaction component peaking around wave VI is actually quite broad. It begins at about 5 ms latency (at the time of wave IV), peaks at 7.35 ms, and returns to baseline at about 9 ms (Fig. 1). The maximum binaural interaction occurring at 7.35 ms represents a 21% reduction of the monaural reposes (Fig. 1).

Binaural interaction in the middle-latency auditory evoked potentials had three maxima: the first peaking at 16.3 ms, the second peaking at 27.1 ms and the third peaking at 39.4 ms. The maximum binaural interaction of these three responses occurs at 39.6 ms and accounts for 49.3% of the sum of the monaural responses

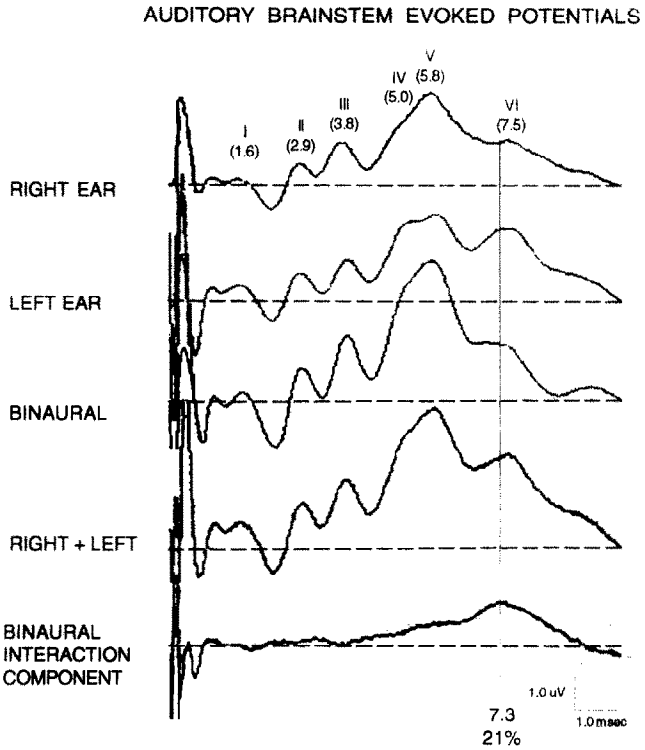

Fig. 1. The grand average of the brainstem auditory evoked potentials across 17 subjects for the sum of the monaural brainstem auditory evoked potentials, the binaural brainstem auditory evoked potentials and the binaural interaction component. Note the long duration of the binaural interaction peaking at 7.3 ms. The binaural response visually appears larger than the sum of the monaural responses due to the presence of a slow sustained potential.

(Fig. 2). Significant differences in the percent binaural interaction occurred for the middle-latency auditory evoked potentials (N20, P30 and N40) when compared to the binaural interaction of the auditory brainstem evoked potential (wave VI) (Table III).

Binaural interaction in the long-latency auditory evoked potentials had several peaks at 83, 152 and 234

TABLE III

t-Test for mean differences between binaural interaction in the auditory evoked potentials extending from the brainstem to the cortex

	II	III	IV	V	VI	N10	N20	P30	N40	N90	P140	N200
I	0.126	0.025	*	*	*	*	*	*	*	*	*	*
II		0.116	0.003	0.001	*	*	*	*	*	*	*	*
III			0.009	0.005	*	*	*	*	*	*	*	*
IV				0.189	0.752	0.049	0.004	*	*	0.005	0.004	0.253
V					0.647	0.05	0.0008	*	*	0.031	0.009	0.171
VI						0.034	*	*	*	0.012	0.001	0.052
N10							0.051	0.010	*	0.226	0.013	0.612
N20								0.147	*	0.924	0.567	0.445
P30									0.252	0.281	0.422	0.094
N40										0.022	0.083	0.003
N90											0.763	0.494
P140												0.372

A MANOVA resulted in F = 1177.20; df = 1,16 and $P < 0.0000$; * Significant differences in binaural interaction for $P < 0.0001$; The Geisser and Greenhouse (1958) and Bonferroni (Bailey, 1977) statistics were used for iterative *t*-tests.

