

CLICK-INTENSITY DISCRIMINATION IN RELATION
TO THE STATISTICS OF THE N_1 RESPONSE

by

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TABLE OF CONTENTS

	<u>Page</u>
ACKNOWLEDGMENTS	iii
LIST OF FIGURES	V
LIST OF TABLES	VIII
Chapter	
I. INTRODUCTION	1
II. THE COMPOUND ACTION POTENTIAL OF THE AUDITORY NERVE	23
III. METHOD	35
IV. RESULTS	50
V. DISCUSSION	72
VI. SUMMARY	121
APPENDIX A	123
APPENDIX B	131
APPENDIX C	133
REFERENCES	136
AUTOBIOGRAPHICAL STATEMENT	140

LIST OF FIGURES

FIGURE		Page
1	Schematic oscillogram of the potentials recorded at the round window of the cochlea in response to a click stimulus	26
2	Representative amplitude-intensity function .	31
3	Block diagram of the apparatus employed in Experiment 1	37
4	Block diagram of the apparatus employed in Experiment 2	45
5	Human Weber functions for click stimuli. Intensity discrimination data for two listeners of Experiment 1 are compared with averaged Weber ratios reported by Avakyan and Radianova (1963).	52
6	The effect of masking noise on click intensity discrimination. Subject A.I.	56
7	The effect of masking noise on click intensity discrimination. Subject H.T.	58
8	Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. Subject 1-25.	61
9	Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. (A): Subject 2-27. . .	63
10	Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. (A): Subject 3-22. (B): Subject 3-20.	65

<u>Figure</u>	<u>Page</u>	
11	Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. (A): Subject 2-14. (B): Subject 3-14.	67
12	Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. (A): Subject 1-31. (B): Subject 3-29.	69
13	Model of click-intensity discrimination based on fluctuations of N_1 amplitude. . .	74
14	Peak-to-peak amplitude of the N_1 response as a function of click intensity. (A): Subject 1-25. (B): Subject 1-31. . .	78
15	Peak-to-peak amplitude of the N_1 response as a function of click intensity. (A): Subject 4-26. (B): Subject 3-22. . .	80
16	Histogram of the peak-to-peak amplitude of the N_1 response as a function of click intensity. Subject 1-31.	82
17	Histogram of the peak-to-peak amplitude of the N_1 response as a function of click intensity. Subject 3-22.	84
18	Standard deviation of N_1 amplitude as a function of click intensity. (A): Subject 4-08. (B): Subject 4-26.	87
19	Standard deviation of N_1 amplitude as a function of click intensity. (A): Subject 1-31. (B): Subject 3-22.	89
20	Intensity function for clicks presented with and without noise background. The peak-to-peak amplitude of the N_1 response is plotted as a function of click intensity. Subject 3-22.	96

<u>Figure</u>	<u>Page</u>	
21	Intensity function for clicks presented with and without noise background. The peak-to-peak amplitude of the N_1 response is plotted as a function of click intensity. Subject 4-26.	98
22	Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. Subject 1-31.	101
23	Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. Subject 3-22.	103
24	Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. Subject 3-14.	105
25	Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. Subject 3-06.	107
26	Graphical computation of the percentage of correct response in the two-alternative forced choice experiment for assymmetric distributions (chi-square, d.f. = 3) of the decision variable. The area under the ROC curve equals percentage of correct responses.	110
27	Standard deviation of N_1 amplitude as a function of click intensity with and without a noise background. (A): Subject 1-31. (B): Subject 3-22.	112
28	Standard deviation of N_1 amplitude as a function of click intensity with and without a noise background. (A): Subject 4-08. (B): Subject 4-26.	114
29	Schematic diagram of the peak-to-peak reader.	125
30	(A) The N_1 response indicating the peak-to-peak amplitude. (B) The peak-to-peak readout of the N_1 response.	127

LIST OF TABLES

<u>Table</u>		<u>Page</u>
1	Comparison of obtained physiological Weber fractions with those predicted from the neural model of intensity discrimination.	92
2	Prediction of the Weber fraction in noise from its unmasked value.	116
3	Variability of N_1 amplitude for clicks masked by random noise and by Pseudo-Random noise.	132
4	Difference limens for click intensity with and without a continuous noise background. Subject A.I.	134
5	Difference limens for click intensity with and without a continuous noise background. Subject H.T.	135

Chapter I

INTRODUCTION

A long-standing theoretical controversy in psychophysics concerns the source of the variability that sets the limit on an observer's sensitivity. During the past twenty-five years, two classes of models have been developed to deal with this problem. One kind of model is based on energy fluctuations inherent in stimuli; the other, on fluctuations in neural effect produced by a stimulus. In general, both sources of variability are considered in each kind of model; depending on the specific theory, however, major emphasis is placed on stimulus-oriented or neurally derived fluctuations. A review of the literature relevant to this controversy will be presented in this chapter.

Stimulus-Oriented Theories of Detection and Discrimination

The modern era in stimulus-oriented theories was introduced by Hecht, Schlaer, and Pirenne (1942) with their "physical quantum theory." The theory suggests that the variability in an observer's response to light near the absolute threshold is attributable to variations inherent in the physical stimulus. "Biological variations" are considered to be so small a part of the total variation that they are of no great importance in

determining sensitivity.

When the intensity of the light stimulus has been attenuated by physical filters and by the ocular media, the number of quanta of light per flash that reach the retina and are actually absorbed by the photoreceptors is subject to a great deal of variation, even though the gross parameters of the physical stimulus (wavelength, intensity, duration) remain constant. If, for example, it is required that eight quanta be absorbed for a visual effect to occur, and a particular stimulus provides, on the average, eight quanta that are absorbed, trials will occur in which eight or more quanta are absorbed by the retina and trials will occur in which fewer than eight quanta are absorbed. In the former case a visual effect will occur; in the later case, the effect will not occur.

The variation in the number of quanta absorbed can be predicted from probability theory. Absorption of a quantum by the retina is a discrete, independent, random event of low probability. It follows, therefore, that the number of quanta absorbed from a flash of a "fixed" energy varies according to the Poisson probability distribution. It is this distribution that describes the relationship between the "retinal threshold," the intensity of the stimulus, and the probability that the number of quanta delivered by the stimulus and absorbed by retinal elements will be equal to or greater than the "retinal threshold."

Tables of the Poisson distribution provide the probability that x or more quanta will be absorbed given that the mean number

of quanta delivered in the stimulus flash is known. The "retinal threshold" for an observer, which is the critical number of quanta necessary for a visual effect, can be empirically determined by generating a "frequency of seeing" curve and fitting this curve to one of the family of Poisson sums. Once the theoretical distribution corresponding to the "retinal threshold" has been established, the probability of a stimulus producing a visual effect can be determined on the basis of one item of information, the average number of quanta delivered by the stimulus.

Quantum fluctuations have also been invoked by Mueller (1956) to account for discrimination between two luminances. Although the basic mechanism of fluctuation is the same as that described by Hecht, Schlaer, and Pirenne, Mueller finds it necessary, when dealing with differential sensitivity, to consider the variabilities of two stimuli; the standard (I_1) and the incremented ($I_2 = \bar{I}_1 + \Delta I$).

The subject's problem is conceived to be one of sampling from two sources or populations of quanta rather than from one source. For discrimination to be correct it is required that the number of quanta absorbed from the incremented stimulus (I_2) exceeds the number absorbed from the standard (I_1) by some fixed number, c , i.e., if I_1 yields n quanta, then I_2 must yield at least $n + c$. That c equals a fixed number of quanta, sets a "retinal difference threshold." The probability that $I_2 - I_1 \geq c$ can be obtained from a distribution of differences whose parameters can be derived from the probability distributions (Poisson) describing the fluctuations of I_1 and I_2 .

One consequence of Mueller's formulation is that for a constant probability of detection, ΔI is a function of the square root of I_1 --the "square root law," rather than Weber's Law. This follows from the relationship between the mean and variance of the Poisson probability distributions--mean equals variance--that describes the quantum fluctuations for both I_1 and I_2 .

Both absolute and differential sensitivity were considered by Barlow (1956, 1957) in an analysis that evaluated the effects of both quantum fluctuations and spontaneous neural activity.

Barlow considered that at absolute threshold, sensitivity is limited by on-going, spontaneous activity at the retina, i.e., neural firing in the absence of a stimulus. The problem for the observer in detecting stimuli near threshold is to discriminate between the small number of quantum-initiated excitations and the small number of spontaneous retinal firings. This task can be considered one in which the observer samples from two distributions of events, one describing spontaneous activity, the other stimulus-initiated activity. The decision rule is: choose as the stimulus-initiated event the larger of the two samples. As the intensity of the stimulus is increased from zero, the probability that the greater excitation arose from the distribution describing stimulus-produced excitations increases. In this theoretical framework, the absolute limen is considered to be a form of differential discrimination between spontaneous and stimulus-initiated firings.

In the case of the difference limen, Barlow concludes, as did Mueller, that sensitivity is limited by statistical fluctuations (over trials) in the number of quanta absorbed from both the standard (I_1) and incremented (I_2) stimuli. The contribution of "retinal noise" to the total variability is disregarded, because these fluctuations are considered negligible when compared to the fluctuations in quantum absorptions at supra-threshold levels.

As a result, differential discrimination turns out to be based on a mechanism exactly like the one proposed by Mueller, with the exception that the "retinal threshold" (c) equals one quantum. Specifically, correct discriminations occur whenever the number of absorptions produced by I_2 exceeds the number of absorptions produced by I_1 .

While the absolute threshold for a flash may be analyzed in terms of the Poisson fluctuations describing the absorption of a small number of photons by the retina, a similar analysis cannot be made in hearing. The absolute threshold for a burst of tone or of noise involves energies (of the order of 10^{-10} ergs) which are many orders of magnitude greater than the energy of a single "phonon" (approximately 10^{-22} ergs at 10 kHz). With threshold sounds containing as many as 10^{12} phonons, Poisson fluctuations are obviously negligible.

As a result, auditory "ideal observers" are described for the case of signals masked by audible noise. Performance of these ideal observers is then given by the energy distributions for signal-plus-noise (SN) and for noise alone (N). Green

(1960) has described such an optimal detector for the case of noise signals masked by a noise background. The decision rule for optimal forced-choice detection requires that the observer measure the energy in the two stimulus waveforms and select the greater of the two energies as the one representing the SN stimulus.

If, in fact, the decision-making apparatus really measures some quantity that is monotonic with energy, then the decision outcome will still be the same. It is frequently assumed that the human auditory mechanism does perform some transformation of the acoustic stimulus that is monotonic with energy.

The probability that the observer votes correctly utilizing the optimal decision rule is the probability that a random observation from the SN energy distribution is greater than a random observation from the N distribution. This probability is easily computed from the appropriate difference distribution, whose parameters are derived from the energy distributions for signal-plus-noise and noise alone. Green has shown that these probability distributions are chi-square in character with the number of degrees of freedom equal to twice the product of signal bandwidth and duration.

Neurally Oriented Theories of Detection and Discrimination

We turn now to a class of theories which propose that the limit on the observer's sensitivity is based not on the physical parameters of the stimulus but on the neural events evoked by

the stimulus.

Crozier (1940), who disagreed vehemently with Hecht, presented a theory which proposes that intensity discrimination is a function of the neural events evoked by the stimuli involved. The theory begins with the assumption that the population of "elements of neural effect" is normally distributed with respect to log threshold. As a result, the relationship between stimulus intensity (I) and neural effect (E) is a log-normal ogive.

The important concept in Crozier's theory of intensity discrimination is that of availability of elements of neural effect. A stimulus which excites the entire population of elements has a "maximal" neural effect ($E_{\max.}$). Stimulus I_1 whose intensity is less than maximal excites E_1 of the population of elements. A proportion of the population remains available for stimulation. Availability equals $E_{\max} - E_1$. For a stimulus $I_2 (=I_1 + \Delta I)$ to be perceived as just noticeably different from I_1 that stimulus must be able to excite a number of the available elements. This ability to excite will depend upon the size of the available pool, being less when availability is less. Crozier proposes, therefore, that the JND is inversely proportional to availability. As the intensity of I_1 is increased, and E_1 increases, availability becomes proportionately smaller. Since the JND is given by the reciprocal of availability, as I_1 is increased, ΔI will increase, and the form of the

Weber function can be specified.

It should be noted that Crozier's availability theory neglects the variations in the neural effects evoked by I_1 and by I_2 .

This very variability of neural effect is central to Blackwell's (1963) model of visual detection and discrimination. Blackwell assumes that both the background stimulus (I_0) and the incremented stimulus ($I_0 + \Delta I$) produce neural effects (E_0 and $E_0 + \Delta E$) whose magnitudes vary from trial to trial. These variations in neural effect are attributed to quantum fluctuations in the light stimulus together with all sources of biological variability. The distributions describing the variability of E_0 and $E_0 + \Delta E$ are assumed to be normal and homogeneous with respect to variance.

The decision process consists of the establishment of a neural criterion, E_c , which is employed by the observer in making judgments about the presence or absence of the stimulus increment. The establishment of E_c is supposed to be an empirical process in which the observer samples values of E_0 produced by I_0 and estimates the distribution of E_0 . As a result of instructions to the observer, the criterion, E_c , is set so that the probability that I_0 will produce a value of E_0 greater than E_c is negligible. Therefore, the observer will not give "sensory false alarms", i.e., since the probability that E_0 will exceed E_c is negligible it can be assumed that the observer will never report the presence of $I_0 + \Delta I$ when, in fact, only I_0 was presented. Once the observer sets the value of E_c , that criterion is fixed for

the remainder of the experiment; E_c is applied to all presentations of $I_0 + \Delta I$. Correct detection of a given ΔI occurs only when $E_0 + \Delta E$ exceeds E_c ; the proportion of correct detections is given by the area under the $E_0 + \Delta E$ distribution to the right of E_c .

The Signal Detection Theory of Swets, Tanner and Birdsall (1961) contains many elements of Blackwell's threshold theory. Unlike Blackwell's theory, however, the observer can "move" his criterion in either direction along the neural decision axis. The location of the criterion depends upon the observer's knowledge of the a priori probability of occurrence of the signals and upon "motivational" factors which can be altered by manipulation of the "pay-off matrix." The principal consequence of this conception is that false alarms are considered to occur for valid sensory reasons. The difference between Blackwell and Swets, Tanner, and Birdsall in their treatments of false alarms need not be considered here.

Hybrid Theories of Detection and Discrimination

The model proposed by Swets, Tanner and Birdsall describes discrimination behavior in terms of distributions of neural effect evoked by noise and by signals in noise. The effects of such parameters as the location of the subject's criterion, the magnitudes of the two variances and the distance (along the decision axis) between the two means are summarized in receiver operating characteristic curves. Discrimination data, e.g.,

psychometric functions, difference thresholds, Weber functions, intensity-duration relationships, are not predicted by the theory since the neural decision axis is not related to stimulus parameters.

One way to deal with this matter is to replace the hypothetical neural decision axis by one based on stimulus energy. Energy distributions are then substituted for the distributions of neural effect. These "ideal detectors" have been discussed in the first section of this chapter. Their inability to account for the Weber functions obtained with "statistic-less" stimuli--tone bursts, high intensity flashes, etc.--is all too well known.

Another way of handling the matter is to introduce explicitly a relation between the neural effects determining behaviors and the stimuli that evoke them. Both stimulus fluctuations--where they occur--and neural "noise" interact to determine discrimination behavior. Two such hybrid theories have recently been proposed by Treisman (1964, 1966) and McGill (1965, 1967). (We may note that at one point in the development of his "neural theory," Blackwell (1963, page 145) proposed that a hyperbolic function relates stimulus intensity to neural effect.)

Treisman, who deals with visual discrimination, begins by considering that each effective quantum absorption evokes neural firings in a sensory "channel." The mean number of absorbed quanta and, therefore, the mean number of activated channels is proportional to flash intensity, and the distribution (over flashes of "constant" intensity) is, of course, Poisson in nature.

The distributions of firings in all channels that are

activated are assumed to be identical and Gaussian and to have a fixed mean-to-sigma ratio. Furthermore, the excitabilities of all these parallel channels are perfectly intercorrelated ($r = + 1.00$), so that all channels fired on a given trial respond with the same number of impulses.

The number of impulses evoked by each presentation of a "constant" intensity flash is the product of the number of channels excited (Poisson-distributed) by the number of impulses per channel (Gaussian). The mean and variance of this distribution of neural effect can be computed; the distribution itself was generated by simulation on a computer (Treisman, 1966).

The foregoing description of Treisman's model is somewhat simplified. Actually, the model contains an expression for "spontaneous firings" ("dark light") to deal with absolute thresholds and expressions for adaptation to deal with the effects of constant backgrounds.

With discrimination between two flashes based on the number of impulses each evokes, the model predicts the transition in the Weber function from the square-root law at low levels to Weber's law at high intensities.

Closely related to Treisman's model is the formulation presented by McGill, (1967). Again, discrimination between two stimuli is based on the number of impulses each evokes in a sensory pathway. Since impulse flow in a multi-channel pathway is a kind of "Poisson noise" the number of impulses delivered to a "central counter" in a critical period of time varies in Poisson fashion. The mean (and variance) of this distribution

of counts is related by McGill to the energy on each trial of the stimulus that was presented.

For "statistic-less" stimuli, only the neural fluctuations (Poisson) are involved in limiting discrimination performance. When the stimulus fluctuates in energy from trial to trial, the Poisson mean also fluctuates. Stimulus noise and neural noise then interact to limit discrimination.

The relationship between stimulus energy and the Poisson mean needs to be mentioned. Originally, McGill (1965) proposed that the mean is directly proportional to stimulus energy. This assumption has two unhappy consequences. First, it predicts a square-root law for pure tone intensity discrimination, since tone bursts do not vary in energy from trial to trial. Second, the dynamic range of the sensory nervous system must equal the intensity range of the stimuli to be processed. The mean number of nerve impulses evoked by a sound 120 dB above threshold would have to be 10^{12} times the number of impulses evoked by the threshold sound.

These difficulties are eliminated if the mapping of stimulus energy on to neural counts is via a power law. Specifically, if the mean number of counts is made proportional to the n^{th} root of energy, not only is the dynamic range of neural counts compressed, but also the Weber function (for statistic-less stimulus bursts) moves from the square-root law towards Weber's law (McGill and Goldberg, 1968).

The hybrid models of Treisman and McGill employ different statistical mechanisms for generating their distributions of neural counts. In Treisman's theory the number of nerve impulses

is the product of two random variables, viz, the number of excited channels and the number of impulses per channel. In McGill's model, the number of impulses is sampled from a Poisson distribution whose mean is "driven" by the energy statistics of the stimulus.

Experimental Studies of Neural Coding of Stimulus Intensity

Experimental studies of neural mechanisms mediating intensity discrimination are of two main types. Ablation studies--in the Lashley tradition--have sought to determine which brain centers are essential for discrimination (by trained animals) of stimulus intensities. Electrophysiological experiments, on the other hand, have been performed in attempts to specify coding relations between stimulus intensity and either single cell firings or whole nerve action potentials. Input-output functions and variability of neural responses (imperfections in coding) will be considered in the following sections in connection with several studies of the visual, auditory and somatosensory systems.

(1) Single Cell Responses

A corollary of the classical all-or-none law is the frequency principle, which states that stimulus intensities are represented in the nervous system as frequencies of discharge (see Adrian, 1928). Microelectrode studies of nearly all sensory systems have described relations between nerve impulse frequency and stimulus intensity. Frequently, such studies have

generated negatively accelerated intensity functions to which logarithmic equations have been fitted.

A modern era in the study of sensory coding began in 1957 when Fitzhugh took pains to analyze firing patterns of single visual cells with respect to both average and variability. Fitzhugh used both an on-line and a statistical procedure in his analysis of ganglion cell responses.

