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BRAINSTEM AUDITORY EVOKED RESPONSES IN MAN:

I. EFFECT OF STIMULUS RISE-FALL TIME AND DURATION

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ABSTRACT

Short latency (under 10 msec) evoked responses elicited by bursts of white noise were recorded from the scalp of human subjects. Response alterations produced by changes in the noise burst duration (on-time) inter-burst interval (off-time), and onset and offset shapes are reported and evaluated. The latency of the most prominent response component, wave V, was markedly delayed with increases in stimulus rise-time but was unaffected by changes in fall-time. Increases in stimulus duration and therefore loudness resulted in a systematic increase in latency, probably due to response recovery processes, since this effect was eliminated with increases in stimulus off-time. The amplitude of wave V was insensitive to changes in signal rise-and-fall times, while increasing signal on-time produced smaller amplitude responses only for sufficiently short off-times. It is concluded that wave V of the human auditory brainstem evoked response is solely an onset response.

During the first 10 msec following the delivery of an auditory stimulus a series of electrical events, reflecting the activation of the eighth nerve and brainstem auditory centers, can be recorded via scalp electrodes in man (Jewett and Williston, 1971; Jewett, Romano and Williston 1970; Sohmer and Feinmesser 1967). Seven positive evoked response components have been identified (waves I-VII, according to the convention of Jewett and Williston 1971) which appear to be generated by sequential activation of the brainstem auditory nuclei and tracts. Since the responses of these centers are to some extent temporally co-extensive, it has not been possible to identify a given component with a single nucleus or tract, although a plausible schema has been devised on the basis of comparisons between simultaneous extracellular and extracranial recordings (Jewett 1970; Lev and Sohmer 1972), extracranial mapping studies (Picton et al. 1974; Plantz et al. 1974), and pathological data (Starr and Achor in press). On the basis of the above studies it appears that wave I represents the volume conducted eighth nerve action potential, while wave V originates from rostral portions of the brainstem auditory tract. However, the relative contributions of each of the brainstem auditory centers to each of the response components has not been determined.

Even less is known about the functional properties of the response than about the location of its generators. Increases in signal intensity produce systemmatic and highly stable decreases in response latency and increases in response amplitude (Lev and Sohmer 1972; Jewett et al. 1972; Hecox and Galambos 1974; Picton et al. 1974; Terkildsen et al. 1973; Starr and Achor in press). Little else is

known about the acoustic dependencies of the response except that a variety of signals including tone pips, tone bursts, clicks, and noise bursts presented at repetition rates from 1 to 90 per second reliably elicit the response (Jewett and Williston 1971; Galambos et al. 1973; Picton et al. 1974). However, we are unaware of any systematic measurements of the effect of signal frequency, rise-fall time, duration or simultaneously presented maskers on response properties. This information seems essential if, as several authors have suggested (Jewett and Williston 1971; Sohmer et al. 1973; Galambos and Hecox 1974), this response is to be used as a clinical or research measure of auditory responsivity. The present experiments are part of a continuing effort to define the origins and properties of this short latency auditory response. They examine the effects of several acoustic parameters - signal duration (on-time), recovery time between the effect of one signal and the onset of the next stimulus (off-time), and rise- and fall-times - upon the latency and amplitude of wave V, the most reliably elicited response component.

METHODS

Subjects

Six female subjects, aged 19 to 28 years, participated in these experiments. The only selection criterion employed was that the subjects have normal hearing as determined by standard audiological procedures. All of the subjects were experienced auditory observers, having participated in previous experiments, and all but NS (a co-author) were paid for their participation.

Apparatus

Differential recordings were made between the vertex and the right mastoid with Beckman Ag-AgCl electrodes. The left mastoid served as ground. The electrodes were led to a differential A.C. preamplifier (Grass Model P15) and the signals, after further amplification (Tektronix FM122), were electronically averaged (Nicolet Model 1072). The bandpass of the system was 100-3000 Hz, and the overall amplification was approximately 400,000. The onset of signal averaging was synchronized to the stimulus onset and continued for 10.24 msec. At the end of each block of stimuli the averaged waveform was displayed and an interrogated address selector was used to obtain the digital address of the response components of interest. These digital addresses were then converted to latency values by multiplying the digital address by the 40μsec dwell time per address. Finally, each tracing was printed out by a Hewlett Packard Model 7035B X-Y plotter for permanent records.

The stimuli used in this study were white noise bursts (20-20,000 Hz) of varying intensity, duration, and rise-fall times. The noise source was a Bruel and Kjaer Type 1024 Sine Random Generator. Stimulus intensity was regulated by a Hewlett Packard Model 350-D attenuator while the rise-fall, repetition rate, and duration of the signals were determined by a Grason Stadler Model 1287B electronic switch, and two Grason Stadler Model 1216A 100-sec timers. Stimuli were presented monaurally to the right ear via Clark Model 100A ear-phones in a sound-treated room (Industrial Acoustic Company, Model 400A).