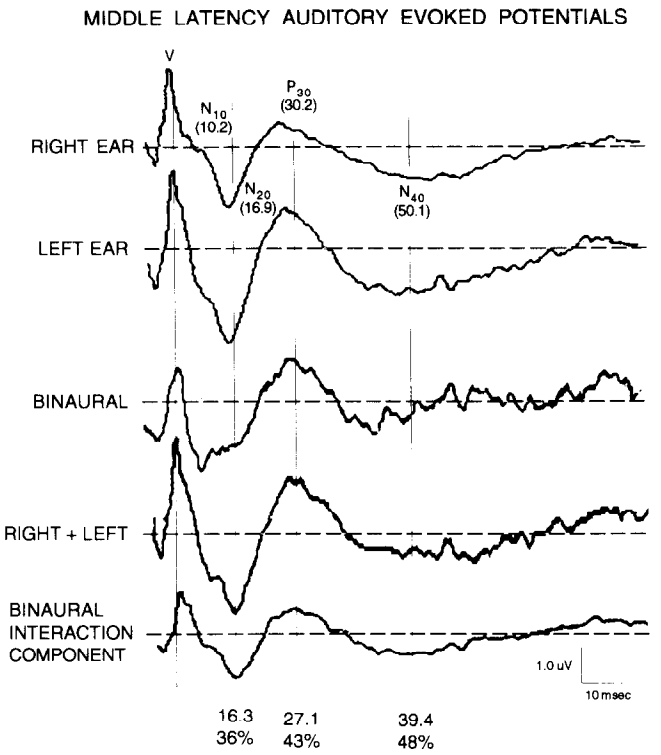

Fig. 2. The grand average of the middle-latency auditory evoked potentials across 17 subjects for the sum of the monaural middle-latency auditory evoked potentials, the binaural middle-latency auditory evoked potentials and the binaural interaction component. The binaural interaction appears with each of the major components and amounts to almost 50% of the sum of the monaural potentials for the N40 component.

ms. The interaction component peaking at 152 ms is quite broad lasting more than 100 ms. The maximal interaction in this time domain amounted to 47.6% of the sum of the monaural responses (Fig. 3). The binaural interaction for P140 is significantly greater than for wave VI of the auditory brainstem evoked potentials (Table III). No significant differences were found between the degree of binaural interaction in the middle-latency (N10-N40) and long-latency components (N90-P200).

Discussion

The results of the study have bearing on both monaural response differences in auditory evoked potentials as well as on the extent of binaural interaction occurring at different periods following transient click stimulation.

Ear differences

One of the issues raised in the description of binaural interaction, especially in the auditory brainstem evoked potentials, was the role of ear asymmetries of auditory brainstem responses that influence binaural interaction (Decker and Howe, 1981; Dobie, 1982; Levine and McGaffigan, 1983). The effects of ear asymmetries on the binaural interaction component were assessed by subtracting the left ear monaural response from the right ear monaural response. There are small asymmetries present at the time of waves III (3.37 ms), V (5.42 ms) and VI (7.12 ms) of the auditory brainstem evoked potential amounting to approximately 3% amplitude difference that did not achieve statistical significance. We consider these asymmetries in the auditory brainstem evoked potentials to monaural stimulation to reflect residual 'noise' of the recordings. Amplitude asymmetries observed for the middle-latency auditory evoked potentials amounted to approximately 4% that again was not statistically significant. Finally, an asymmetry present in the long-latency auditory evoked potentials at about 215 ms amounting to a decrease of approximately 9% that also did not achieve statistical significance. Peronnet and Michel (1977) found a 12% monaural difference between the two ears in the amplitude of the components occurring between 100 and 200 ms.

Binaural interaction

Binaural interaction occurred over the entire time domain of the auditory evoked potential from brainstem to cortically generated events (Fig. 4). The form of binaural interaction was always a decrease of the