In the on-line procedure, increment flashes were presented against various levels of background illumination, and responses were recorded from ganglion cells in the cat's retina. The cell discharge was displayed on an oscilloscope and was also presented through a loudspeaker. With the increment flash set at some given intensity, observers would look at and listen to the evoked discharge in order to determine whether each flash was followed by a detectable response in the form of a brief increase in the frequency of discharge. The increment threshold was set by the observer at the weakest intensity at which he could detect a response on roughly half the flashes. Fluctuations in firing patterns from flash to flash required that this determination be based on examination of at least five consecutive responses.

These fluctuations were also studied directly by repeatedly recording the number of nerve impulses evoked in a ganglion cell during the period (30 msec in duration) between 70 msec and 100 msec after each stimulus. Histograms of this number of firings were plotted and compared with histograms obtained from control (no stimulus) trials. Differential intensity sensitivity was computed and found to correspond to the "limens" determined by the on-line procedure.

Jacobs (1965) has investigated "intensity discrimination" in broad band excitatory and inhibitory cells in the lateral geniculate nucleus of the squirrel monkey. There is evidence that both classes of cells carry brightness information. The firing rate of excitatory cells is increased or decreased as stimulus intensity is increased or decreased. Inhibitory cells respond in the opposite manner.

Weber fractions for both classes of cells were generated by assuming that the just discriminable difference in neural activity, ΔR , was equal to a 3 spike/sec increase or decrease in the firing rate of each class of cell in the appropriate direction. The response rate evoked by an adapting stimulus (S_1) was recorded. The value of the "difference limen" at S_1 was then determined from the function relating the change in R to the magnitude of ΔS .

Weber functions obtained in this manner were found to have the same general form as the psychophysical functions for brightness discrimination obtained from squirrel monkeys in behavioral experiments.

Werner and Mountcastle (1965) recorded impulses from single afferent fibers in the saphenous nerve in response to mechanical indentation of the skin. The criterion for a "just discriminable difference" in neural activity (ΔR) was arbitrarily chosen as 6 impulses per response. It was assumed that this critical increment in neural response is constant at all levels of stimulation.

Using an on-line computer, the number of discharges, R_1 , to a base stimulus (S_1) and the number of discharges R_2 , to an incremented stimulus, $S_2 (=S_1 + \Delta S)$ were stored in the computer memory. This was done for blocks of approximately 30 trials at a given level of ΔS . The computer would decide that the difference in neural activity was "just discriminable" when S_2 produced a response larger than the response to S_1 by the critical value $\Delta R = 6$. If otherwise, the computer directed either an increase or decrease in ΔS , and the experiment continued until the value of ΔS was found that produced the critical ΔR of 6 impulses per response. Weber functions thus computed for these first order touch fibers of cats and monkeys closely resembled those determined for tactile sensation in man.

More recently, Mountcastle (1967) has essayed a formal justification for relating behaviorally determined Weber functions to functions obtained from first-order sensory cells. He proposes that, while the coding of stimulus intensity into impulses in first order cells may be linear or non-linear, succeeding transformations performed by higher-order cells are all linear. As a result the two Weber functions (one, neural; the other, behavioral) are not superimposed, but must be identical in form.

It has recently been demonstrated (see Kiang, 1965) that evoked activity in single units of the auditory nerve varies statistically from presentation to presentation of an identical

acoustic waveform.

Siebert (1965) has postulated that the stochastic nature of this coding of acoustic information in the nerve acts as a type of noise, limiting auditory detection and discrimination behavior. In investigating the limits imposed by this sensory noise, Siebert found it necessary to determine the complete statistical description of the firing pattern in every fiber of the nerve. Since it is impossible to record individually from the thousands of units that comprise the auditory nerve, the nerve was "reconstructed" by putting together single unit records from many animals. This was done in the case of the cat using data obtained with high frequency tone bursts (see Kiang, 1965).

Given such a description of the "total" spike activity in response to a stimulus in the high-frequency fibers of the nerve, and employing the methods of statistical decision theory, it becomes possible to describe the performance of an "ideal computer." For intensity discrimination, the input to the computer would be the complete record of the detailed activity of the "whole" nerve in response to two stimuli of different intensity. The ideal decision mechanism would then determine which set of neural records corresponds to the stimulus of greater intensity. The decisions of the computer would be optimal in that no device--given the identical input--could discriminate a smaller difference in intensity. The performance of the ideal computer constitutes a bound on the sensitivity of the human observer.

In the optimum decision procedure the number of impulses in the whole nerve evoked by the standard stimulus (I_1) is compared with the number of firings evoked by the incremented stimulus (I_2). That stimulus which delivers the greater number of spikes to the decision making apparatus is called "incremented."

Fluctuations in frequency of neural activity in the auditory nerve evoked by successive presentations of a high frequency tone burst are described by the Poisson distribution. Given an estimate of the mean frequency of discharge evoked by a stimulus, the variance in frequency is then established.

In two-alternative forced choice procedures, the probability that the ideal computer will vote correctly, i.e., designate as "incremented" that stimulus which evokes the greater total discharge, can be computed from a difference distribution whose parameters are derived from the distributions describing the fluctuations in neural activity evoked by I_1 and I_2 . "Physiological Weber functions" are derived and compared with human psychophysical data.

Siebert's approach, it should be noted, represents a significant improvement over the procedures of Fitzhugh, Jacobs, and Werner and Mountcastle. The sampling error inherent in the generation of discrimination data from the discharges in one cell of a sensory nerve is appreciably diminished in Siebert's use of a "reconstructed" nerve. A considerable error still remains, however; Kiang's comprehensive monograph,

which provided single cell records for Siebert's model, presents data on only 1500 units from 50 cats.

(2) Whole Nerve Action Potentials

One interesting way of coping with the sampling problem just discussed is to record from a whole nerve with a gross electrode. Synchronized discharges can be recorded in response to impulsive stimuli; the amplitude of the compound action potential is, presumably, a measure of the number of neurons activated.

Radianova (1963) investigated the relationship between the intensity of an acoustic click and the amplitude of the compound action potential of the auditory nerve (N_1). The potential was recorded in cats at the round window of the cochlea. (A detailed discussion of the nature of the N_1 potential is presented in the next chapter of this thesis.) Amplitude-intensity functions were plotted; amplitude was shown not to increase monotonically with click intensity. Specifically, in the region of intermediate intensities the function has a plateau. Radianova considered that the two-stage growth of the intensity function demonstrates that the entire set of nerve elements does not have a single distribution with respect to threshold. She concluded that the presence of a dichotomous population of neurons would lead to a deterioration in differential sensitivity in the region of intermediate intensities, where only a small increase in neural response

amplitude results from a relatively large increment in stimulus intensity.

The following sample calculation of a difference limen based on N_1 responses is taken from Radianova's report and serves to illustrate her assumptions and procedures. Measurement of N_1 responses to 36 successive clicks (intensity = 25 dB above threshold) gave the following result: Mean amplitude = 134 μ V; S.D. = 10 μ V. A t-test for uncorrelated means ($df = 35 + 35$) was used to compute the mean N_1 amplitude that would exceed 134 μ V with a probability equal to 0.5. (The standard deviation of the "sample" of 36 responses to the incremented click was taken to be 10 μ V.) This mean was found to be 135.58 μ V. The click intensity corresponding to this N_1 amplitude was then read from the smoothed amplitude-intensity function and found to be 25.2 dB. The relative difference limen equalled 0.2 dB.

"Physiological difference limens" were determined in this fashion in steps of 10 dB. Plotted against the corresponding values of I , they produce a W-shaped Weber function. As predicted, differential sensitivity was found to deteriorate in the region of stimulus intensities corresponding to the plateau on the intensity function. In a companion study, Avakyan and Radianova (1963) measured differential sensitivity for click stimuli with human subjects. Weber functions for humans were found to be similar in form to the physiologically derived data. The human functions are displaced vertically--discrimination is poorer--but the same characteristic hump appears

in the mid-region of intensity.

This similarity between the two Weber functions provides a kind of confirmation--in the case of whole nerve responses--of the "linearity hypothesis" proposed by Mountcastle (see page 16) for the case of single cell responses.

Purpose of the Present Experiments

The present research was undertaken in the conviction that the relations between difference limens for impulsive stimuli and whole nerve responses deserve more consideration than they have thus far received. Indeed, Radianova's two papers are the only ones that have attempted to relate differential sensitivity to the statistics of whole nerve responses. Although her experiments are pioneering, they are somewhat flawed and merit critical analysis and replication.

Radianova's use of the t-test to compute the increment in neural effect at the 0.5 level of significance is questionable. The distributions of N_1 amplitude are clearly not normal (see Radianova, page 352). Although she presents no evidence that the two distributions of neural effect have identical variances, the assumption of homogeneity of variance is somewhat more acceptable.

Translation of increments in N_1 amplitude to increments in click intensity were made by reference to the amplitude-intensity function. Data points for this function had been obtained at intervals of 10 dB; the smoothed function was then fitted to

these points. Radianova's interpolation over ranges as small as 0.2 dB employing values that are separated by 10 dB is a procedure that is fraught with danger since it provides only crude estimates of the slope of the intensity function in the region of interest.

Intensity discrimination with click stimuli is admittedly a difficult psychophysical task (see Avakyan and Radianova). The adjustment method employed by these authors produced extremely variable results. More sophisticated psychophysical procedures, such as the staircase method, can reduce variability by shaping the subject's responses.

In the present experiments, an attempt was made to relate both the difference limen for clicks and the Weber function to the statistical fluctuations of N_1 responses measured at the round window of the guinea pig. In the physiological experiments, an on-line procedure was employed which provided for non-parametric determinations of the "physiological difference limens." In addition, the procedure made it possible to avoid completely the kind of crude interpolation on the amplitude-intensity function that was employed by Radianova. Staircase procedures were used in both animal and human experiments.

Finally, the effects of masking noise on differential sensitivity were explored in both psychophysical and electrophysiological experiments.

Chapter II, which follows, presents a brief discussion of the N_1 potential. The origins and characteristics of this response are analyzed before the present experiments are discussed.

CHAPTER II

THE COMPOUND ACTION POTENTIAL OF THE AUDITORY NERVE

Origin of the N_1 Potential

Information about the intensity of a stimulus is undoubtedly represented in the auditory nerve by the frequency of impulses in the nerve. Two mechanisms may contribute to the total number of impulses per unit time: increases--with intensity--in the frequency of discharge in given neurons, and increases in the number of active neurons. Katsuki, Sumi, Uchiyama, and Watanabe (1958) have shown that the range over which the frequency of discharge in a given eighth nerve fiber varies as a function of intensity is limited to 20-25 decibels. It would appear, therefore, that the neural encoding of intensity information expressed as the frequency of discharge in the auditory nerve is more directly represented by the number of units that are excited than by the frequency of discharge in individual elements.

In the case of a very brief acoustic stimulus, e.g., a click, the encoding of intensity information must be predominantly a function of the total number of neurons excited since the neurons of the auditory nerve can produce only a few spikes in response to a click--even at very high levels (Tasaki, 1954; Katsuki et. al., 1958; Kiang, 1965). At best,

individual neurons could relay only a very coarse measurement of the intensity of a click.

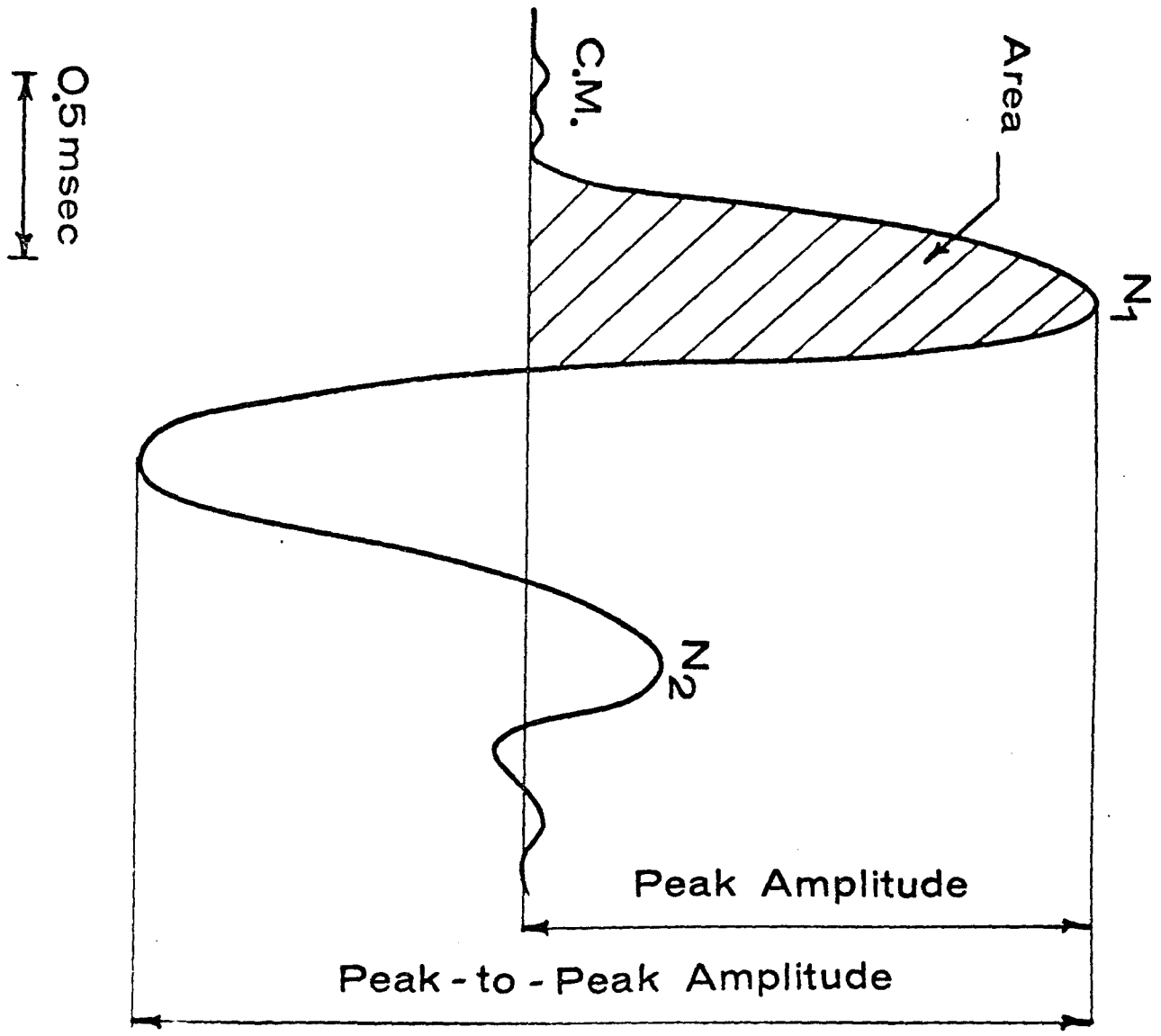
It has been considered by several investigators (Davis, 1935; Tasaki, Davis and Legouix, 1952; Frishkopf, 1956; Deatheridge, Eldridge and Davis, 1959; Peake, 1959; Radianova, 1963; Kiang, 1965) that an index of the number of elements that respond in synchrony to a click is the compound action potential of the eighth nerve. This potential has been designated N_1 . A convenient location from which to record this action potential is the round window of the cochlea. The signal recorded by a gross electrode at the round window is schematically represented in Figure 1. The response consists of several components: the microphonic response of the receptor elements which reproduces the mechanical input to the inner ear (Derbyshire and Davis, 1935; Davis, 1935); N_2 which is thought to be the result of repetitive firing of eighth nerve fibers (Tasaki, 1954) or the response of elements in the cochlear nucleus (Frishkopf, 1956); and N_1 , a large, diphasic response, which is first negative then positive (see Figure 1).

Frishkopf (1956), in the course of developing a probabilistic model of neuroelectric events in the eighth nerve, has employed the N_1 response as an index of click-intensity coding. Recording from the round window with a gross electrode, Frishkopf found it necessary to make three principal assumptions. First, that all of the fibers respond with the same amplitude; second,

Figure 1

Schematic oscillogram of the potentials recorded at the round window of the cochlea in response to a click stimulus. The trace begins with the electrical pulse delivered to the earphone. Upward deflection indicates negatively at the cochlear electrode.

C.M.: Cochlear Microphonic



that the gross electrode records equally well from all of the fibers in the nerve; and third, that elements excited by a click stimulus all fire synchronously. The last assumption is perhaps the most interesting, especially since data are available which bear on the question of synchronous firing.

Microelectrode studies of eighth nerve cells have shown that although a sufficiently intense click stimulus will evoke responses from units arising along the entire basilar membrane, only fibers arising in the basal turn contribute significantly to the N_1 response. Since the excitatory travelling wave arrives at the apical turns only after a relatively long delay, elements arising in the apex fail to contribute to the N_1 response (Tasaki, Davis and Legoux, 1952; Teas, Eldridge and Davis, 1962; Kiang, 1965). As a matter of fact, Kiang (1965) recorded simultaneously from the round window and from single eighth nerve elements and demonstrated that only units with a high characteristic frequency have spike latencies that fall with the N_1 envelope. These units have been found predominantly in the basal turn of the cochlea.

Furthermore, these basal turn cells, whose discharges constitute the N_1 potential, respond to a click stimulus with only one spike (Tasaki, 1954; Kiang, 1965). Apical turn elements, on the other hand, may produce up to three impulses in response to a high intensity click (Tasaki, 1954). We have noted, however, that even the earliest discharge from an apical cell falls outside the N_1 envelope. The amplitude

of the N_1 response is a function of the number of basal elements that respond to the click stimulus.

It has been indicated above that acoustic intensity coding is undoubtedly represented by the frequency of impulses in the auditory nerve. However, if we employ the N_1 response as a neural index of click intensity, the discharges of apical cells which do not contribute to the N_1 response are disregarded. In this study, we shall consider that all the elements that contribute to the N_1 response (1) have equal response amplitudes, (2) fire in a synchronous manner and (3) are detected equally well by the gross electrode. We will further suppose that the discharge that contributes to the N_1 response is monotonically related to the discharge of cells in the apical region of the cochlea. Specifically, an increase (with click intensity) of basal turn impulses is accompanied by an increase in apical cell impulses.

We note that data from single auditory nerve cells do not support assumptions (1), (2) and (3). There are no data bearing on the fourth assumption, which concerns the relation between basal and apical cell discharges. These simplifying assumptions are required, however, if the N_1 response is to be employed as an index of click intensity. Validity for the procedure is provided by the results presented in Chapters IV and V.