Procedure

At the beginning of each session the subject's threshold was determined, by the method of limits, for a 30 msec white-noise burst presented 16 times per sec., with instantaneous rise-fall times. All intensities are referenced to the threshold intensity of this standard signal for a given subject within a recording session. The peak to peak voltage of the 30 msec test signal at the average threshold across all observers was equal to that of 15 dB SPL continuous white noise. Insofar as possible, all the data within a particular comparison (e.g., rise-fall time) were collected in a single session. However, the threshold determinations and the evoked responses were sufficiently reliable to permit comparisons across sessions also.

In the first experiment, responses were collected from four subjects to the 60 dBSL, 30 msec signal, presented 16 times per sec, with the following rise-fall times: 0, 1, 2.5, 5, and 10 msec. Data were

also obtained from one of the subjects with shorter stimulus durations, 5 msec and 2 msec, at several rise-fall times. To assess the contribution of variations in fall-time independently of changes in rise-time, a custom-designed electronic switch was used to present all the possible combinations of 0 and 5, and 0 and 1 msec rise and fall-times for the 30 and 2 msec signal, respectively.

In the second experiment four subjects received 60 dB monaural 16 per sec noise bursts of various durations: 0.5, 2, 5, 20 and 30 msec (instantaneous rise-fall). Since changes in stimulus duration at a fixed repetition rate affect both the stimulus on-time and off-time, these two parameters were subsequently varied independently. The third experiment was thus the parametric examination of on and off-times with three off-times--15, 30, and 60 msec--and four on-times--2, 5, 20 and 30 msec--presented in all possible combinations.

The latency, and when appropriate the amplitude, of the most prominent response component, wave V, were taken as the dependent variables in all of the experiments to be described. The latency values presented throughout this paper generally refer to the mean of the three replications for each signal configuration (2048 stimulus presentations per replication), while amplitude comparisons are based upon the summed average waveforms from the three replications (thus containing responses to over 6000 stimulus presentations), since amplitudes were much more variable than latencies. All threshold measurements were obtained by the method of limits.

Test sessions lasted from one to two hours during which subjects lay quietly or slept in a bed in the sound-treated room. The

order of presentation of signals in all experiments was randomized to eliminate possible order effects.

RESULTS

Rise-fall time

The waveforms of subject VB for rise-fall times of 0, 1, 2.5, 5, and 10 msec are shown in Figure 1. Wave V is clearly discernible in all waveforms and its latency increases from 7.1 msec at a rise-fall time of 0 msec to 8.4 msec for a rise-fall time of 10 msec. A plot of the wave V latencies for this and two other subjects are shown in Figure 2. Multiple t-tests between latency values at each of the successive rise-fall times showed significant ($p<.01$) differences for all paired comparisons for each of the three subjects. The amplitude of wave V, on the other hand, did not systematically

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INSERT FIGURES 1 AND 2 HERE

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vary with rise-fall time.

The effect of rise-fall time on latency did not crucially depend upon signal duration, as shown in Figure 3 for subject GE. Although absolute latency varied with signal duration (see below), the slopes of the three functions are nearly identical (1.07, 1.00, and 0.97 for 2, 5, and 30 msec stimuli, respectively).

To assess the separate contributions of stimulus rise and fall time to the latency increase, signals at 60 dB, 30 and 2 msec were presented to two subjects with all combinations of 0 and 5, and 0 and 1 msec rise- and fall-times, respectively. The results are shown in Table I, which clearly indicates that the variation of wave V latency cannot be attributed to stimulus fall-time.

Average threshold differences, obtained from four of the subjects between the 0 and 10 msec rise-time signals did not exceed 2 dB. This value agrees well with theoretical predictions from the literature for similar stimuli (Dallos and Olsen 1964; Dallos and Johnson 1966).

INSERT FIGURE 3 AND TABLE I HERE

Signal Duration

The waveforms of subject NS for signal durations of 0.5, 2, 5, 20 and 30 msec are shown in Figure 4. A plot of the wave V latencies as a function of increasing signal duration for this subject are shown in Figure 5, along with similar data from 3 other subjects. An increase in latency of approximately 0.5 msec is obtained as duration increases from 0.5 to 30 msec.

INSERT FIGURES 4 AND 5 HERE

To assure that there was nothing unusual about the particular stimulus configuration that would interfere with temporal integration, threshold and loudness judgements were obtained from the subjects. Subjects were asked to match the loudness of a variable-intensity 30 msec signal with that of a fixed intensity 0.5 msec signal. Signals were presented alternately to the same ear until the subject felt a

match had been made; control of the 30 msec, variable-intensity signal was by the experimentor who raised or lowered its intensity according to the subject's request. There was an average decrease in threshold of 16 dB and a 22 dB increase in loudness from the short to the long signal, demonstrating that temporal integration was indeed taking place. Both threshold and loudness changes agree reasonably well with theoretical and empirical values reported in the literature (Green et al. 1957; Scharf 1974). As seen in Table II, wave V amplitude remains virtually constant over the range of duration studied. Thus, as expected on the basis of its short latency, there is no simple correlate of temporal integration in the wave V response.

INSERT TABLE 2

At fixed repetition rates increasing the signal duration (on-time) will diminish the interval between its offset and the onset of the following signal (off-time). To determine the relative roles played by signal on- and off-times in the effect shown in Figure 5, four duration values and 3 recovery times were presented in all combinations. The overwhelming importance of off-time is shown in Figure 6 and Table III. Increasing off-time produces shorter latencies, while increasing on-time has an effect only when off-time is sufficiently short.