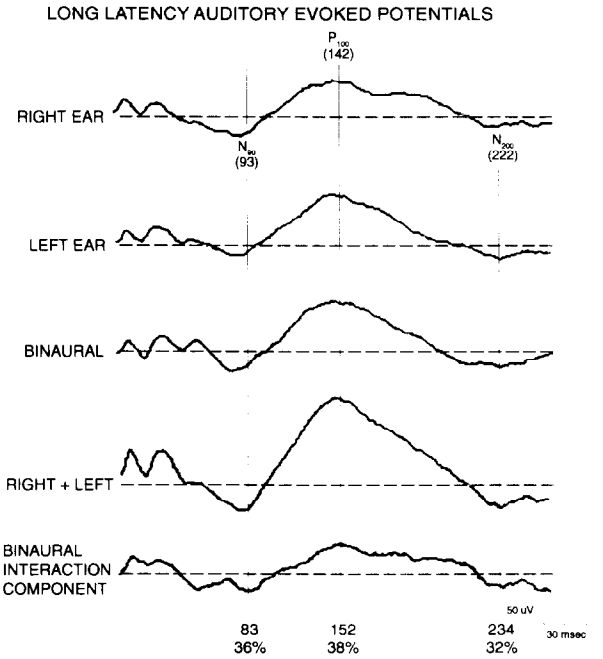

Fig. 3. The grand average of the long-latency auditory evoked potentials across 17 subjects for the sum of the monaural long-latency auditory evoked potentials, the binaural long-latency auditory evoked potentials and the binaural interaction component. The binaural interaction component appears with each component amounting to approximately 40% of the sum of the monaural responses.

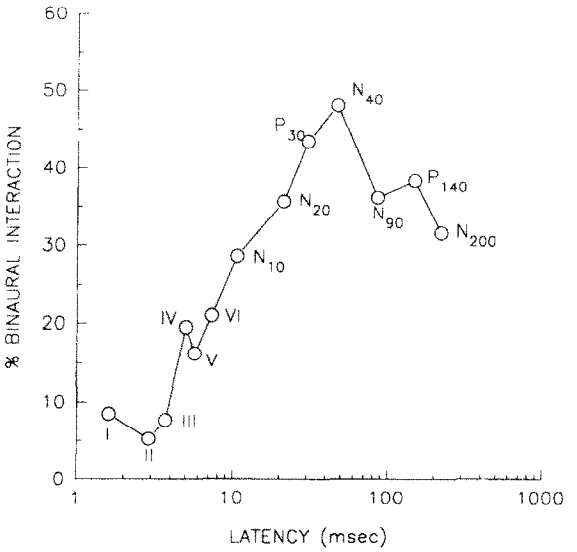

Fig. 4. Percent binaural interaction at each of the major components of the auditory evoked potentials (from tables I and II).

binaural response compared to what would have been expected by summing the monaural evoked potentials (Fig. 5). There is evidence that both inhibition and excitation accompany binaural processes at the single cell level (Butler, 1969; Brugge et al., 1973; Moore, 1991). It is attractive to consider that the evoked potential changes accompanying binaural interaction are secondary to specific excitatory and inhibitory single unit events but must await evidence of such specific relationships. We will use the terms 'inhibition' and 'facilitation' to designate the type of binaural interaction in the remainder of the discussion with awareness that the terms are descriptive. 'Inhibition' is considered to be a relative decrease of the evoked potentials to binaural stimulation compared to the sum of the monaural responses. Binaural interaction appears as an 'inhibition' over a considerable time domain of the brainstem auditory evoked potentials beginning at 4.4 ms, peaking at 7.3 ms and ending at about 9 ms corresponding in time to the appearance of waves IV, V, VI and VII. The onset of binaural interaction occurs at a time when activity is engaging the superior olivary complex, the site where binaural processes are initiated in the auditory pathway as well as the site of generation for waves IV and V (Jewett and Williston, Starr and Hamilton, 1976; Møller et al., 1981; McPherson et al., 1989). The finding that binaural interaction peaks at the time of wave VI and continues until the time of wave VII could represent continuing binaural processing at the superior olive rather than involvement of the generators for waves VI and VII (which have not yet been clearly identified).

In the middle-latency domain, there is a significant increase in binaural interaction (to 40%) relative to the brainstem potentials (16%). The increase occurs at: 1)