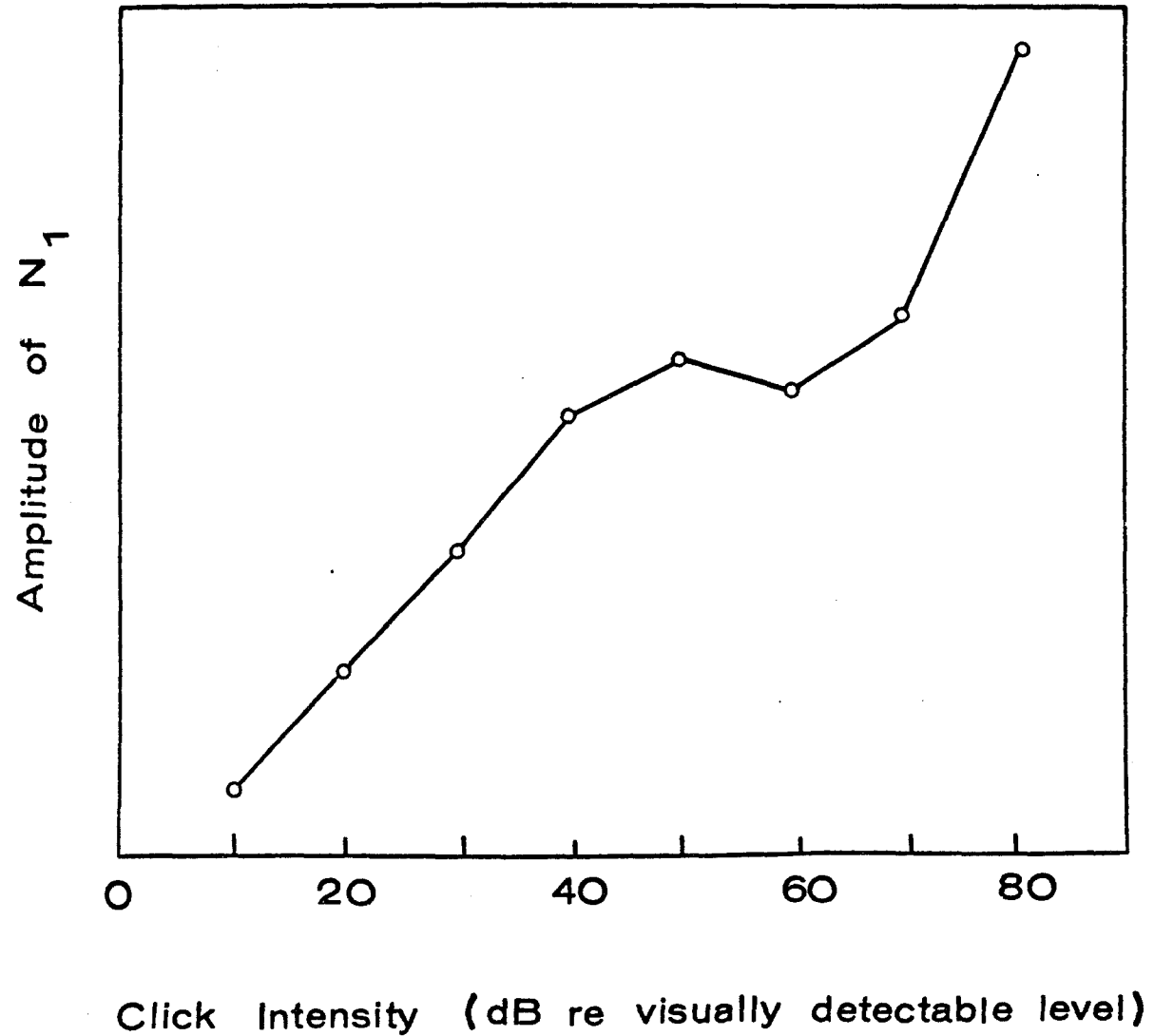
The growth of N_1 amplitude with increasing stimulus intensity was first described by Davis (1935) in his classical

study of eighth nerve potentials. More recently, Tasaki, Davis and Legoux, 1952; Frishkopf, 1956; Davis et. al., 1958; Peak, 1959; Radianova, 1963; and Dewson, 1967 have shown--for tone pips and clicks--that as the intensity of the stimulus is increased from "threshold", there is a concurrent increase in the amplitude of the N_1 response (see Figure 2). As indicated above, the growth in the size of the action potential implies an increase in the number of fibers which simultaneously respond to the stimulus. The increase in the size of the N_1 response is not, however, monotonic with increase in stimulus intensity. In the range of intermediate intensities (approximately 40-50 dB above "threshold") there is a plateau in the amplitude-intensity function with a range of about 10 dB. Further increase in stimulus intensity after the levels required to reach the plateau produces a subsequent growth in the size of N_1 (see Figure 2). The most commonly employed measure of the size of the N_1 response has been its peak amplitude (see Figure 1). Frishkopf (1956) has demonstrated, however, that both the peak-to-peak amplitude and the area of the response vary with stimulus intensity in a manner virtually identical to that of peak amplitude.

The two-stage growth of the amplitude-intensity function has suggested to both Frishkopf and Radianova that the set of responding nerve elements is distributed in two groups that differ with respect to their thresholds. There is a

Figure 2

Representative amplitude-intensity function. In this plot, a 0 dB click evokes an N_1 response that is just detectable on the oscilloscope.



group with lower threshold and a group with higher threshold. Katsuki, Suga and Kanno (1962) in an investigation of the response properties of single fibers in the auditory nerve, have presented evidence that the population of auditory neurons is, in fact, distributed in two groups differing with respect to their thresholds. Furthermore, anatomical investigations of the primary hair cells on the basilar membrane demonstrate that the inner and outer hair cells have different thresholds. Davis, et. al. (1958) destroyed external hair cells with streptomycin, while leaving the inner cells intact--producing an ear with an elevated threshold. The authors further demonstrated that the external cells are 30-40 dB more sensitive than the inner cells; the latter were found to be more rugged and less sensitive to mechanical deformation. Kiang (1965), on the other hand, considers that no criterion has been found which allows the primary units to be divided into two distinct types with respect to threshold.

Eighth Nerve Responses to Clicks in Noise

If the response to a click in a noise background is compared with the response in the absence of noise, one finds a reduction in the amplitude of the response evoked in the noise condition. This "masking" effect was first demonstrated by Davis (1935) and has since been confirmed by other investigators (e.g., Fishkopf, 1956; Dewson, 1967). The "masking" phenomenon is interpreted in terms of threshold shifts for

the units involved. In the presence of the noise background, neural units are continuously being excited by the noise stimulus. When the click is presented, some of the units that would normally have responded to the click are unavailable because they are in a more or less refractory state, having been excited by the noise stimulus. These units are, therefore, unable to respond to the click. To the extent that the amplitude of the N_1 response is an index of the number of basal turn neurons which do respond to the click, the N_1 potential evoked in the noise condition is diminished in amplitude. This "neural masking" phenomenon was termed the "line-busy" effect by Davis.

Dewson (1967) has considered and rejected the possibility that the neural masking effect is produced by activation (by the noise) of the olivo-cochlear bundle.

Variability of N_1 Amplitude

It is possible to produce an acoustic stimulus whose parameters are essentially constant from trial to trial. A click is an example of such a stimulus. Trial by trial presentations of identical clicks evoke N_1 responses of somewhat variable amplitude. Frishkopf (1956), who first studied the variability of the N_1 response, attributed the variability to momentary fluctuations in the thresholds of the neural units involved. A unit will fail to respond to a stimulus when that stimulus fails to exceed the threshold of the unit at the

instant of presentation. Since the threshold for a unit fluctuates over time, a situation arises in which a given unit may respond to a click on one trial and fail to respond on some other trial. Since the amplitude of N_1 is assumed to be an index of the total number of responding units, and since each of the units in the population may or may not respond to a fixed stimulus on a given trial, the amplitude of the N_1 potential is a function of the number of elements whose momentary thresholds were exceeded by the stimulus. Physiological evidence that auditory neurons do have fluctuating thresholds has been presented by Rosenblith (1964). Recording from single units in the cochlear nucleus, it was found that a stimulus of fixed intensity would excite a unit on one trial and fail to excite the same unit on another trial. Probability of response was found to vary with click intensity over a range of approximately 15 dB.

Chapter III

METHOD

Experiment 1

In Experiment 1, human subjects were employed in determining the magnitude of the difference limen and the form of the Weber function for acoustic clicks. Difference limens were obtained with and without a continuous noise background.

(1) Apparatus

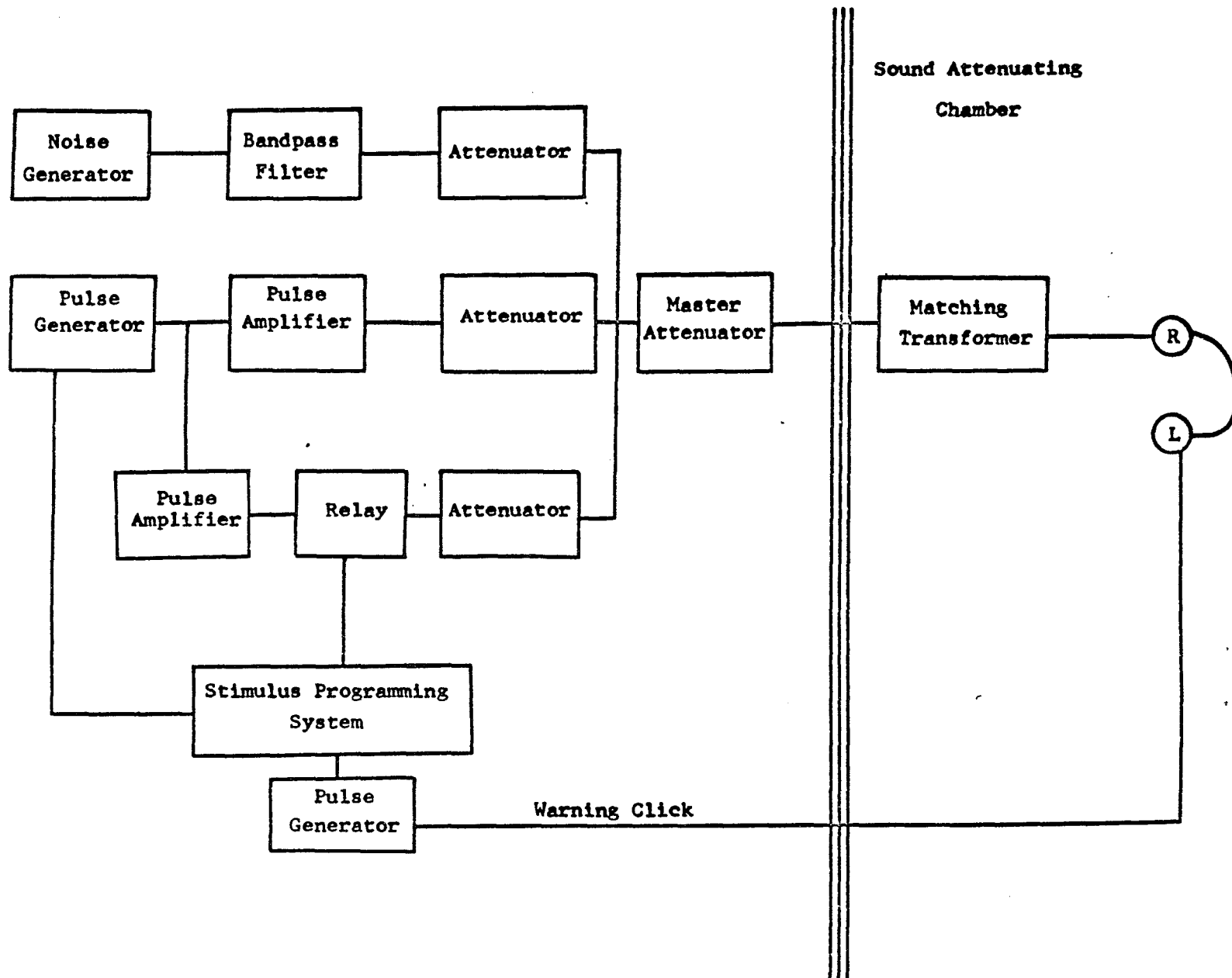
A block diagram of the apparatus is presented in Figure 3.

Stimuli were generated by a Tektronix 161 pulse generator, whose output (0.1 msec pulses) were fed to two channels--one for the standard (I), the other for the increment signal. Each channel contained pulse amplifiers and level setting attenuators. The signal channel included a relay whose closure inserted the increment in either one or the other of the two stimulus intervals comprising a trial.

The first pressure change in each click was a rarefaction. The peak amplitude of this rarefaction was recorded by a Western Electric 640 AA microphone in an ASA type 1 coupler. At the reference level of 0 dB employed in Experiment 1, a 0.75-volt pulse across the listener's earphone (Permoflux PDR-10) produced a peak output pressure of 124 dB with reference to 0.0002 dyne/cm^2 .

Figure 3

Block diagram of the apparatus employed in Experiment 1.



A General Radio (1390-B) noise generator, the output of which was band limited (100-6000 Hz) by a Krohn-Hite (310-CR) filter, provided the noise source. At the 0 dB reference level for noise, the voltage across the earphone was 0.011 volts as measured by a Ballantine (320) true RMS voltmeter. This provided an overall sound pressure level of 87 dB.

The outputs of the three stimulus channels were added in a resistive mixing network; their sum was passed through a master attenuator, a matching transformer, and finally into the subject's earphone, which was mounted in an MX/41-AR cushion.

An Iconix (6255-6010) timing unit was employed in programming the stimulus sequences required in the experiment.

(2) Procedure

Monaural (right-ear) thresholds were determined using a two-interval forced choice variation of the staircase method. Each trial began with a faint warning click to the subject's left earphone. The first of two stimulus clicks, which were spaced 0.8 seconds apart, occurred 1.5 seconds after the presentation of the warning click. The stimulus increment (ΔI) was randomly placed in either the first or second interval. The subject reported which of the two stimulus intervals contained the increment by pressing one or the other of two micro-switch buttons. Immediate knowledge of results was given to the subject by red and green indicator lights. Trials were

self-paced and were spaced approximately 6 seconds apart. Testing was begun with the incremented click ($I + \Delta I$) easily discriminable from the standard (I). If the subject voted correctly on at least five out of six trials at a given level of ΔI , the signal pulse was decreased by 3 dB for the next block of six trials. On the other hand, if the subject voted incorrectly on two of the six trials, in a block, the signal pulse was then increased by 3 dB. At the end of a run of trials, the median value of attenuation in the signal channel was computed and then converted to increment power (ΔI), taking into account the in-phase addition of the standard and signal pulses. This staircase procedure allows the subject to cross and re-cross the threshold ΔI several times in a single session. For each stimulus condition at least three thresholds were determined. Each determination required a minimum of 100 trials.

Experiment 2

In Experiment 2, a novel on-line voting procedure was employed in determining the magnitude of the "physiological difference limen" for clicks. This difference limen was based on the neural response (N_1) recorded at the cochlea of the guinea pig. Limens were obtained with and without a continuous noise background.

(1) Subjects

Eleven female Hartley guinea pigs, approximately 250 grams in weight were used as subjects.

(2) Surgical Procedure

Guinea pigs were anesthetized by intramuscular injection of Thorazine (16 mg/kg) followed, after a fifteen minute interval, by intraperitoneal injection of urethane in distilled water (800 mg/kg).¹ The initial dose brought about deep anesthesia in approximately 45 minutes. During the course of the recording procedure, which lasted about three hours, the level of anesthesia was tested by means of the corneal and withdrawal reflexes. A supplementary dose of urethane (one-third of the initial dose) was administered when the corneal and withdrawal reflexes could again be elicited. The object of using this combination of anesthetics was to produce a deep and constant anesthesia over the full course of the experiment. Following anesthetization the animal was placed on a surgical table and the external ear canals were gently cleaned. Surface hair was removed from the vicinity of the pinna by means of a commercial depilatory. After removal of surface hair from around the pinna, the area was locally anesthetized by injection of Xylocaine (0.5 ml of a 1% solution).

The initial surgical incision was made just posterior to the line of attachment of the pinna to the scalp. This incision exposed the masseter muscle and the posterior mandible.

¹Dr. Juergen Tonndorf suggested the use of this Thorazine-plus-urethane anesthetic. Pentobarbitol and Equithesin had been found to be unsatisfactory agents in that they produced respiratory seizures within forty minutes after administration.

After ligating the external jugular vein, both the masseter muscle and the mandible were cut across at the middle and the posterior half of the mandible was removed. Following the mandible resection, parts of the sterno-cleido-mastoid and diagastric muscles were removed. Next, all of the tissue around the bony styloid process was separated from the surface of the bone and the process was resected by means of a bone-cutting forceps. By suturing the posterior masseter muscle to its anterior half the posterior bulla was exposed. After removal of periosteum from the surface of the bulla a small hole was made by means of a #4 dental burr exposing the round window of the cochlea.

Immediately after the round window exposure was completed, acrylic dental cement was applied to the periphery of the opening in the bulla. This served two purposes: first, to retard seepage of fluids from surrounding tissues, and second, to provide an adhesive layer for further applications of cement. The surgical procedure was completed by the removal of the pinna.

A six-inch length of enamelled silver wire (100 μ in diameter) was employed as the active electrode. The tip of the electrode was heated over an alcohol burner and melted into a ball whose diameter was approximately 200 μ . The spherical tip of the electrode was placed at the periphery of the round window under the bony niche in order to minimize interference with the normal movement of the membrane. Placement of the electrode was facilitated by means of a micromanipulator. The shank of

the electrode was cemented to the periphery of the bulla opening with acrylic dental cement.

An alligator clip attached to expose muscle tissue at the neck served as the reference electrode.

The operations of opening the bulla and placing the electrode were performed under a binocular microscope of variable power. A light mounted on the microscope provided illumination as required.

(3) Stimulation

Following the placement of the active electrode, the animal was moved to a ventilated, sound attenuated, shielded chamber (Industrial Acoustics Corporation AC-3).

Stimuli were introduced by means of a Telephonic TDH-39 earphone coupled to a three-inch length of 0.75 inch diameter rubber tubing. Coupling to the ear canal was accomplished by placing the tubing over the circular sheath of skin produced by removal of the pinna.

A rectangular pulse, 0.1 msec in duration, was used to drive the earphone. The pulse was generated by a Tektronix 161 pulse generator. The output of the pulse generator was split into two channels each containing pulse amplifiers and attenuators for level setting. The signal channel included a relay whose closure inserted the stimulus increment in one of the two stimulus intervals comprising a trial. Our 0 dB click is produced by a 1.4 V pulse delivered to the earphone.

A block diagram of the apparatus employed in Experiment 2

is presented in Figure 4.

A Grason Stadler (455B) noise generator provided the noise source. The output of the noise generator was band limited (100-6000 Hz) by a Krohn-Hite (310-CR) filter. With 0 dB of attenuation in the noise channel, the noise voltage as measured by a Ballantine (320) true RMS voltmeter connected across the earphone terminals was 0.055 volts.