There was a significant effect of off-time ($F_{2,8} = 101$, $p<.01$), on-time ($F_{3,12} = 33$, $p<.01$) and their interaction ($F_{6,24} = 3.8$, $p<.05$)

on response latency. It is noteworthy that the effect of on-time diminishes markedly at sufficiently long off-times. The amplitude of wave V was affected only by signal off-time ($F_{2,12} = 6.97$, $p<.01$) but was unaffected by on-time. This explains why wave V amplitude appears not to vary with stimulus duration in Table II: with a repetition rate of 16 per second and a maximum signal duration of 30 msec, off-time never falls below 30 msec.

INSERT FIGURE 6 AND TABLE III HERE

DISCUSSION

These experiments clearly demonstrate that latency and amplitude of the brainstem auditory evoked response are established exclusively at the onset of the stimulus, not at its offset, nor by its duration, provided sufficient time is allowed for response recovery. These results closely parallel similar demonstrations that stimulus onset characteristics control the eighth nerve response (Goldstein and Kiang, 1958), the mid-latency evoked responses (Skinner and Antinoro 1971; Lane, Kupperman and Goldstein 1971; Reiter and Hogan 1973) and the late (50-500 msec) components (Lamb and Grahan, 1967; Skinner and Jones 1968; Onishi and Davis 1968). The only exception to this rule is that increases in stimulus duration up to about 30 msec can enhance the amplitude of the late components (Onishi and Davis 1968). One interesting difference between these and the earlier results is that wave V amplitude does not decrease with increasing stimulus rise time (Fig. 7), in contra-distinction to both eighth nerve (Goldstein and Kiang 1958), and middle latency responses (Skinner and Antinoro 1971) over the range of values used in this study. This difference may be due to our use of noise bursts rather than the tone bursts employed in the middle latency evoked response studies, and higher signal intensities than those employed to obtain the eighth nerve data. Settling such points of difference will require information not now available.

If we presume that a given stimulus can mask the stimulus that follows it, then our reported effects on wave V of stimulus on- and

off-time can be explained on the basis of forward masking (Hawkins and Kniazuk 1950; Rosenblith, Galambos and Hirsh 1950). For eighth nerve responses a "duration effect" and its dependence upon the amount of recovery time before the presentation of the next stimulus have been well described (Coats 1964; Eggermont and Spoor 1973); increases in masker duration and/or decreases in the amount of recovery time both depress response amplitudes and increase their latency.

The correlations between subjective loudness or threshold and the physiological responses observed in this study were poor. Thus no important threshold or loudness changes accompanied the striking shifts in latency observed with altered rise-time. In addition, the impressive increase in loudness and improvement in threshold associated with increasing signal duration actually produced little if any change in the physiological responses. One must conclude from these facts either that the "psychological processing" of the acoustic parameters varied in this study occurs above the brainstem level of the auditory pathway, or that it is mediated by brainstem cell populations not involved in the production of the brainstem evoked response.

Certain microelectrode studies on cats may be pertinent to the pattern of results reported in this study. At the superior olivary level a particular cell population is known to respond to stimulus onset with great precision: their latency, which is short, actually differs depending upon whether the stimulus at its onset produces a positive or a negative pressure wave in the ear canal (Galambos et al. 1959). This population (the so-called "time-keepers") presumably conveys information about stimulus onset promptly and accurately to

higher brainstem levels. A similar differentiation of auditory neurons into functional groups is suggested for inferior colliculus (Gersuni et al. 1971) and cochlear nucleus (Radionova 1971) neurons. here so-called "short-latency" neurons, are highly sensitive to the structure of stimulus onset but unaffected by signal duration. Gersuni et al. (1971) suggest that the short-latency neurons signal stimuli onset and perform precise short-time sound analysis, while the long-latency neurons are useful in frequency and intensity analysis. The response latencies of these short-latency neurons in cochlear nucleus, superior olive and inferior colliculus are within the range of those responses we have studied here. The latency-intensity functions of these short latency units also resemble that of the human and cat brainstem evoked potential (Lev and Sohmer 1972; Hecox and Galambos 1974) and, like the human brainstem evoked response, their latency variability is remarkably low (Hecox and Galambos 1974). The idea that short latency neurons comparable to those described in the cat may be responsible for the various waves in the human auditory brainstem response is an attractive possibility. Simultaneous recordings from scalp and intracellular electrodes implanted in "short-time constant" neurons in subhuman species should prove informative in evaluating this hypothesis.

FOOTNOTES

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Table 1. Latency of wave V, in milliseconds, as a function of signal rise and fall time for two subjects. Each value represents the mean of three replications. Signals were presented monaurally at a rate of 16 per second, at a level of 60 dBSL.

Subject	Duration (msec)	Rise Time (msec)	Fall Time (msec)			
			0	1	0	5
GE	2	0	6.5	6.6	-	-
		1	6.9	7.0	-	-
	30	0	-	-	7.1	7.2
		5	-	-	8.3	8.1
JH	2	0	6.6	6.6	-	-
		1	7.0	6.9	-	-
	30	0	-	-	7.3	7.2
		5	-	-	8.7	8.6

Table II. Mean of wave V amplitude, in microvolts, as a function of signal duration for each of four subjects. Each value represents the amplitude of the summed averaged response to three replications.