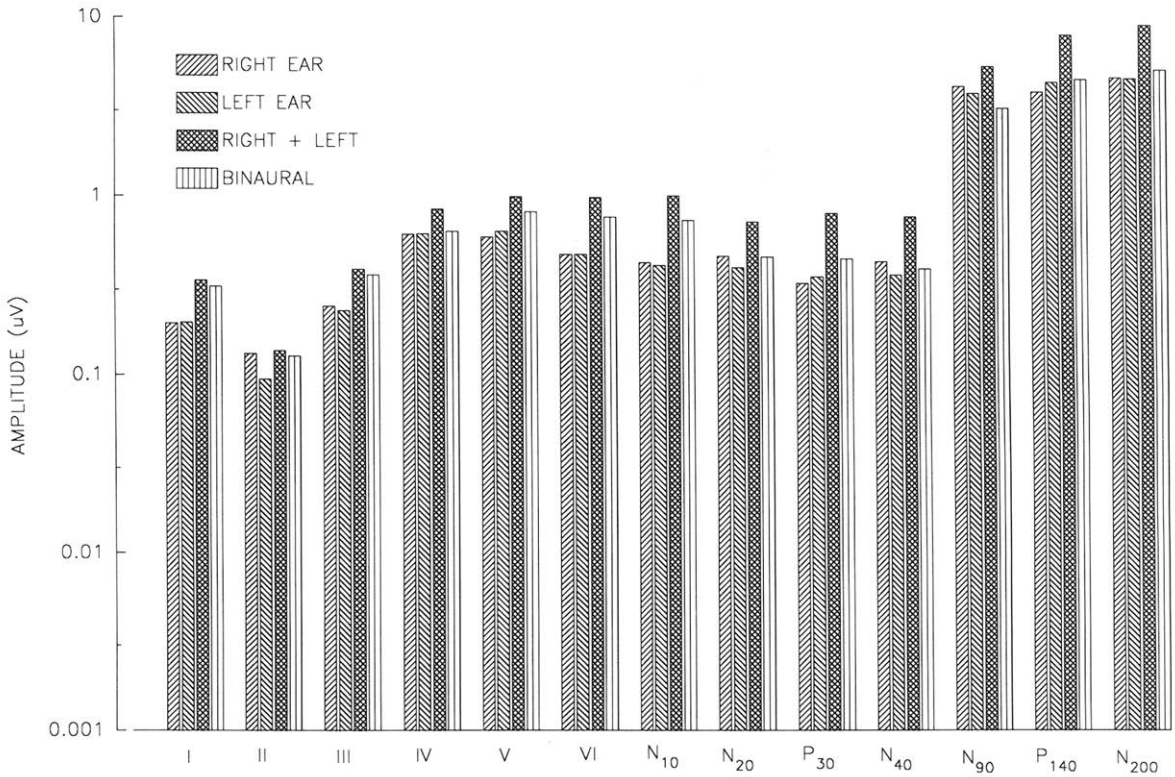

Fig. 5. Bar chart of the absolute amplitudes for the right and left monaural responses, sum of the monaural responses (right ear + left ear) and binaural responses (from table II).

the time of the N10 component and appears as 'inhibition' peaking at 16 ms; and 2) the time of the P30 and N40 components, taking the form of 'inhibition' peaking at 27 ms and 32 ms, respectively. These results are comparable to the findings of Dobie and Norton (1980). Although the precise generators of the middle-latency auditory evoked potentials are not known, Kraus et al. (1982) reported that N20 and P30 are generated separately with N20 deriving from subcortical structures and N40 is generated in auditory cortex. Lott et al. (1986) in a case of hydranencephaly found that the earliest middle-latency auditory components N10 and P18 were present and that N20, P30 and N40 components of the middle-latency auditory evoked potentials were absent. There was imaging evidence of an intact brainstem and thalamic structures with small amounts of posterior temporal lobe present. Lott et al. (1986) interpret this as suggesting that both the brainstem auditory evoked potential and the earliest components of the middle-latency auditory evoked potential (N10 and P18) depend on intact structures up to and including the thalamus. Additional research suggests that the middle-latency auditory evoked potentials are believed to represent activity of the medial geniculate and the polysensory nuclei of the thalamus (Picton et at., 1974). Studies of the magnetic field evoked responses (Tihonen et al., 1989) in the time domain of the middle-latency potentials (Pelezzone et al., 1987) suggest that the components at 30, 50 and 65 ms latency are generated by supratemporal auditory cortex and not thalamus.

In the long-latency domain binaural interaction occurs around the N90 component with an initial low amplitude bimodal peak at 63 and 83 ms. A long-lasting interaction then follows which peaks at 150 ms at the time of the P140 component. These interactions are all 'inhibitory' in form. The extent of binaural interactions in the long-latency potentials is approximately 40%, similar to that found in the middle-latency time period.

These experiments outline the temporal sequence of binaural processes to a transient click stimulus using the appearance of binaural interaction as their marker. Binaural processes begin at 4.4 ms and can be identified through the end of the 200 ms analysis window. The form of the binaural interaction is a relative decrease ('inhibition') of the evoked potentials to binaural stimulation compared to the sum of monaural responses. These evoked potential results suggest that inhibition may be the major mechanism utilized in binaural processes. Furthermore, the finding of an increase in binaural interaction at middle- and long-latency potentials relative to brainstem potentials suggests that the relative allotment of the auditory pathway to binaural processes is greater during cortical activity than during brainstem activity.

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