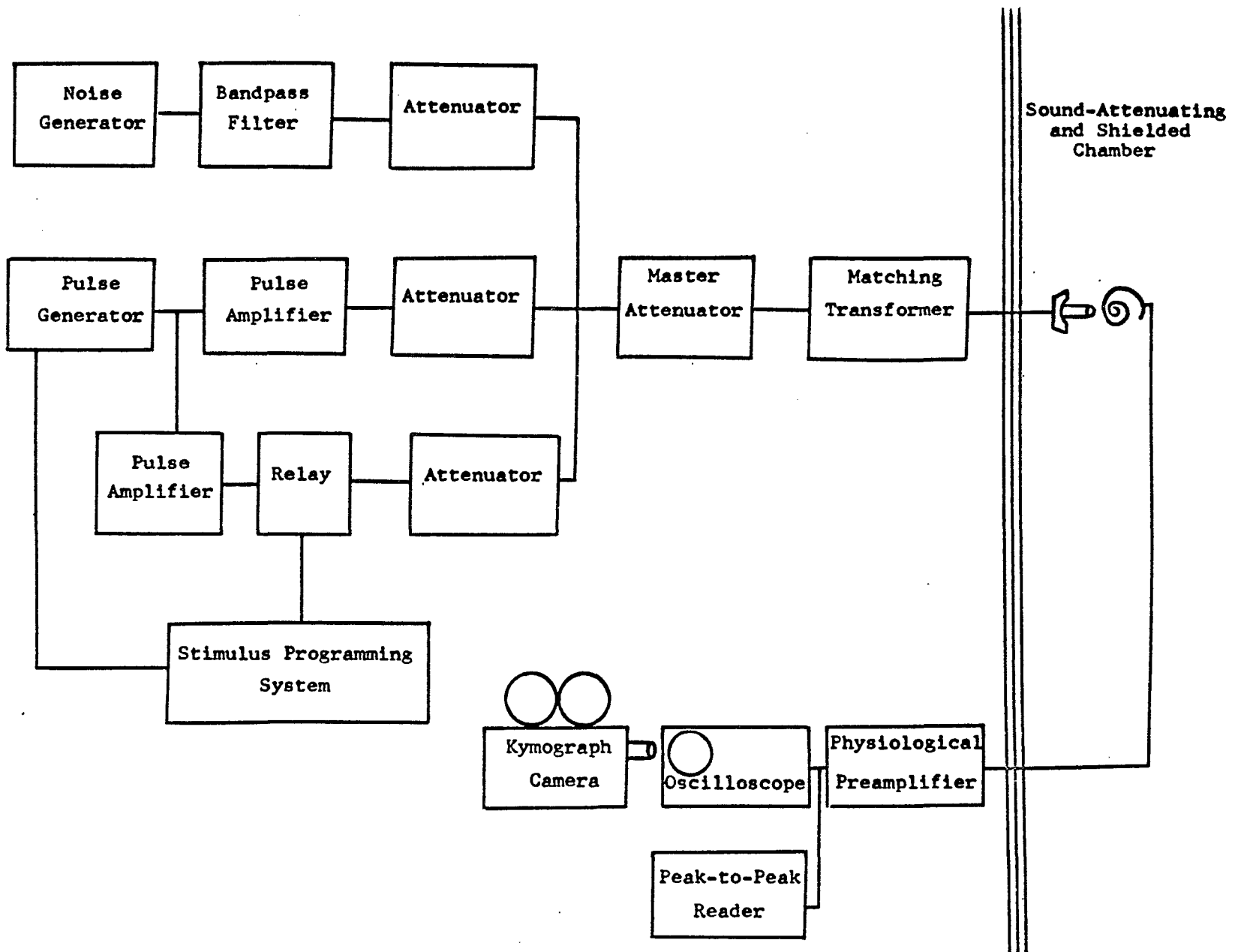
The outputs of the three stimulus channels were added in a resistive mixing network. Their sum was passed through a power attenuator, a matching transformer, and finally to the TDH-39 earphone.

Tektronix pulse and waveform generators were used to program the required sequences of stimuli.

In this experiment with guinea pigs, the click and noise levels are specified in decibels referred to the peak and RMS voltages, respectively, at the input terminals to the TDH-39 earphone. Sound pressures at the eardrum of the guinea pig corresponding to these voltages are difficult to specify exactly because the acoustic impedance of the earphone housing, the rubber tube, and the external meatus of the animal are difficult to simulate. However, an approximate assessment of the stimulus pressures was achieved by having one of the listeners of Experiment 1 (H.T.) hold the TDH-39 earphone-plus-tube against his ear. Absolute thresholds for both the click and noise stimuli were then obtained. Signal voltages (for absolute threshold) at the TDH-39 earphone were found to be within 2 dB

Figure 4

Block diagram of the apparatus employed in Experiment 2.



of those applied to the PDR-10 earphone (in its cushion) used in Experiment 1. Approximate comparisons between the stimuli employed in the two experiments can, therefore, be made. Specifically, for the animal experiments 0 dB clicks are approximately 88 dB SL (human); while the 0 dB noise is approximately 94 dB above human threshold.

(4) Recording

The active electrode and the reference electrode were connected to the differential input of a Grass (F-5) physiological preamplifier. The output was taken single-ended with a maximum gain of 14,000. The pass band of the preamplifier was 60-5000 Hz at the half-amplitude points. Neural responses were displayed on a Tektronix oscilloscope (502A).

For computing statistics of the N_1 responses, trains of clicks spaced one second apart were photographed and subsequently analyzed. A Grass 35 mm kymograph camera was employed to record the responses. Evoked response amplitudes were measured on a Kodagraph microfilm reader (11OR) by projecting the N_1 response onto a ruled grid. The accuracy of this system (oscilloscope, camera, microfilm reader, and grid) was checked by photographing calibration waveforms of high precision and was found to be linear to within 1%.

System noise, which could contaminate measurements of neural variability (see Chapter V), was evaluated by measuring the cochlear microphonic responses to a train of clicks. This

was done with two animals (1-31, 3-06). For the first guinea pig, relative variability (σ/M) equalled 0.8%; for the second experiment, variability equalled 3.2%. These figures, it should be noted, include noise contributed by all components of the stimulating and recording systems.

(5) "Physiological Difference Limens"

"Difference limens" for clicks were determined using a two interval staircase procedure resembling that of Experiment 1. Each trial consisted of two clicks spaced one second apart. A trial was initiated every three seconds.

The "limens" were obtained by comparing the amplitudes of the N_1 responses evoked by a standard (I) and an incremented ($I + \Delta I$) click. The decision rule employed required that the experimenter designate that stimulus as "incremented" which evoked the larger N_1 response.

On-line comparison of base-to-peak N_1 amplitudes (see Figure 1) presents severe difficulties. Low frequency shifts in the base line move the responses up or down on the oscilloscope tube face and add irrelevant variance to the voltage of the negative peak. Taking the difference between the baseline voltage and the negative peak could eliminate this variance, but this is difficult to accomplish. One would have to clamp to the voltage of the baseline at the moment when the response begins, but this moment fluctuates slightly in time and shifts markedly with changes in click intensity.

Peak-to-peak amplitudes (see Figure 1) are not affected by additive low-frequency noise. On-line read-out of peak-to-peak amplitudes was readily accomplished by the device described in Appendix A.

On each trial, which consisted of the I and $(I + \Delta I)$ stimuli, this "peak-to-peak reader" would generate and display two deflections whose amplitudes (from a fixed baseline) were each proportional to the peak-to-peak voltage of the N_1 response. The intensity of the oscilloscope trace was adjusted so that the first deflection persisted until after the presentation of the second waveform. The experimenter could rapidly and precisely determine which of the two successive evoked responses had the greater amplitude and he could not utilize the decision rule mentioned above.

On a single trial, the experimenter, who had foreknowledge of the temporal position of the $(I + \Delta I)$ stimulus, would determine whether the amplitude of the N_1 response produced by that stimulus was greater than the amplitude evoked by the standard click. If this was the case a "correct neural vote" was recorded. If, on the other hand, the $(I + \Delta I)$ click evoked the smaller response, that (smaller) response was deemed "incorrect".

If, in a block of four trials at a given level of ΔI , three "correct votes" were recorded, the level of ΔI was maintained for the next block of four trials. If "correct votes" were produced on all four trials in a block, the signal

for the next block was decreased by 2 dB. On the other hand, the signal was increased by 2 dB following only two "correct votes" in four trials.

Difference thresholds were computed as in the case of Experiment 1. At the end of a series of trials, the median value of attenuation in the signal channel was computed and then converted to increment power (ΔI), taking into account the in-phase addition of the standard and signal pulses.

Since early experiments revealed no difference in the magnitude of the limen as a function of the temporal sequence of the two clicks, the increment was always placed in the first position in later experiments.

Approximately 100 trials were employed for a single determination. Determinations were made for standard clicks from the visually detectable threshold (VDL--the minimum click intensity at which N_1 responses were first evoked--to 40 dB above VDL. At this level the cochlear microphonic usually began to distort the evoked response. "Physiological Weber functions" were, therefore, generated for approximately 40 dB of acoustic intensity.

The same procedure was employed in determining the difference limens and Weber functions for clicks presented against a noise background.

Chapter IV

RESULTS

Human Weber Functions

Figure 5 presents differential intensity limens for clicks in the absence of any noise background. The Weber functions for two subjects cover a range of approximately 80 dB and resemble those reported by Avakyan and Radianova (1963).

It should be noted that the DLs reported in this study are smaller than those reported by Avakyan and Radianova. The staircase procedure employed in this study avoids much of the variability of response encountered by Avakyan and Radianova, who used a method of adjustment.

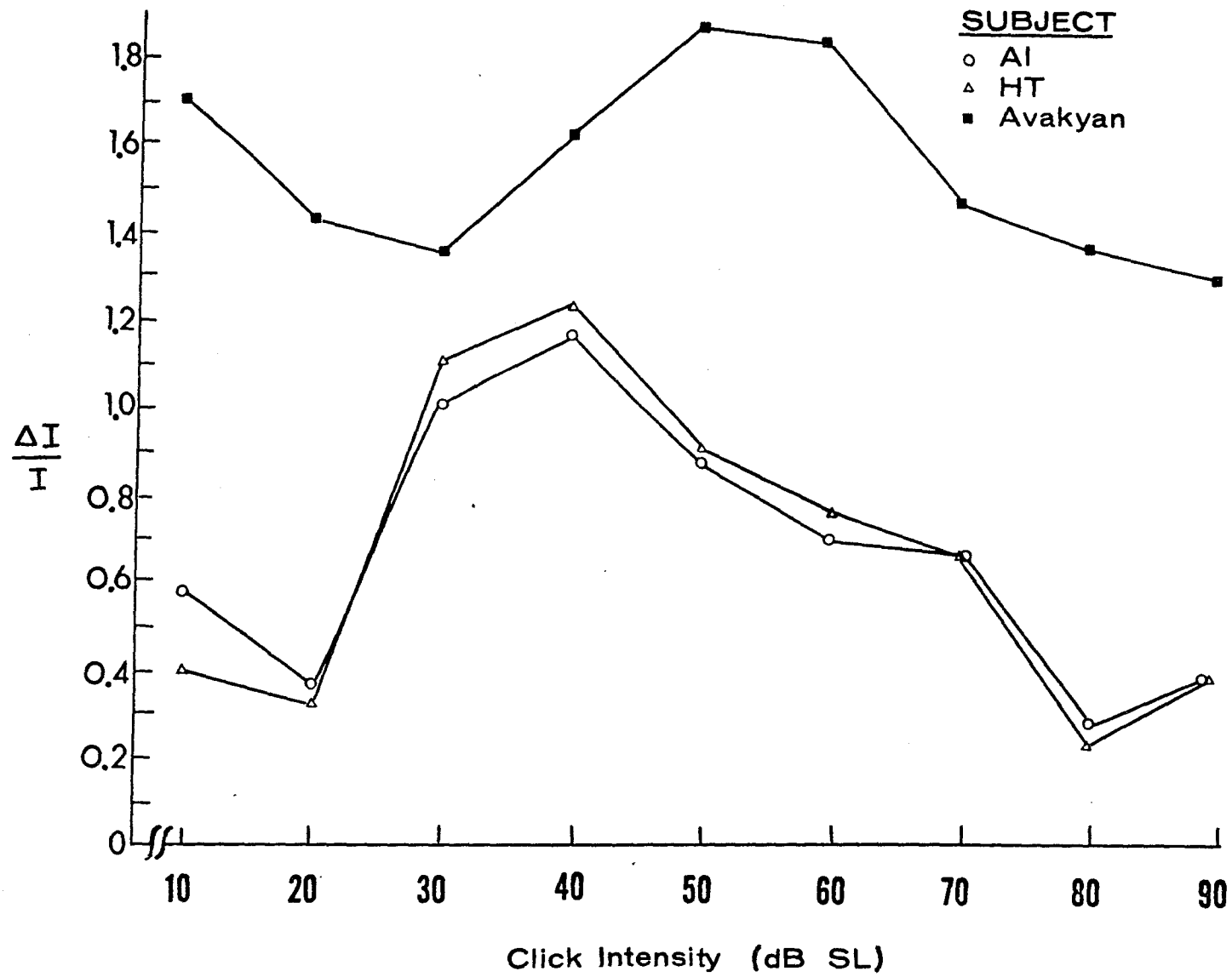
Noteworthy is the finding that these brief acoustic pulses are very difficult to differentiate with respect to intensity. At best, the Weber fraction is 0.25; at worse, ΔI exceeds I .

Markedly poor discrimination between brief pulses was also exhibited by the listeners in a recent study by Campbell and Lasky (1967). Signal/masker ratios for brief tone bursts (duration = 0.02 sec; frequency = 1000 Hz) are reported. Values of $\Delta I/I$ were computed from the authors' Figure 1. They range between 0.55 at 90 dB SPL and 1.50 at 40 dB SPL.

These Weber ratios compare poorly with those obtained with tones and noises of longer duration. Pettie (1959) presents

Figure 5

Human Weber functions for click stimuli. Intensity discrimination data for two listeners of Experiment 1 are compared with averaged Weber ratios reported by Avakyan and Radianova (1963).



relative intensity DLs for 0.75-sec tones of 1000 Hz and 6000 Hz. Both I and ΔI were gated together. Discrimination is much poorer for the high-frequency stimulus, but even here the largest Weber fraction is only 0.25.

Campbell (1966) employed one-second tone bursts of 250 Hz, 1000 Hz and 4000 Hz. Differential sensitivity was approximately the same for all three test frequencies over most of the intensity range. Values of $\Delta I/I$ were computed from signal/masker ratios plotted in Campbell's Figure 2. The Weber fraction for the 1000 Hz tone decreases from 1.1 at 5 dB SL to 0.18 at 85 dB SL.

Discrimination between long-duration noise bursts is also good. Harris (1950) reported that the DL for one-second bursts of noise (bandwidth = 7000 Hz) was approximately 0.18. More recently, difference thresholds for gated noise were presented by Green and Sewell (1962). The standard stimulus had a sound pressure level of 75 dB and a bandwidth of 4100 Hz. The average DL for one-second bursts was 0.16; for 0.1-sec bursts, the limen was 0.33.

It is clear that intensity discrimination is more difficult with brief click stimuli than with longer-lasting bursts of tone or of noise. Explanations for this finding are not to be found in the relative energy fluctuations of the various stimuli. Clicks and tone-bursts are, of course, virtually statistic-less. The 0.1 sec noise bursts used by Green and Sewell did vary somewhat from stimulus to stimulus, but the relative energy variation

(σ/M) was less than 5%. Neural variations rather than stimulus fluctuations must underlie all these results.

The finding (see Figure 5) that differential sensitivity deteriorates in the mid-range of click intensities is at variance with the Weber functions usually reported in hearing. Avakyan and Radianova (1963) seem to have been the first to call attention to this effect. More recently, humps in the Weber function have been described by Campbell (1964) for the case of noise bursts masked by continuous noise and by Campbell and Lasky (1967) and by McGill and Goldberg (1968) for pure tone intensity discrimination.

This phenomenon, too, seems to require an explanation in terms of neural mechanisms. Radianova, it will be recalled, attributed the elevation in the Weber function to diminished availability of eighth-nerve units in the mid-region of click intensity. Her argument is reminiscent of Crozier's. In this thesis, we shall endeavor to show that differential sensitivity for clicks is related to the slope of the amplitude-intensity function and the variability of neural response amplitude.

The effect of a continuous background noise on click intensity discrimination is given in Figures 6 and 7. The results are clear cut. So long as the clicks are audible in the noise, intensity discrimination is improved by the presence of the noise. The more intense the background, the greater is the improvement in differential sensitivity.

That discrimination between statistic-less pulses is improved by the addition of a noise background is unexpected.

Figure 6

The effect of masking noise on click intensity discrimination. Each data point is the mean of at least three determinations of the DL. The parameter is the sound pressure level of the continuous background noise. (Subject A.I.)

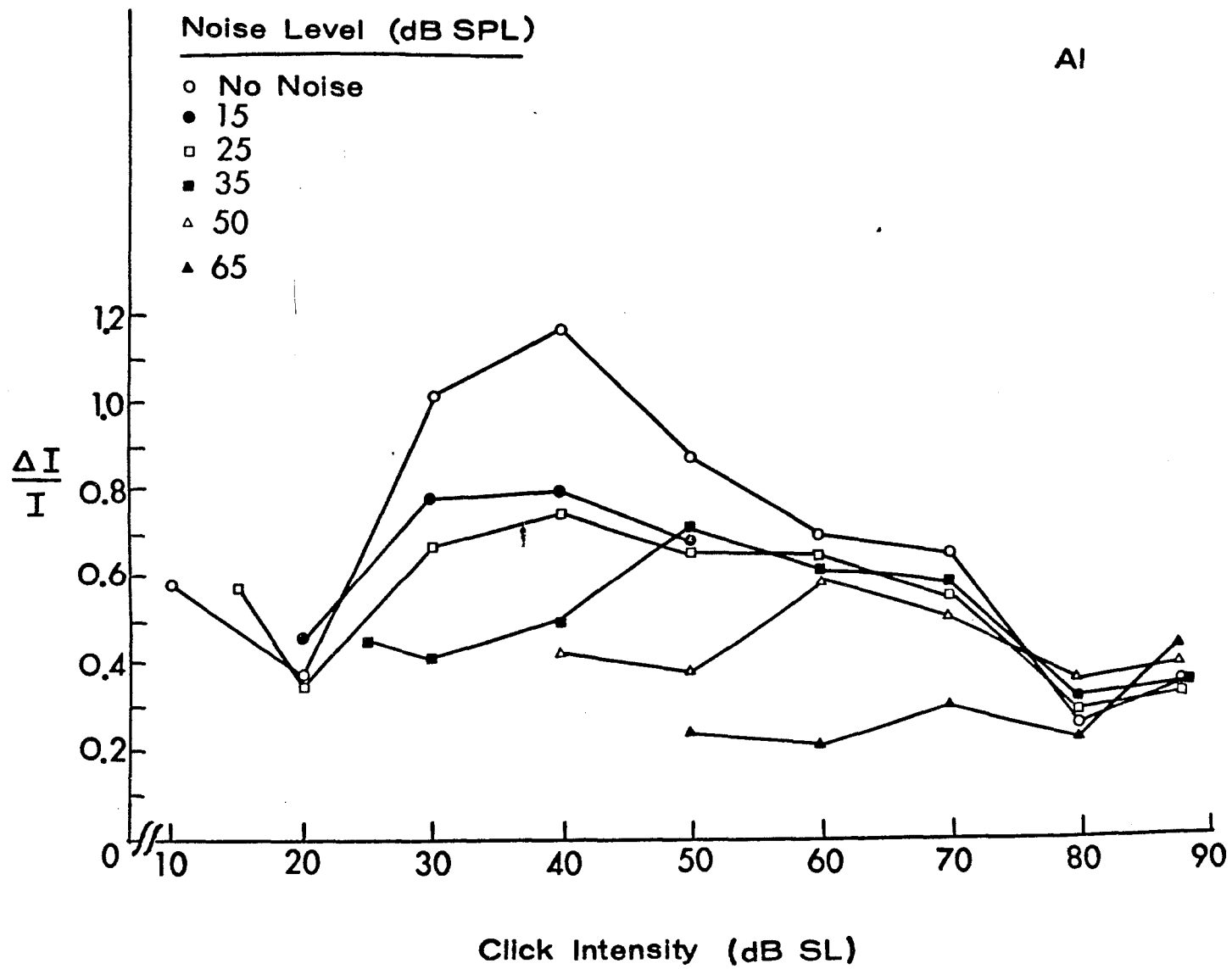
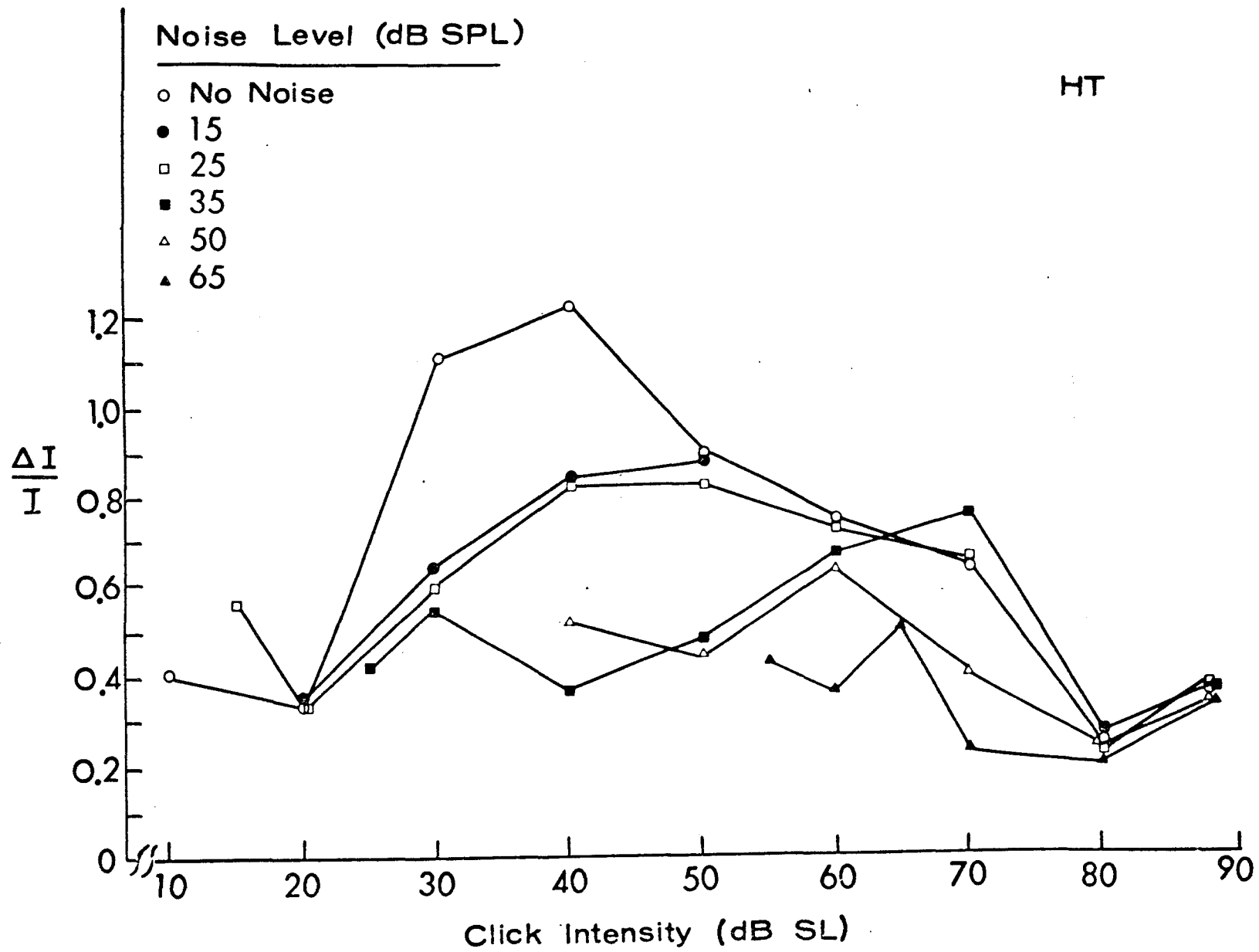