Subject	Signal Duration				
	.5	2	5	20	30
GE	1.60	1.40	1.40	1.46	1.60
KB	1.02	.93	.89	.78	1.16
NS	.77	.66	.87	.95	.96
JH	1.25	1.34	1.50	1.28	1.20
Mean	1.16	1.07	1.06	1.12	1.23

Table III. Mean of wave V amplitudes, in microvolts, as a function of stimulus on and off-time time. Each value represents the mean of three replications for each of the subjects. All signals were presented monaurally at the same spectrum level (75 dBSPL).

Subject	On-Time (msec)	Off-Time (msec)		
		15	30	60
VB	2	0.57	0.92	0.94
	5	0.54	0.88	0.98
	10	0.65	0.80	0.94
	30	0.38	0.92	0.84
NS	2	0.23	0.44	0.68
	5	0.64	0.66	0.76
	10	0.44	0.68	0.80
	30	0.55	0.78	0.73

FIGURE LEGENDS

Figure 1. Evoked responses of subject GE as a function of rise-fall time. Each tracing represents the sum of 2048 stimulus presentations, with three superimposed replications at each value of rise-fall time. Positivity to the vertex is upwards in this and all subsequent waveforms.

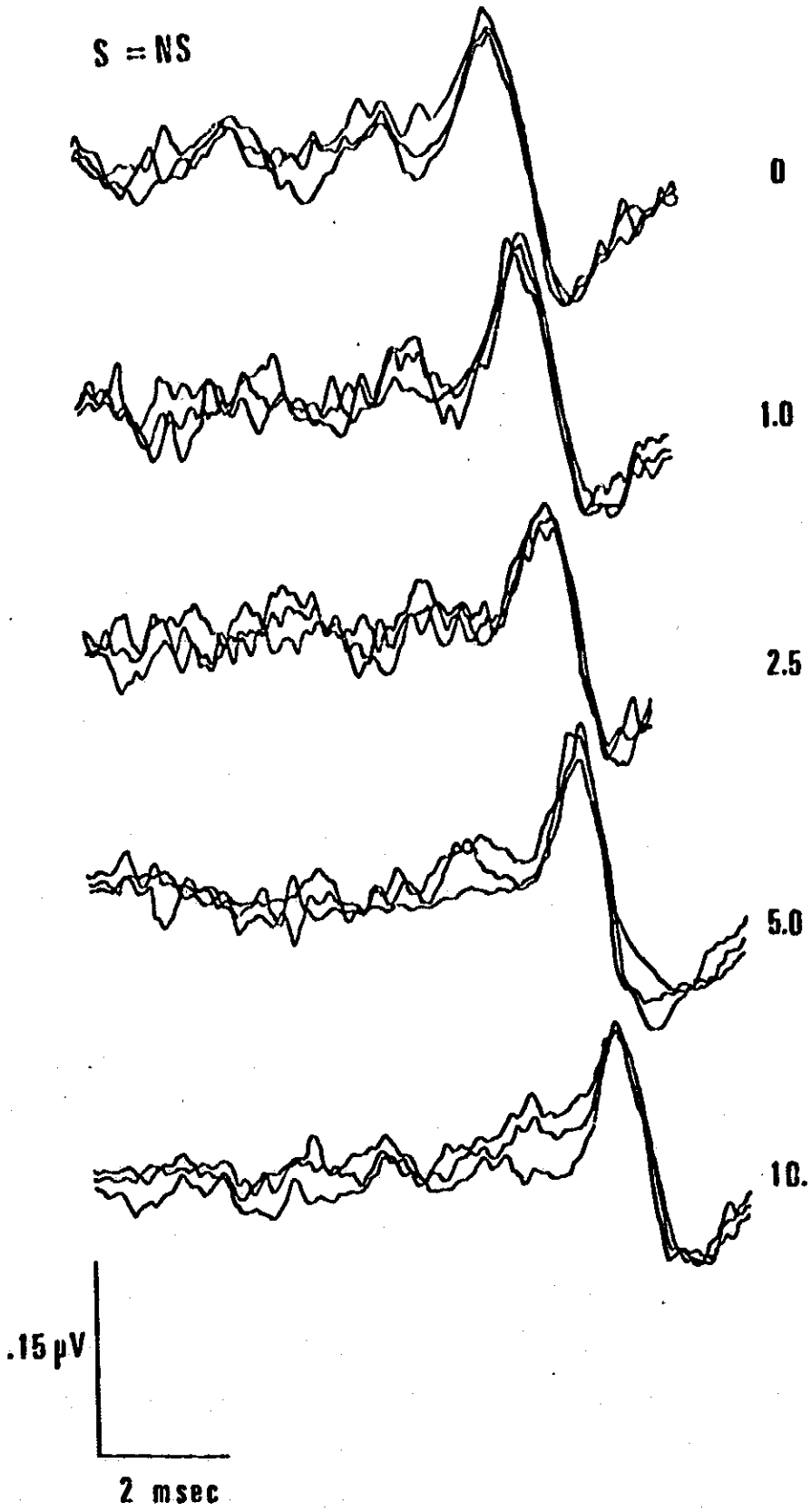
Figure 2. Latency of wave V as a function of stimulus rise-fall time for three subjects. Each value, for each subject represents the mean of three replications. The stimulus was a 75 dBSPL, 30 were white noise burst, presented 16 times per second.

Figure 3. The latency of wave V as a function of rise-fall time with duration as a parameter. Each point represents the mean of three replications. The rise-fall times are plotted on a logarithmic scale to facilitate comparisons of the slopes at each duration. The stimulus in each case was a monaural 75 dBSPL noise burst presented 16 times per second.

Figure 4. Evoked responses of subject NS as a function of stimulus duration. Each tracing represents the summed response to 2048 presentation of a monaural 75 SPL noise burst presented 16 times per second. Three superimposed replications are shown at each stimulus duration.

Figure 5. The latency of wave V as a function of stimulus duration. Each point represents the average three replications for each of the four subjects. The stimuli were as described in Figure 4.

Figure 6. The latency of wave V as a function of stimulus on and off time. Each point represents the average of three replications for each of the subjects. The stimulus was in each case a monaural noise burst whose spectrum level was fixed at 75 dBSPL, but where which rate was determined by the independent variation is stimulus on and off times depicted below.



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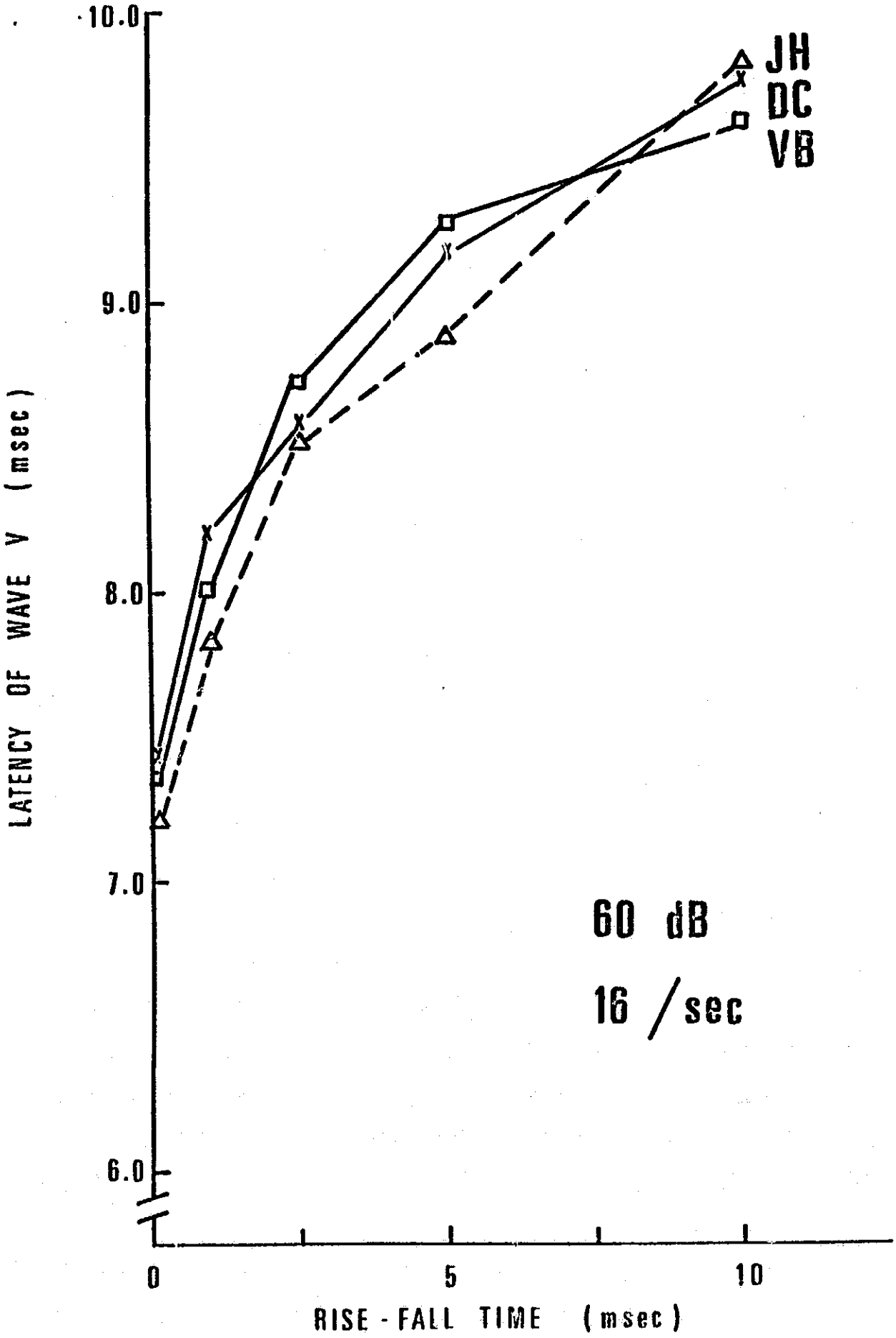
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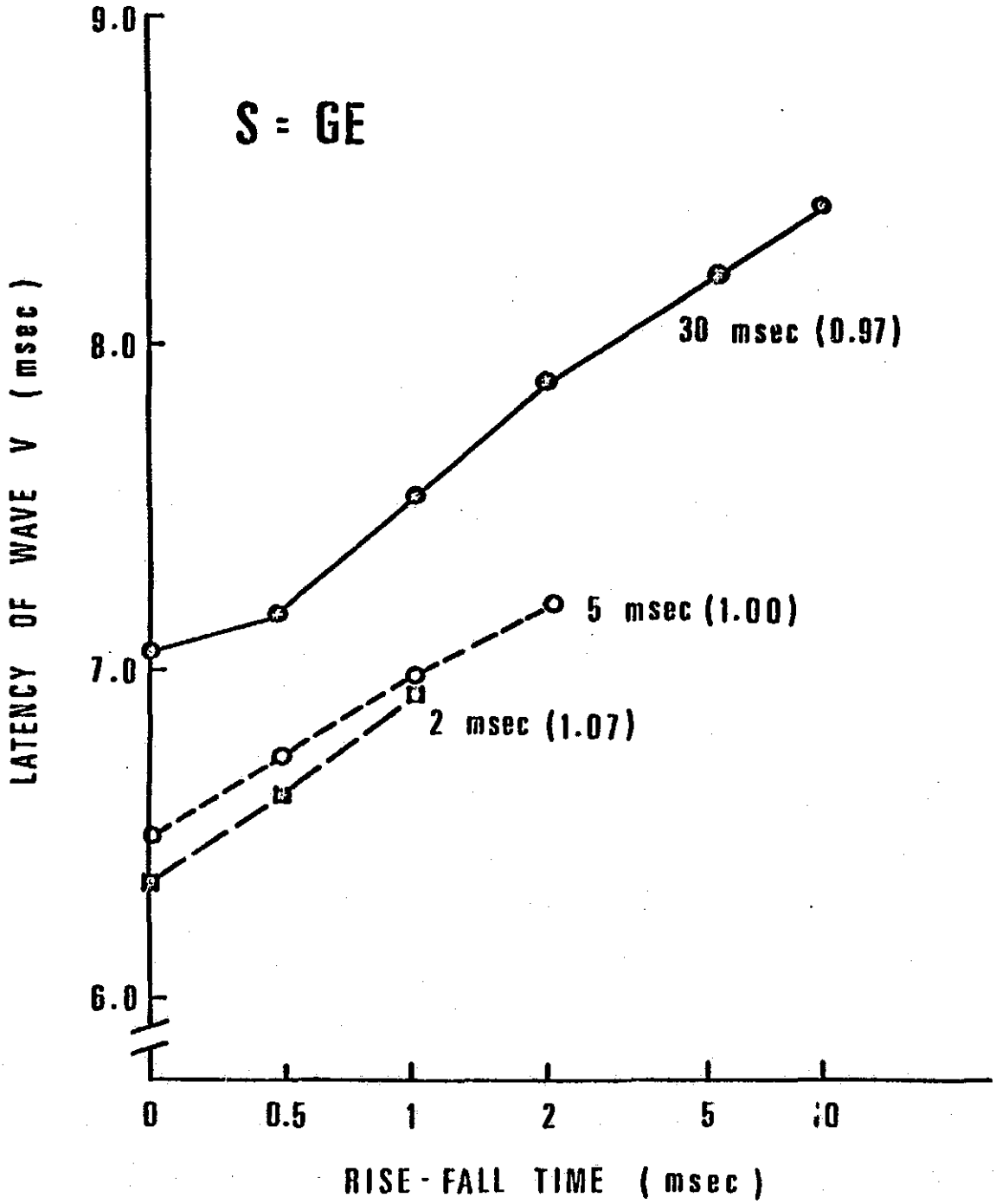
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2 msec (1.07)