Figure 7

The effect of masking noise on click intensity discrimination. Each data point is the mean of at least three determinations of the DL. The parameter is the sound pressure level of the continuous background noise. (Subject H.T.)



Intensity discrimination between bursts of tone (Pettie, 1959) or of noise (Green and Sewell, 1962) does show little or no deterioration when a background noise is added. But no one has reported enhanced sensitivity to intensity differences in the presence of noise. We are constrained, once again, to look to the nervous system for possible mediating mechanisms.

In the section that follows, "physiological difference limens" are presented which are based upon the guinea pig's N_1 potential. The statistics of the N_1 response to clicks and to clicks in noise are analyzed and compared in the following chapter.

Physiological Weber Functions

Physiological difference limens for nine animals are presented in Figures 8 through 12. These were obtained by the on-line, non-parametric procedures described on pages 47 through 49.

With the exception of subject 1-25, whose limens are plotted in Figure 8, no attempt was made to obtain DLs above -40 dB. At higher levels of click intensity, the N_1 response was distorted by the cochlear microphonic. In the case of subject 1-25, an extremely "quiet" location for recording was found and the entire Weber function was obtained at this location. The unmasked function displays the characteristic hump found in the human Weber plots (see Figure 5) in the mid-region of intensity.

Figure 8

Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. The abscissa is the level of the standard click. A 0 dB click is produced by a 1.4 V pulse delivered to the earphone. The parameter is the level of the continuous background noise. At 0 dB, the noise voltage across the phone was 0.055 V. (Subject 1-25).

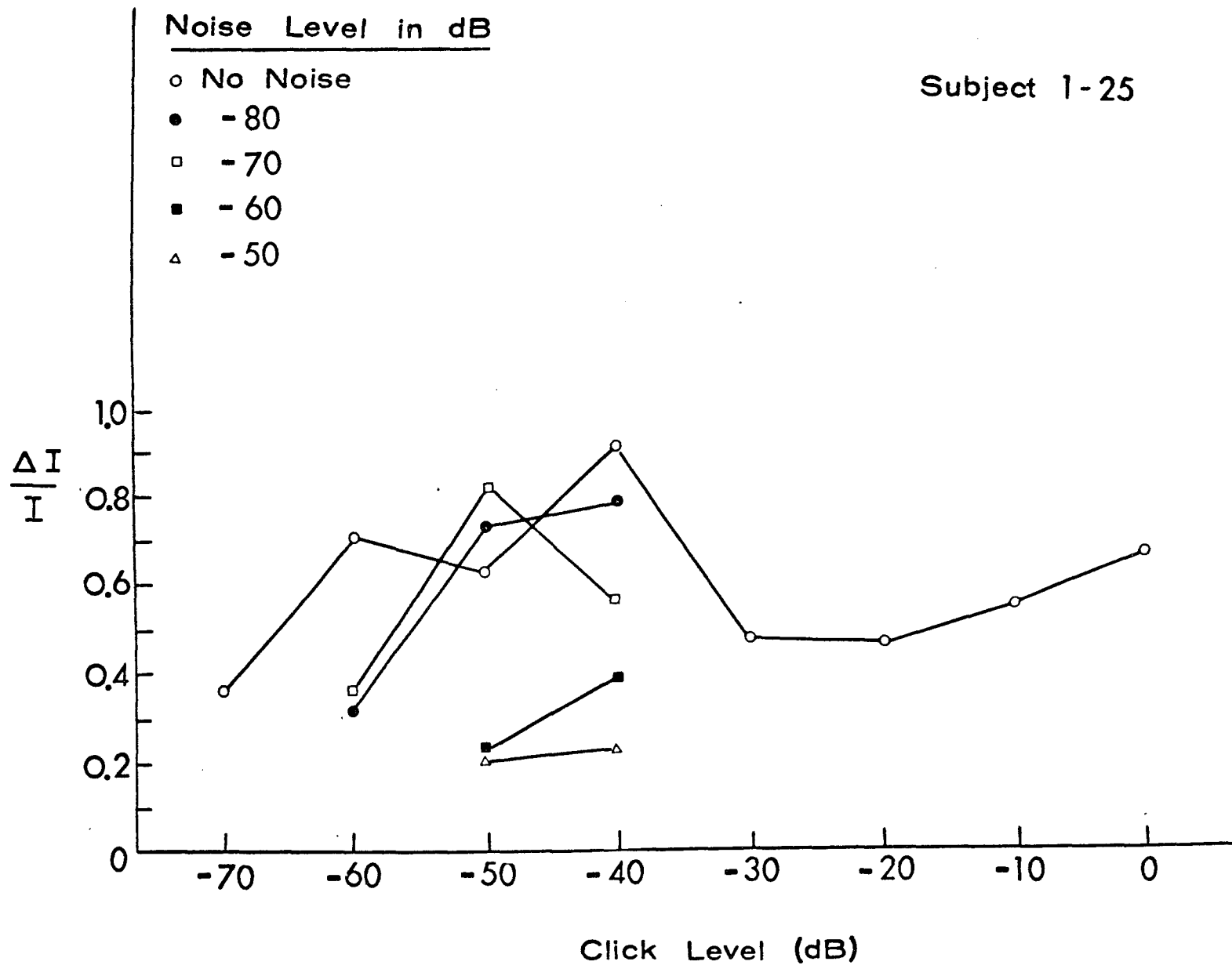


Figure 9

Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. The abscissa is the level of the standard click. A 0 dB click is produced by a 1.4 V pulse delivered to the earphone. The parameter is the level of the continuous background noise. At 0 dB, the noise voltage across the phone was 0.055 V. (A): Subject 2-27. (B): Subject 1-29.

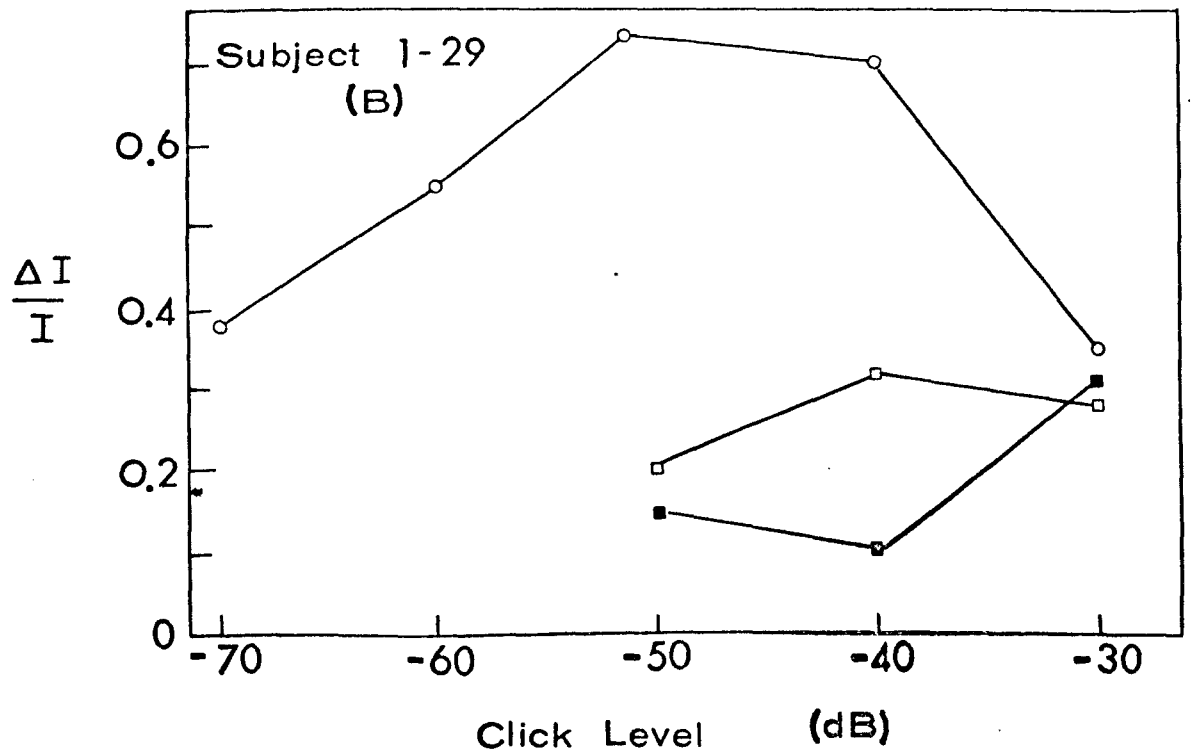
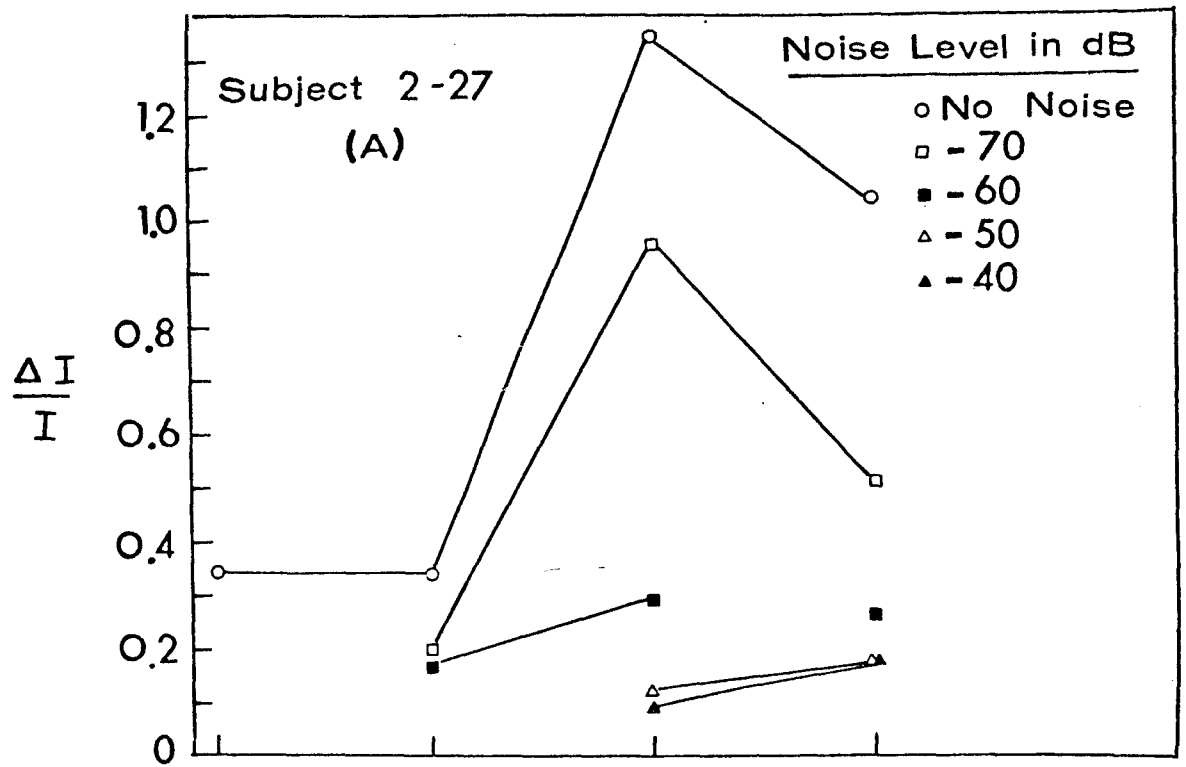


Figure 10

Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. The abscissa is the level of the standard click. A 0 dB click is produced by a 1.4 V pulse delivered to the earphone. The parameter is the level of the continuous background noise. At 0 dB, the noise voltage across the phone was 0.055 V. (A): Subject 3-22. (B): Subject 3-20.

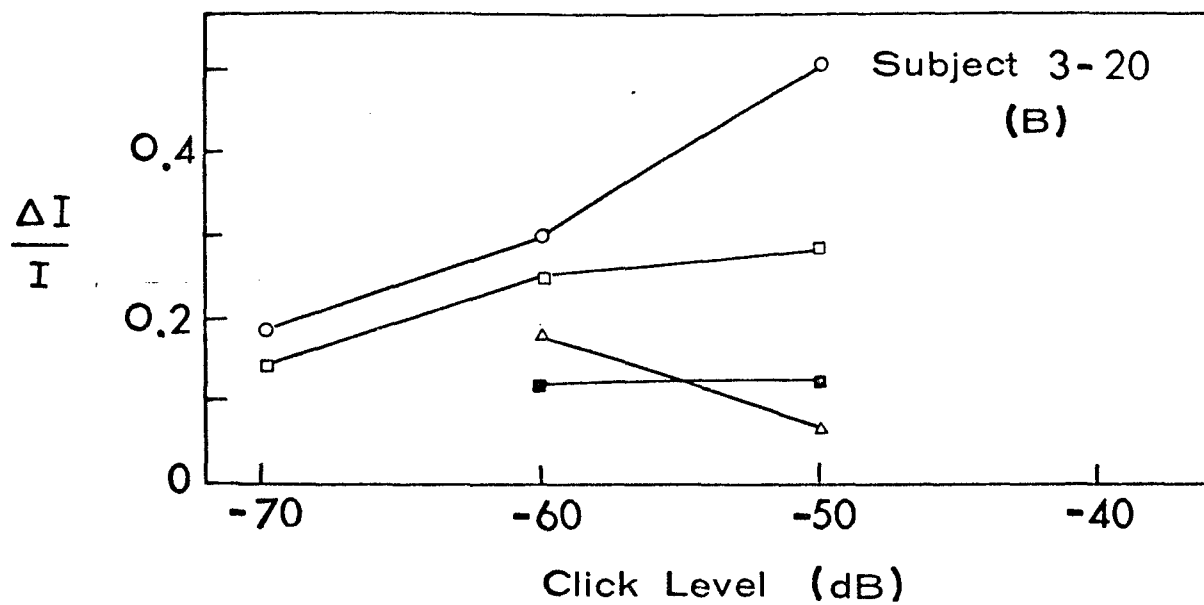
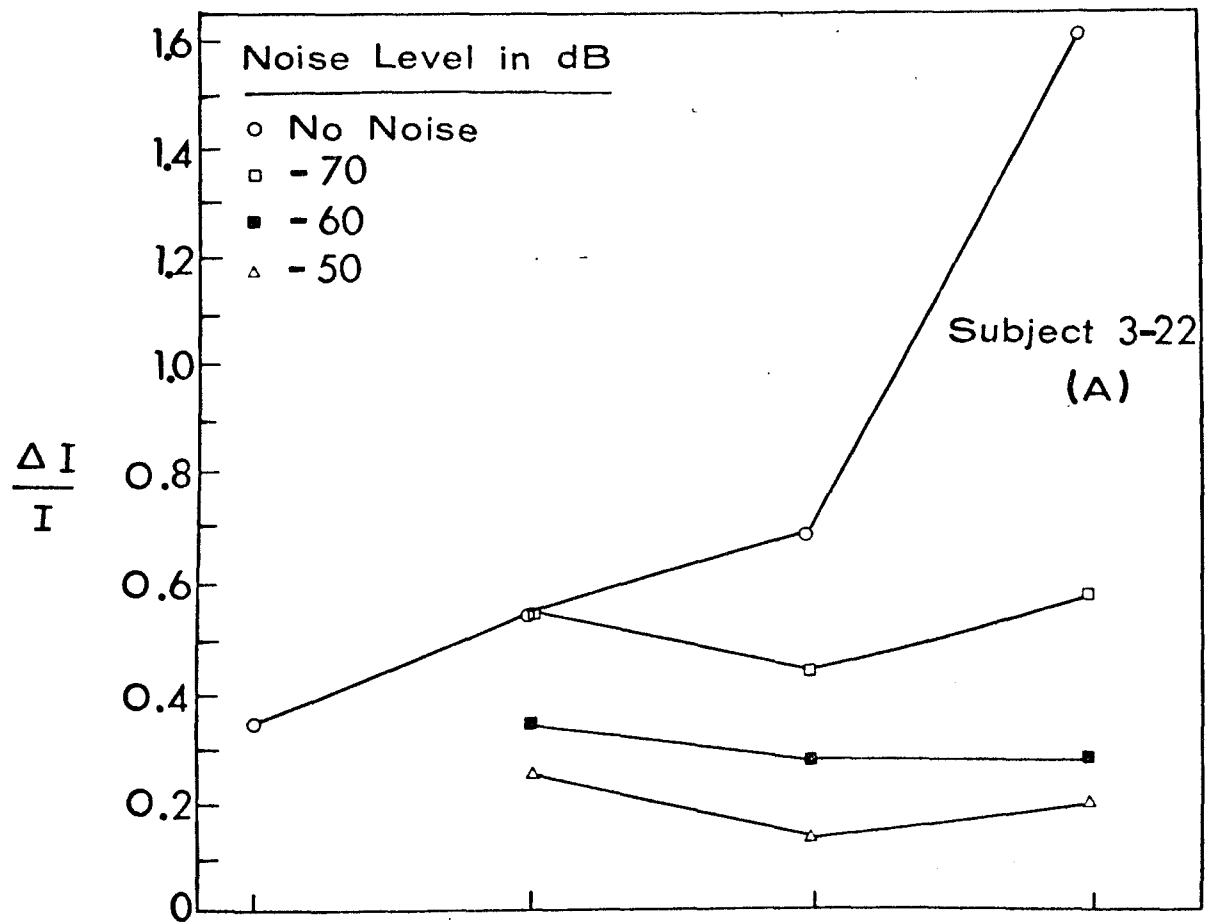


Figure 11

Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. The abscissa is the level of the standard click. A 0 dB click is produced by a 1.4 V pulse delivered to the earphone. The parameter is the level of the continuous background noise. At 0 dB, the noise voltage across the phone was 0.055 V. (A): Subject 2-14. (B): Subject 3-14.

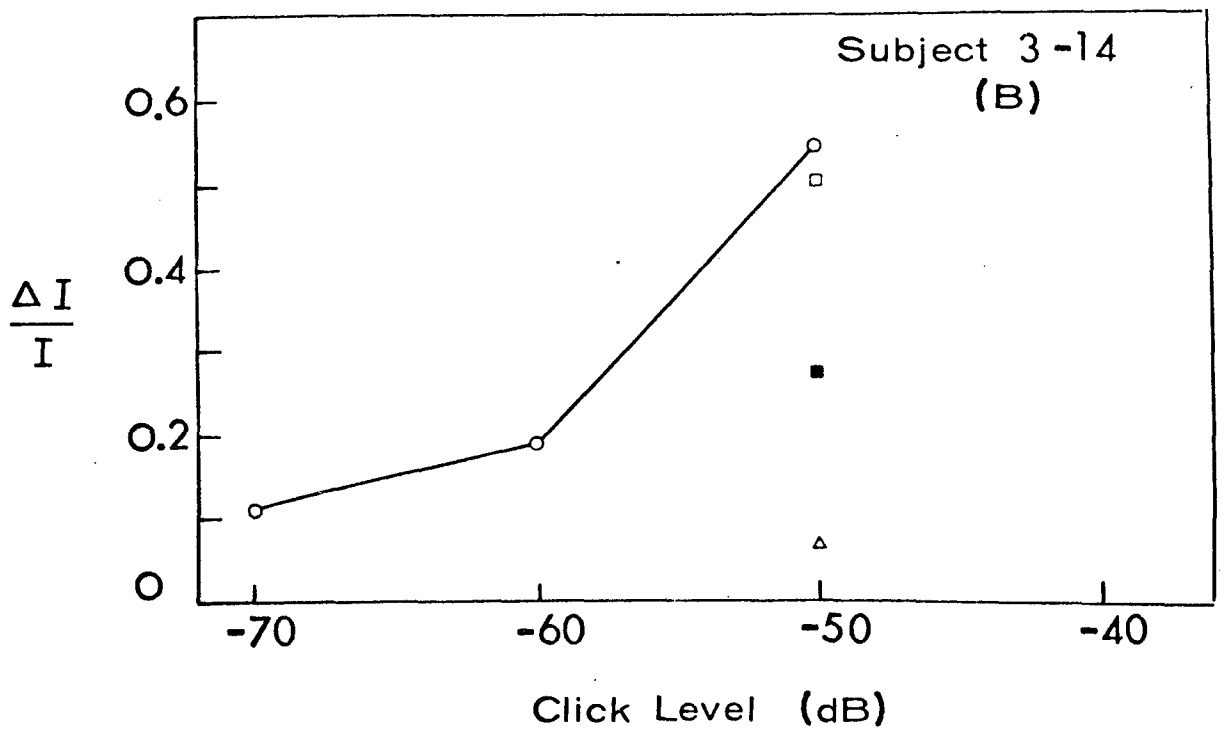
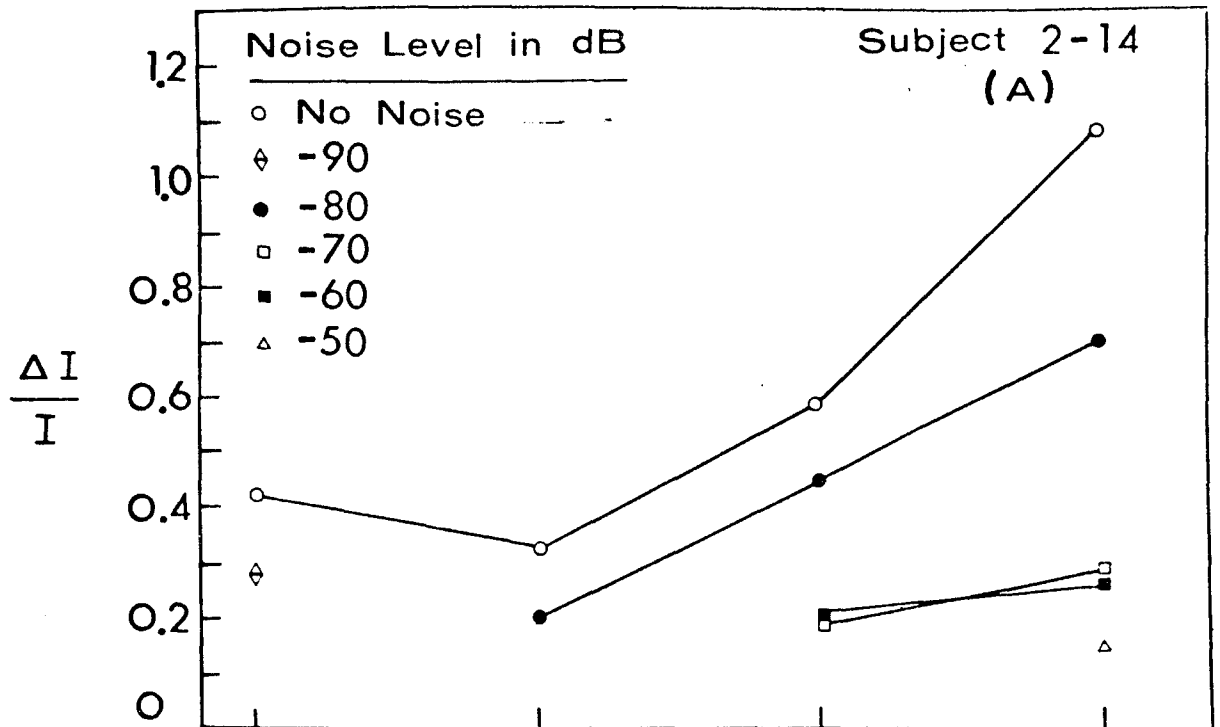
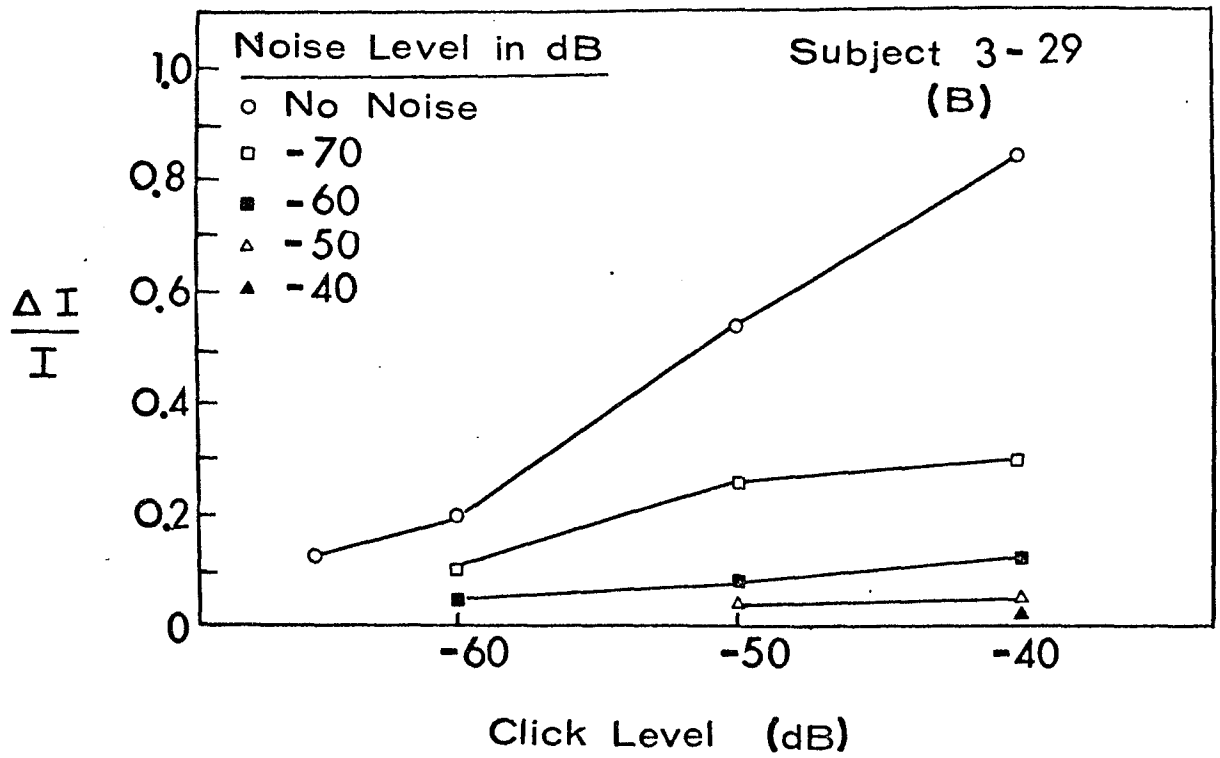
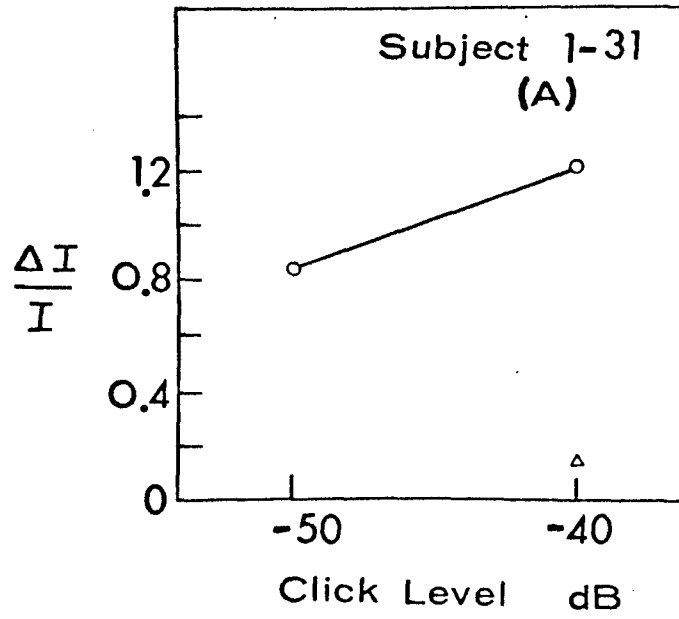


Figure 12

Physiological Weber functions for click stimuli. Each data point represents the value of $\Delta I/I$ that produced approximately 75% correct neural votes. The abscissa is the level of the standard click. A 0 dB click is produced by a 1.4 V pulse delivered to the earphone. The parameter is the level of the continuous background noise. At 0 dB, the noise voltage across the phone was 0.055 V. (A): Subject 1-31. (B): Subject 3-29.



The remaining unmasked Weber functions (Figures 9-12) show that differential sensitivity deteriorates as click intensity increases from "threshold." The Weber fractions are maximal in the region of -45 dB. For subjects 1-29 and 2-27 (Figure 9), sensitivity improves at click levels above -40 dB. Again, the similarity in the form of the Weber functions obtained with humans and with guinea pigs should be noted.

The effect of a continuous background noise in the physiological difference limen is described for nine animals in Figures 8 through 12. As was the case with human listeners, the DL is seen to diminish upon addition of a masking noise. Furthermore, the higher the level of masker, the greater is the improvement in differential sensitivity.

It is worthwhile to summarize the results presented this far.

Discrimination of click intensities is described by an unusual Weber function. We find, in agreement with Radianova (1963), that difference limens are maximal in the mid-region of click intensities.

Discrimination is improved by the addition of a continuous noise. This psychoacoustic result is both paradoxial and unprecedented.

These two effects are each closely paralleled by our "psychophysical" data based upon the N_1 response. In view of this parallelism and in view of the failure of stimulus-oriented theories to account for these findings with statistic-less

pulses, it is sensible to propose that the primary limitation on the human DL for clicks is set by the statistics of auditory nerve encoding--as exemplified by the N_1 potential.

If we propose as a model that the decision axis employed in click intensity discrimination is N_1 amplitude, then it becomes worthwhile to describe the statistics of the N_1 response under the various stimulus conditions we have employed. Additional experiments, whose results are presented in the following chapter, were performed in order to determine (1) the growth of N_1 amplitude with click intensity, (2) the form of the distributions of N_1 amplitude, and (3) the variability of N_1 amplitude.

Chapter V

DISCUSSION

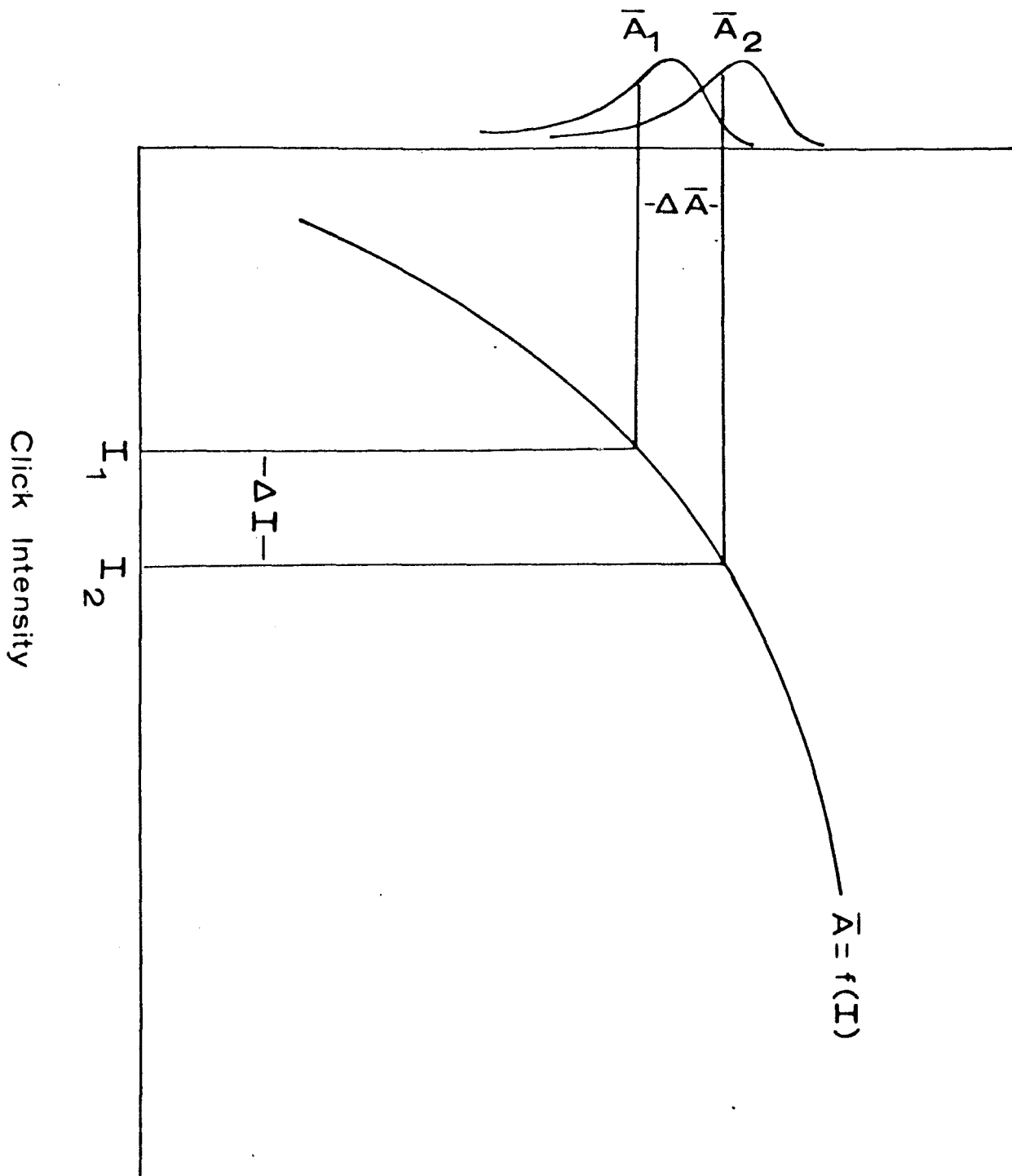
In the preceding chapter, discrimination of click intensities by human subjects was shown to be paralleled by "difference thresholds" based on the N_1 response of the guinea pig. The similarity between the psychophysical and physiological results now leads us to consider a model of click-intensity discrimination in which the decision axis is based upon N_1 amplitude. Inherent in the use of this decision axis is Mountcastle's "linearity hypothesis," which has previously been discussed (see pages 16 and 21). Essentially, the hypothesis states that the decision axis employed by real observers is a linear transform of the axis based on responses of first-order cells.

The model whose principal features are portrayed in Figure 13 is in the tradition of Blackwell and Treisman (see Chapter I). A click of intensity I_1 evokes--upon repeated presentation--a number of N_1 responses. The distribution of N_1 amplitudes has some form and has a mean (\bar{A}_1) and a standard deviation. The relation between I_1 and \bar{A}_1 is given by the "intensity function." In similar fashion, a more intense click (I_2) evokes a second distribution of N_1 amplitudes. In a forced-choice discrimination trial, the probability of a correct decision (based on N_1 amplitudes) is the probability that A_2

Figure 13

Model of click-intensity discrimination based on fluctuations of N_1 amplitude.

Amplitude of N_1



exceeds A_1 (see Figure 13). This probability is a function of the forms of the two amplitude distributions, their standard deviations, and their separation ($\Delta \bar{A}$).

It is commonly assumed that the two distributions have the same form and dispersion in the region defined by the psychometric function. If this form is known (e.g., normal, Poisson, etc.) and the sigma is specified, then the probability of a correct response, $p(A_2 > A_1)$, is a function of the separation of the distributions ($\Delta \bar{A}$). For a specified percentage of correct discriminations, e.g., 75%, the separation ($\Delta \bar{A}$) is then given. The click intensity (I_2) required to evoke \bar{A}_2 is then determined from the intensity function whose slope in the region of the threshold is obviously of critical importance.

(1) The Effect of Click Intensity on the Magnitude of the Physiological DL

The Weber functions presented by Radianova (1963) and those presented in the preceding chapter of the thesis demonstrate that differential sensitivity ($\Delta I/I$) for click intensity deteriorates as the intensity of the click is increased from "threshold" to approximately the mid-range of click intensities.

It follows from the present model of discrimination (see Figure 13) that deterioration in differential sensitivity could result from (a) a decrease in the slope of the log-intensity function in the region of I_1 , or (b) some change in the shape of the distributions of N_1 amplitude, or (c) an increase in the

variability of the N_1 distributions, or (d) some combination of these neural factors.