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2
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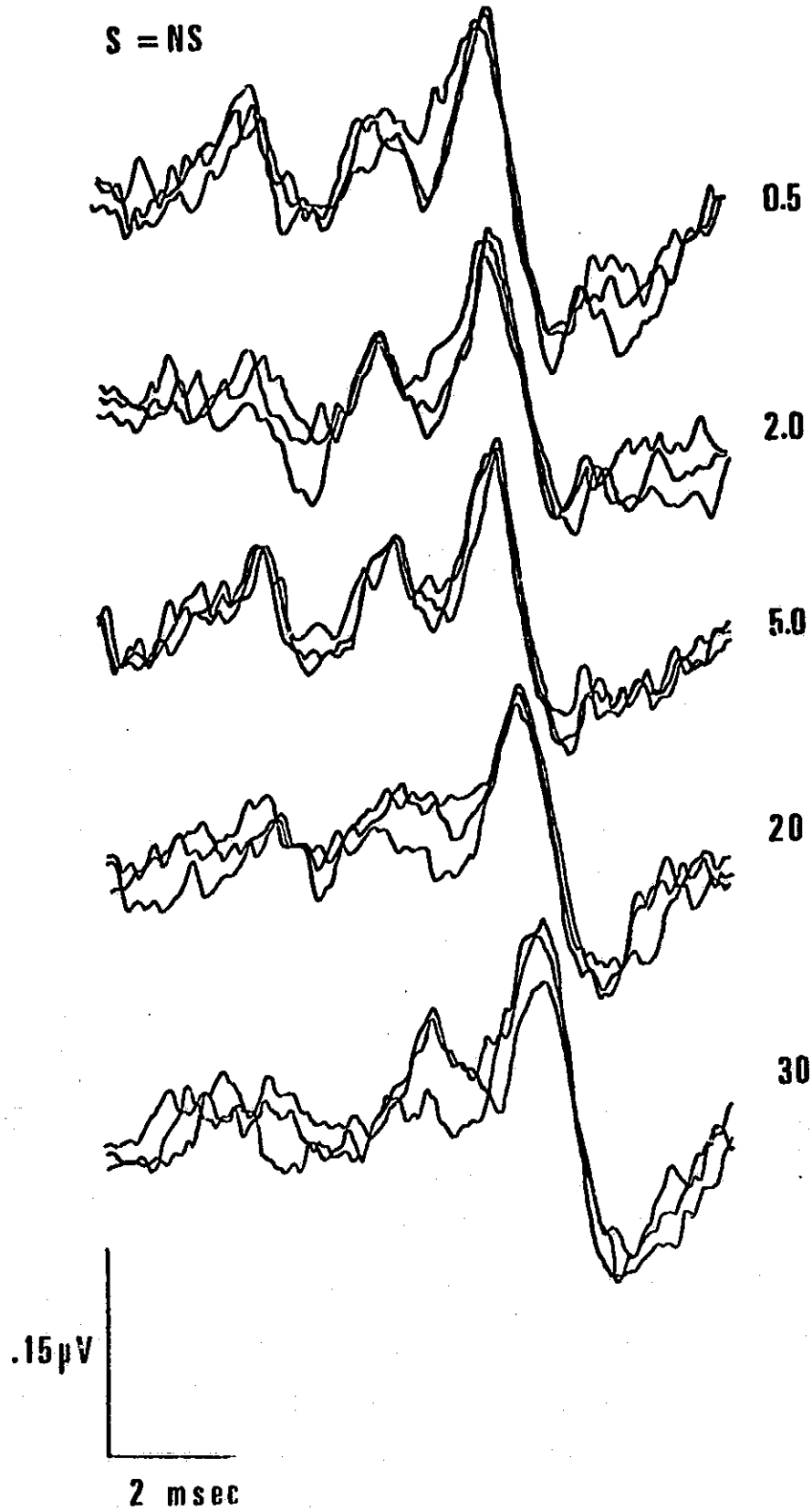
10
RISE-FALL TIME (msec)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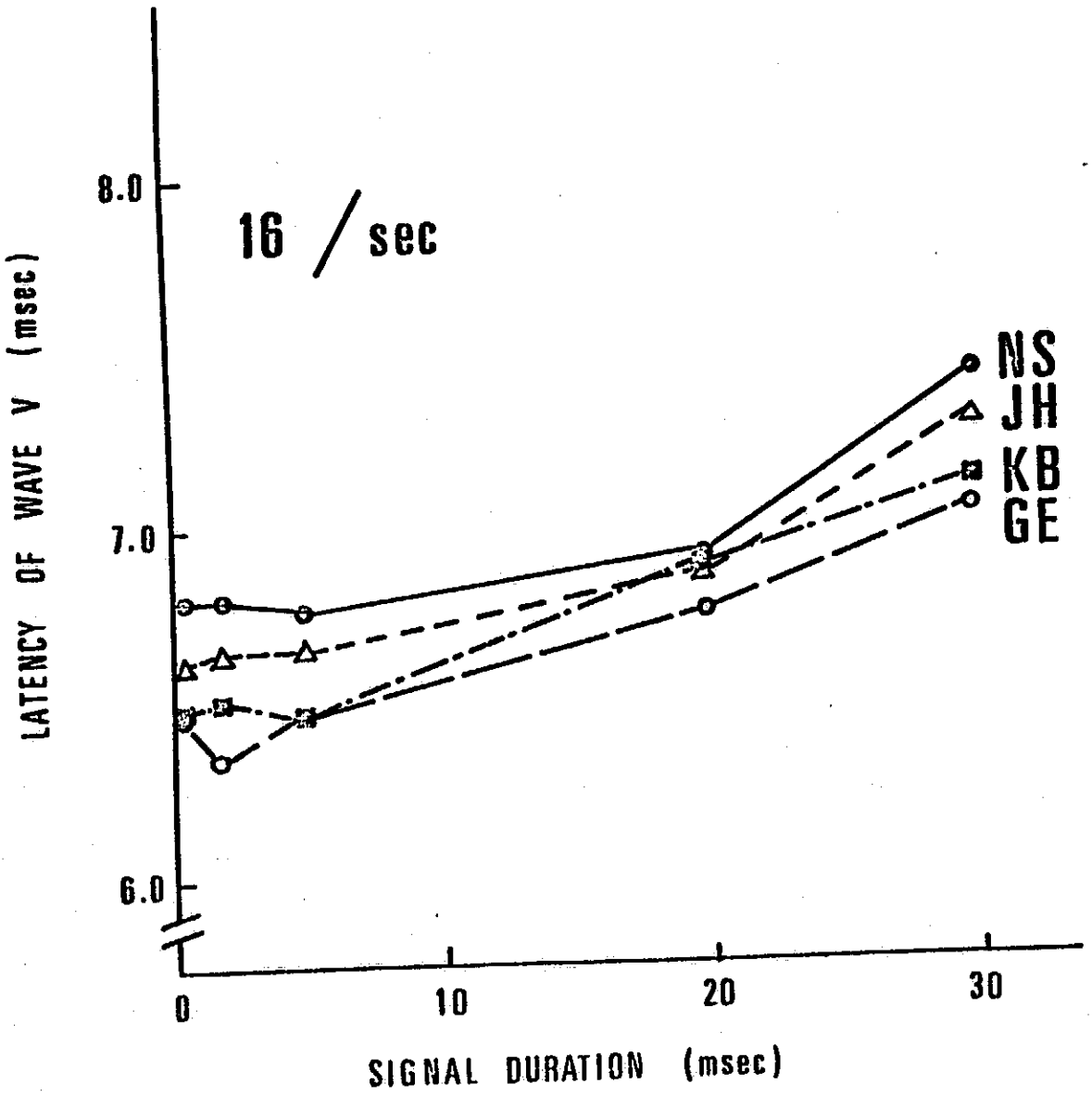
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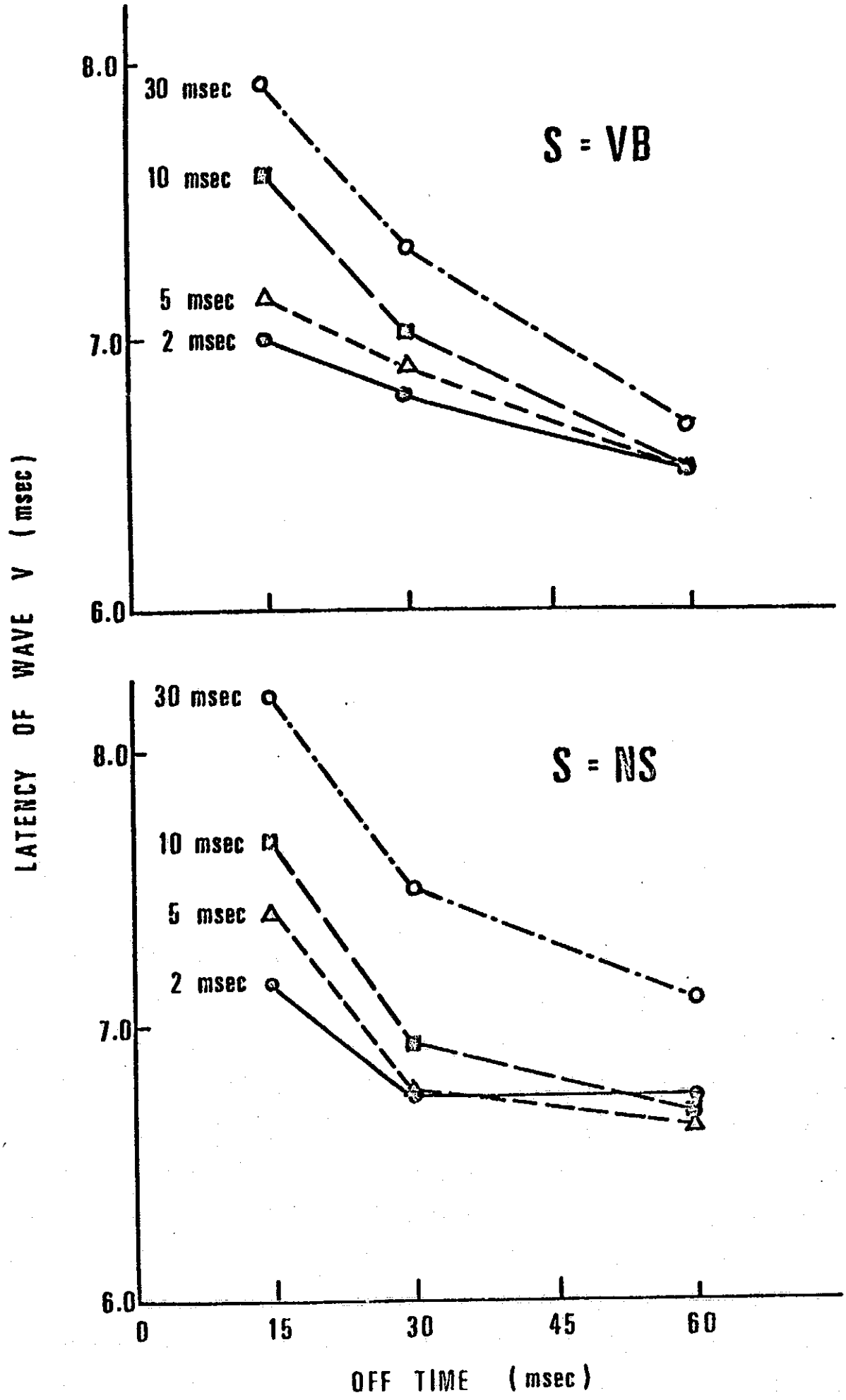
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LATENCY OF WAVE V (msec)
OFF TIME (msec)