(a) While differential sensitivity progressively deteriorates from "threshold" to approximately 40 dB above, there is no corresponding change in the slope of the intensity function over this range of click intensities. Inspection of Figures 14 and 15 demonstrates for 4 animals (1-25, 1-31, 3-22 and 4-26) that the growth of N_1 amplitude with $\log I$ is approximately linear over the first 30 dB of click intensity. The linearity of this portion of the intensity function confirms data previously presented by Davis, 1935; Tasaki, Davis and Legouix, 1952; Frishkopf, 1956; Deatheridge, Eldridge, and Davis, 1956; Peake, 1959; Radianova, 1963; Kiang, 1965 and Dewson, 1968. The neural basis for the increasing Weber fraction is to be sought in other neural mechanisms.

(b) Radianova (1963) has presented the only data we have concerning the form of the distribution of N_1 amplitudes. Unfortunately, for our purposes, distributions are plotted for only one animal. Since histograms are based on only about 35 measurements, they are too crude for us to determine whether the distribution changes with intensity.

Figures 16 and 17 present amplitude distributions for N_1 responses measured in two animals (1-31, 3-22). For each distribution, approximately 105 click stimuli were presented at a fixed intensity. Inspection of these figures reveals that the form of the distribution does not change significantly over

Figure 14

Peak-to-peak amplitude of the N_1 response as a function of click intensity. Each data point is based upon approximately 15 responses to a click of fixed intensity. (A): Subject 1-25. (B): Subject 1-31.

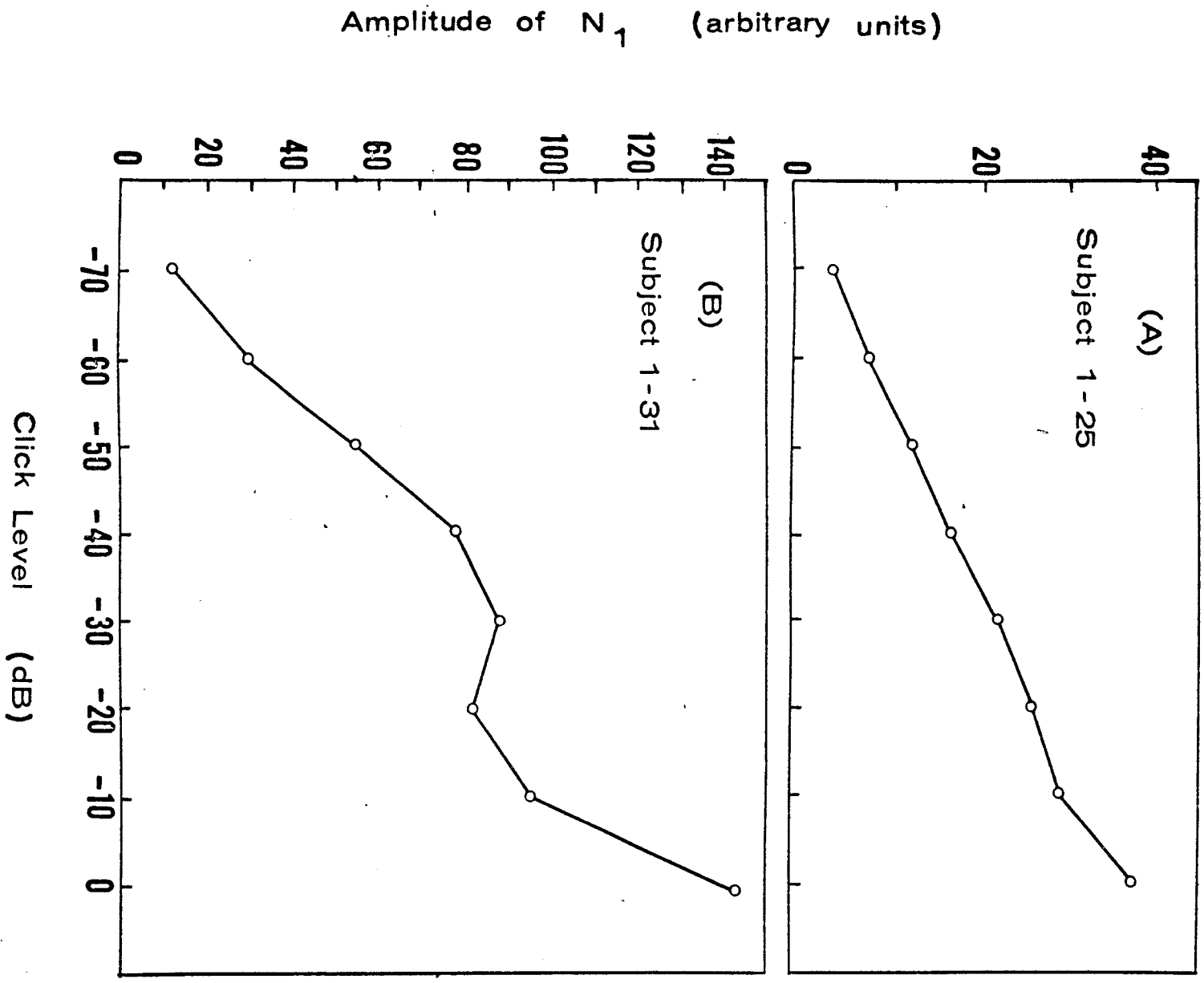


Figure 15

Peak-to-peak amplitude of the N_1 response as a function of click intensity. Each data point is based upon approximately 30 responses to a click of fixed intensity. (A): Subject 4-26. (B): Subject 3-22.

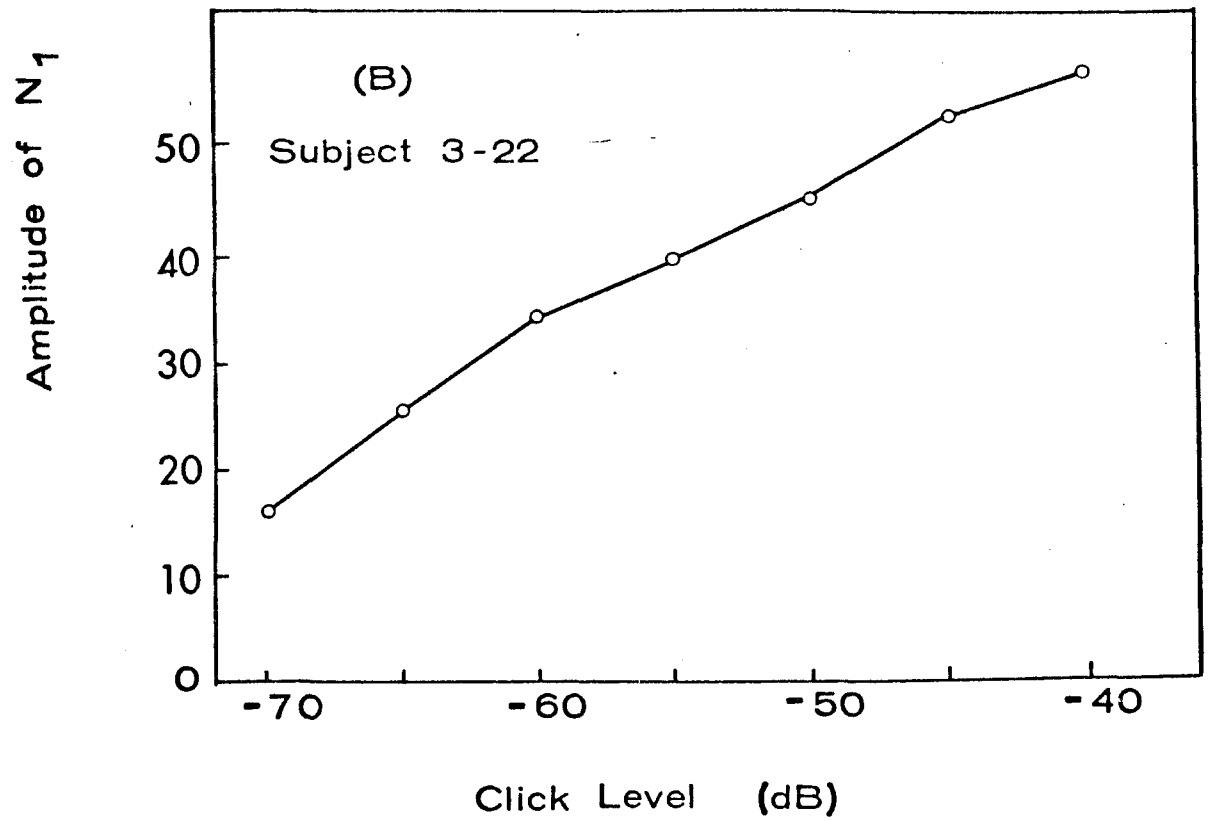
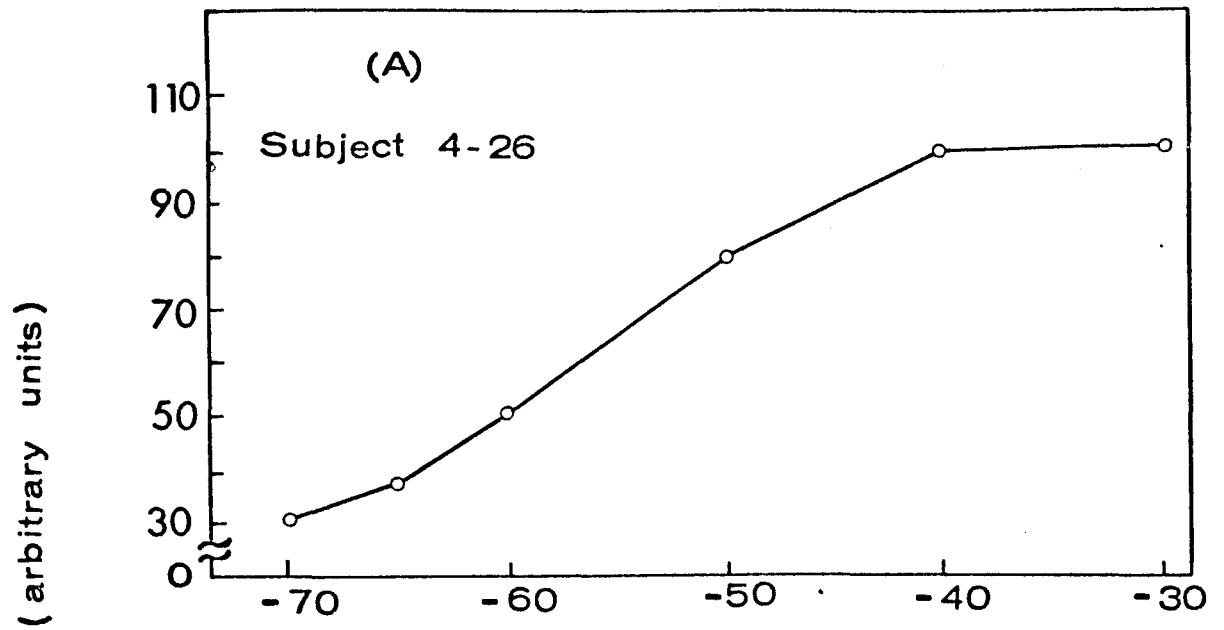


Figure 16

N_1 amplitude histograms as a function of click intensity.
For each histogram, approximately 130 responses were measured.
Subject 1-31.

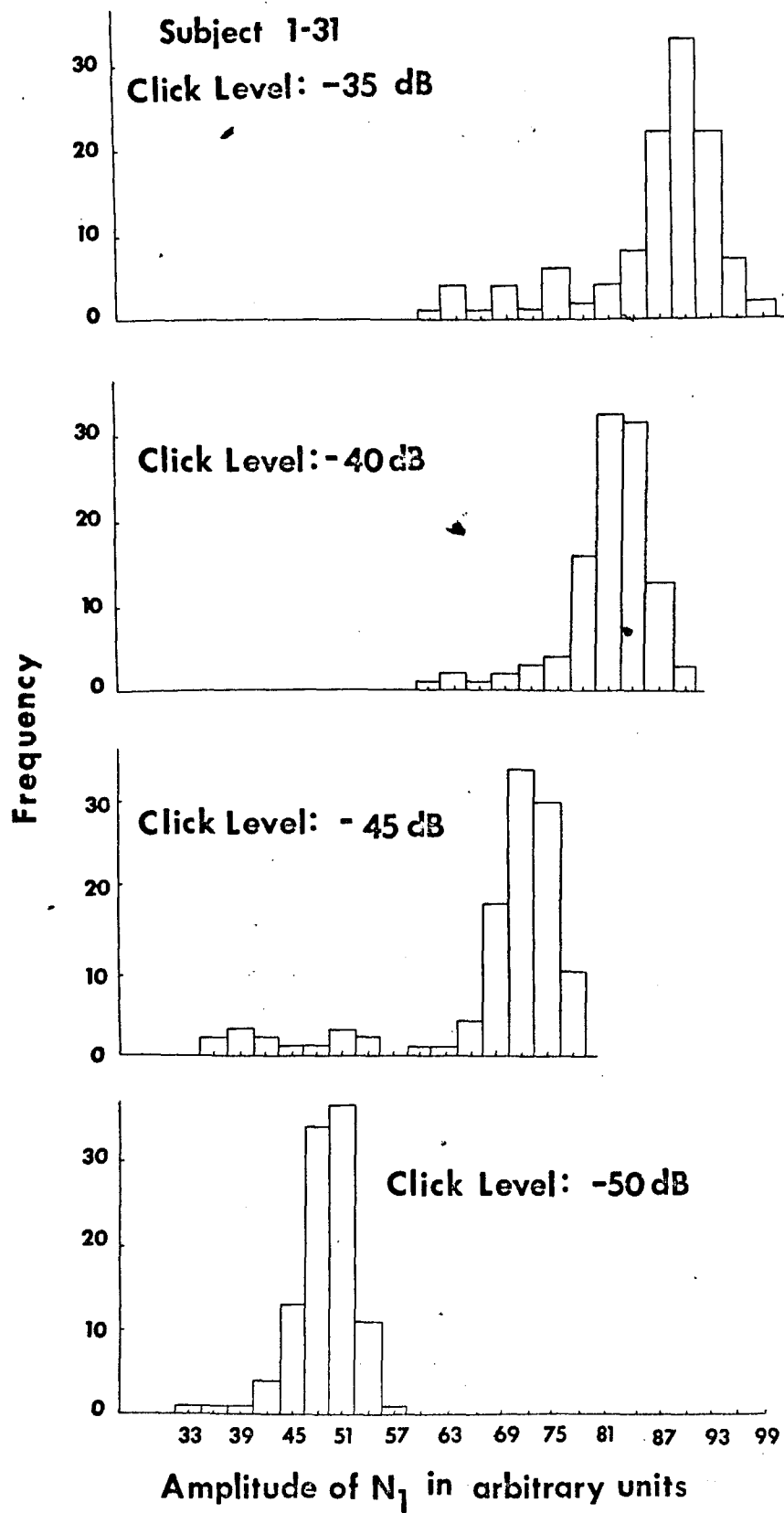
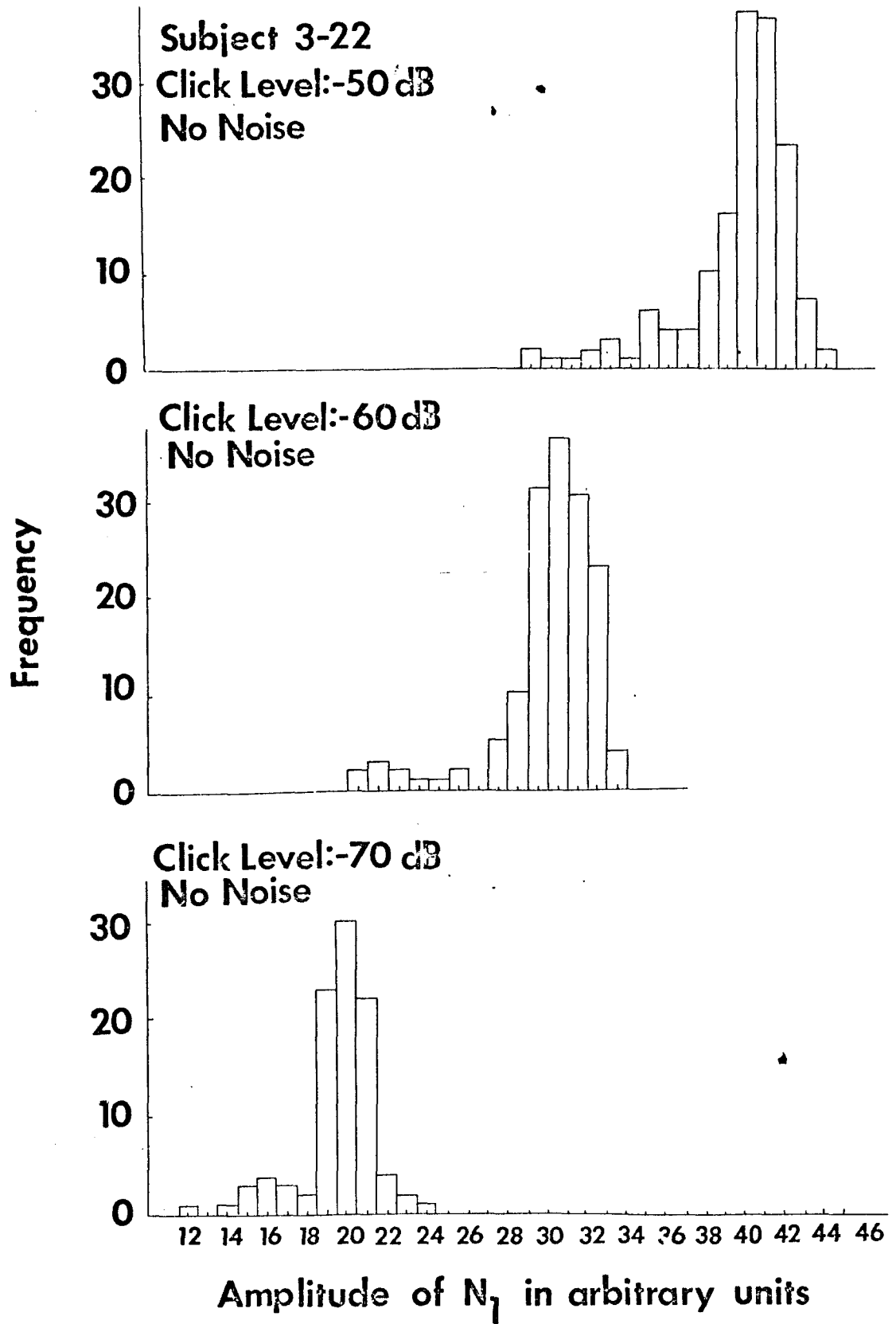


Figure 17

N_1 amplitude histograms as a function of click intensity.

For each histogram, approximately 130 responses were measured.

Subject 3-22.



the intensity-range of interest. For both animals, the amplitude distributions remain skewed as click intensity is changed. What is clear from an examination of Figures 16 and 17 is that variability of N_1 amplitude increases as stimulus intensity is increased.

(c) Frishkopf (1956) and Radianova (1963) have both investigated changes in the variability of N_1 as a function of click intensity. These investigators report that, as click intensity increases, dispersion of response amplitudes first increases, then decreases, then increases once again, and finally decreases. Unfortunately, Radianova's data (plotted as sigma functions) were not reproduced in the English translation of her paper. As described verbally, her functions do seem to resemble the variability functions presented by Frishkopf. Of interest to us are Frishkopf's plots which demonstrate that dispersion of N_1 increases for the first 20 to 30 dB above "threshold" and then decreases through the next 20 to 30 dB.

Figures 18 and 19 present sigma functions which are essentially similar in form to those given by Frishkopf. Each data point of Figure 18 is based upon approximately 40 responses to a click of constant intensity. The data points of Figure 19 are based upon approximately 150 responses each.

Special note should be made of the similarity in the form of these sigma functions and the physiological Weber functions presented in the previous chapter. The deterioration in differential sensitivity for approximately the first 30 dB of click

Figure 18

Standard deviation of N_1 amplitude as a function of click intensity. Each data point is based upon approximately 40 responses to a click of fixed intensity. (A): Subject 4-08. (B): Subject 4-26.

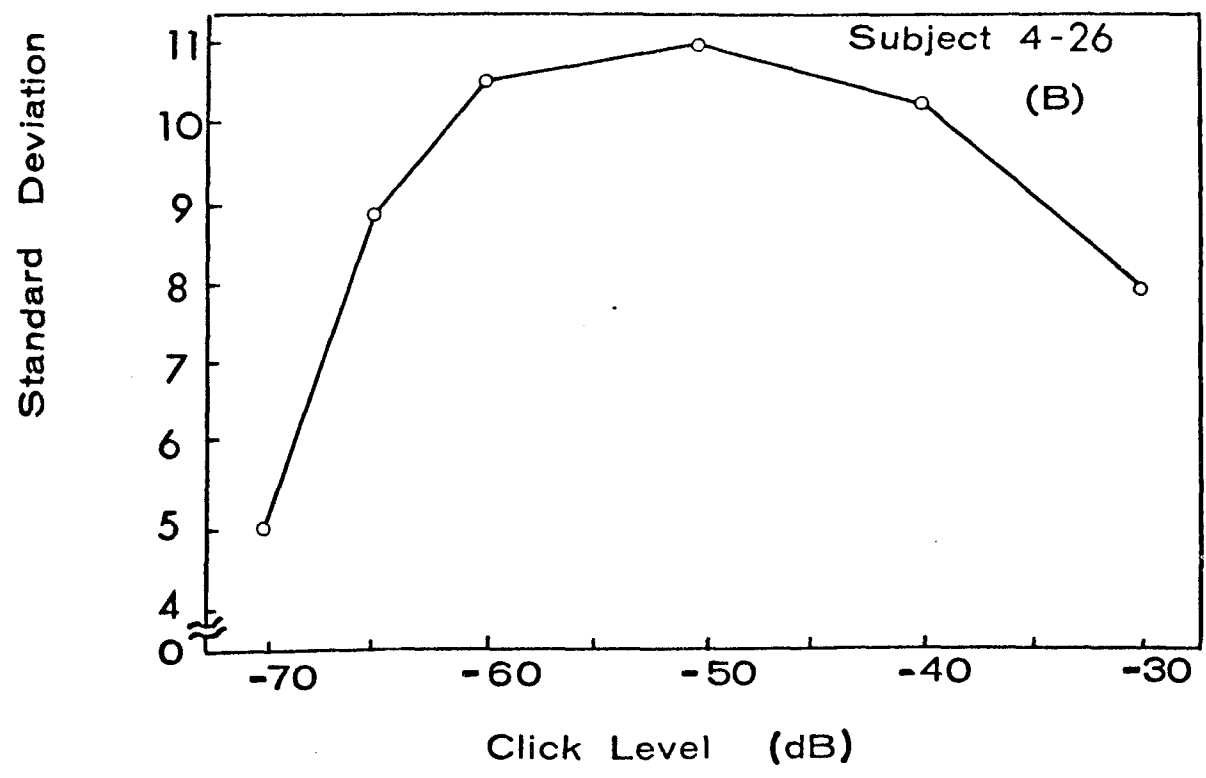
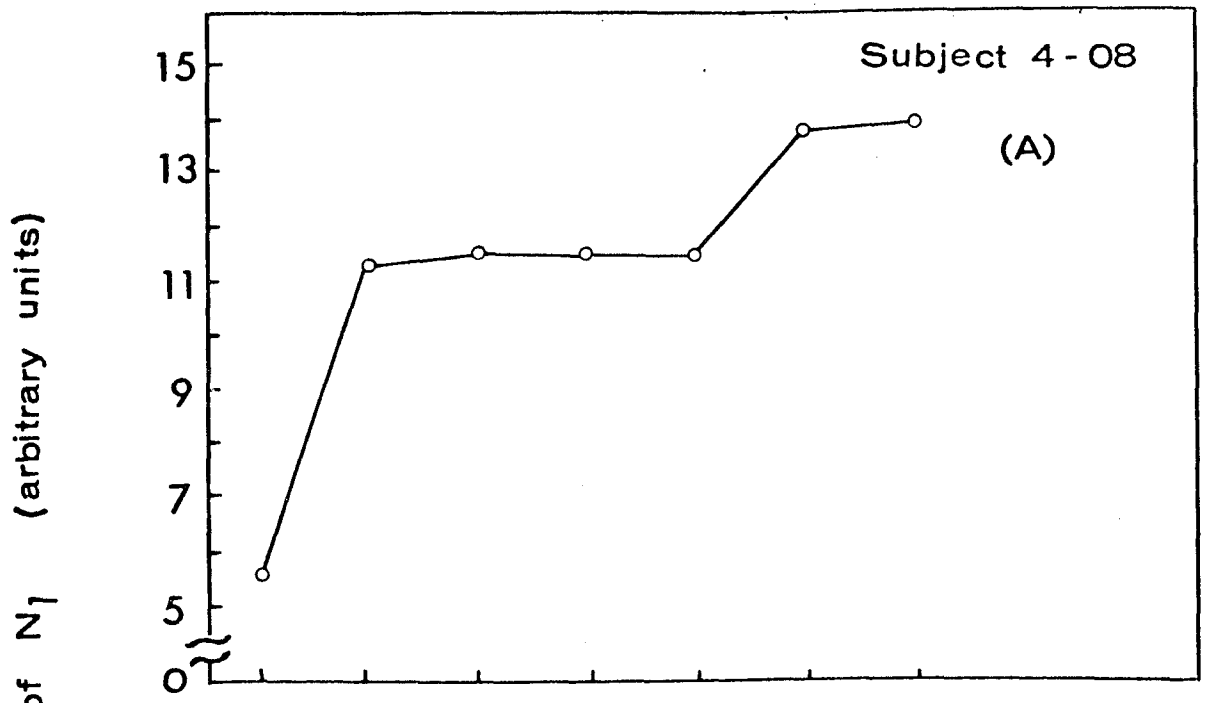
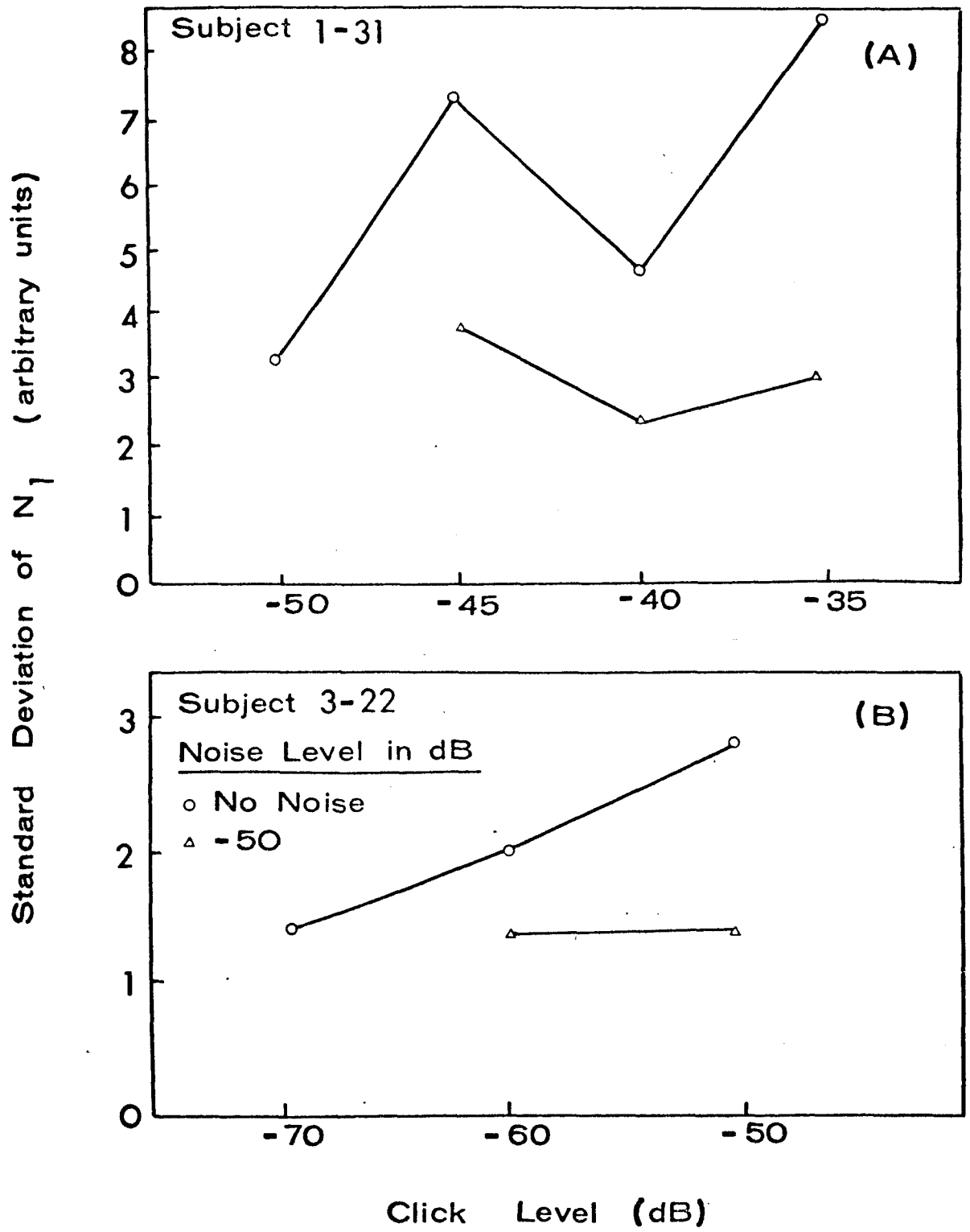


Figure 19

Standard deviation of N_1 amplitude as a function of click intensity. Each data point is based upon approximately 150 responses to a click of fixed intensity. (A): Subject 1-31. (B): Subject 3-22.



intensity is found to correspond to an increase in dispersion of N_1 over roughly this range of intensities. Likewise, recovery of differential sensitivity over approximately the next 20 dB is reflected in a diminution in the variability of N_1 .

The relation between differential sensitivity and response variability is directly available in the case of two animals (1-31, 3-22) whose intensity functions have already been presented.

For subject 1-31, examination of Figure 14B and 16 shows that both the slope of the intensity-function and the form of the amplitude distribution are unchanged as click intensity is increased from "threshold" to about 40 dB above. Physiological Weber fractions were determined at -50 dB and at -40 dB. They are respectively, 0.86 and 1.24 (see Figure 12A).

Standard deviations of N_1 amplitude were computed for clicks at -50 dB and at -40 dB. They are 3.21 and 4.37 respectively (see Figure 19A). The sigma at -40 dB is larger by a factor of 1.36. For constant detectability involving essentially identical neural distributions this increase in sigma requires an increase in the separation between the overlapping amplitude distributions. Specifically, the difference between the means ($\Delta \bar{A}$) at -40 dB must be larger than the difference at -50 dB by the same factor of 1.36.

Differences between the neural means ($\Delta \bar{A}$) are projected onto the log-intensity axis by means of the transfer function. Since the function appears linear over the intensity range in

question (see Figure 14B), the relative DL (in dB) at -40 dB must be greater than the DL at -50 dB by the factor of 1.36.

At -50 dB ($\Delta I/I = 0.86$); the relative DL was 2.69 dB. If this is inflated by the factor of 1.36, the relative DL at -40 dB would be predicted to be 3.66 dB and the Weber fraction would be 1.32. The Weber fraction actually obtained at -40 dB was 1.24.

A similar analysis of the data from subject 3-22 yields the following: The Weber fractions at -70 dB, -60 dB, and -50 dB were 0.35, 0.55, and 0.69 respectively. Standard deviations of neural amplitudes were 1.45, 2.00 and 2.54 at these three intensities. Again, the neural distributions were all essentially identical and the intensity function was more or less linear (see Figures 17 and 15B). The standard deviation at -60 dB is larger than at -70 dB by a factor of 1.37; at -50 dB sigma is larger by a factor of 1.75. Given the Weber fraction of 0.35 (at -70 dB), the value predicted for -60 dB and -50 dB are 0.51 and 0.69, respectively. These predictions are compared with obtained DLs in the table that follows.

It is clear that the effect of increasing click intensity on the physiological DL is almost entirely accounted for by the increase in N_1 variability. The slope of the intensity function and the form of the amplitude distribution remained constant with increasing click intensity.

The effect of click intensity on variability of N_1 amplitude has been treated by Frishkopf (1956), who has provided the most

Table 1

Comparison of obtained physiological Weber fractions with those predicted from the neural model of intensity discrimination. For the case of unmasked clicks, the principal parameter of the model was variability of N_1 amplitude (see text).

Subject	Obtained	Predicted
1-31	1.24	1.32
3-22	0.55	0.51
	0.69	0.69

extensive data together with a binomial model of the N_1 response. The sigma-functions presented in this thesis resemble those plotted by Frishkopf. That sigma increases and then decreases as click intensity is increased from "threshold" to about 40 dB above "threshold" is predicted by Frishkopf's model. Frishkopf considers that the probability of firing a neural element (p) increases with click intensity. While the same threshold probability distribution characterizes all N elements comprising a population, the threshold fluctuations of all elements are independent. As a result, the mean number of elements (Np) fired by a click increases with stimulus intensity. The standard deviation, which equals $\sqrt{Np(1-p)}$ ^{1/2} increases with p ; is maximal at $p = 0.5$; and then decreases when $p > (1-p)$.

It is clear from the above analysis that the changes (with intensity) in the physiological DL reflect changes in N_1 variability. The similarity between the psychophysical and the physiological Weber functions leads to the notion that the former are based on the latter. Additional support for these notions comes from an analysis of the effects of masking noise.

(2) The Effect of Addition of a Continuous Background Noise on the Physiological DL.

It will be recalled (Chapter IV) that the addition of a continuous background noise produced the unusual effect of improved differential sensitivity. Furthermore, increasing

the intensity of the background noise resulted in greater improvements in sensitivity. This result was obtained with both human observers and guinea pigs (physiological DLs).

As indicated in Chapter III, theories based on stimulus statistics cannot predict these results. In fact, the addition of noise serves to introduce energy variations to a statisticless click stimulus. Theories based on energy fluctuations would predict a deterioration in differential sensitivity with the introduction of masking noise--a result opposite to the one actually obtained.

The basis for improved discrimination was sought, therefore, in the parameters of the neural model discussed above. Specifically, the effect of noise on the slope of the intensity function and on the form and variance of the neural amplitude distributions will be considered

(a) Intensity functions for two animals (3-22 and 4-29) were obtained with and without masking noise. These are plotted in Figures 20 and 21. It can be seen that the effect of noise is to diminish the amplitude of the N_1 response--a phenomenon first described by Davis (1935). Increasing the intensity of the ongoing noise results in greater diminutions of N_1 amplitude. Furthermore, as has been reported by Frishkopf (1956), Peake (1960), and Dewson (1967), a given level of noise diminishes the N_1 response more or less equally along the intensive continuum. The effect, as displayed in Figures 20 and 21, is to shift the intensity function downward while maintaining approximately equal slopes throughout the family of

Figure 20

Intensity function for clicks presented with and without noise background. The peak-to-peak amplitude of the N_1 response is plotted as a function of click intensity. The parameter is the noise level in dB re 55mV across the earphone. Each data point is based upon approximately 30 responses to a click of fixed intensity. Subject 3-22.

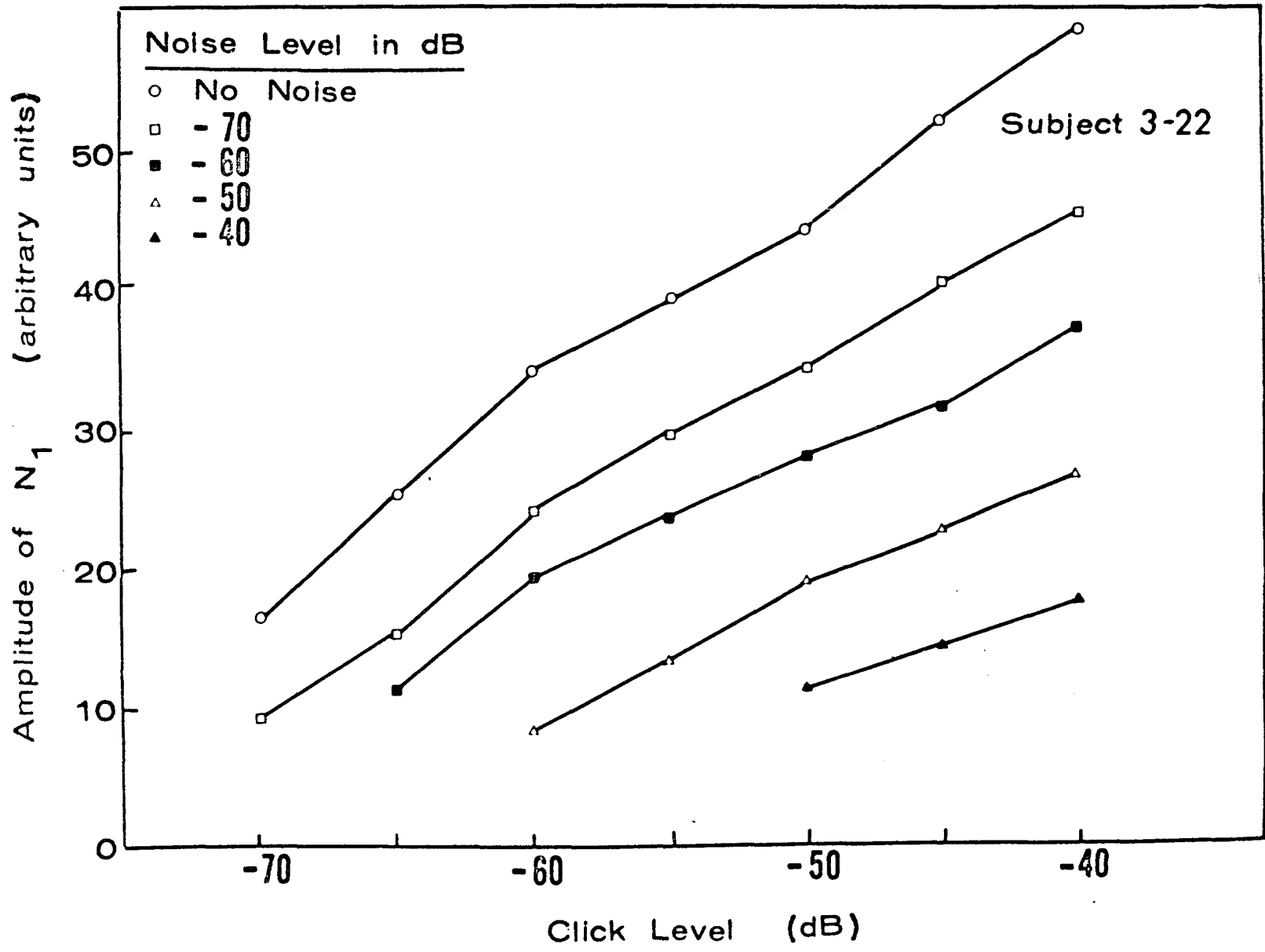
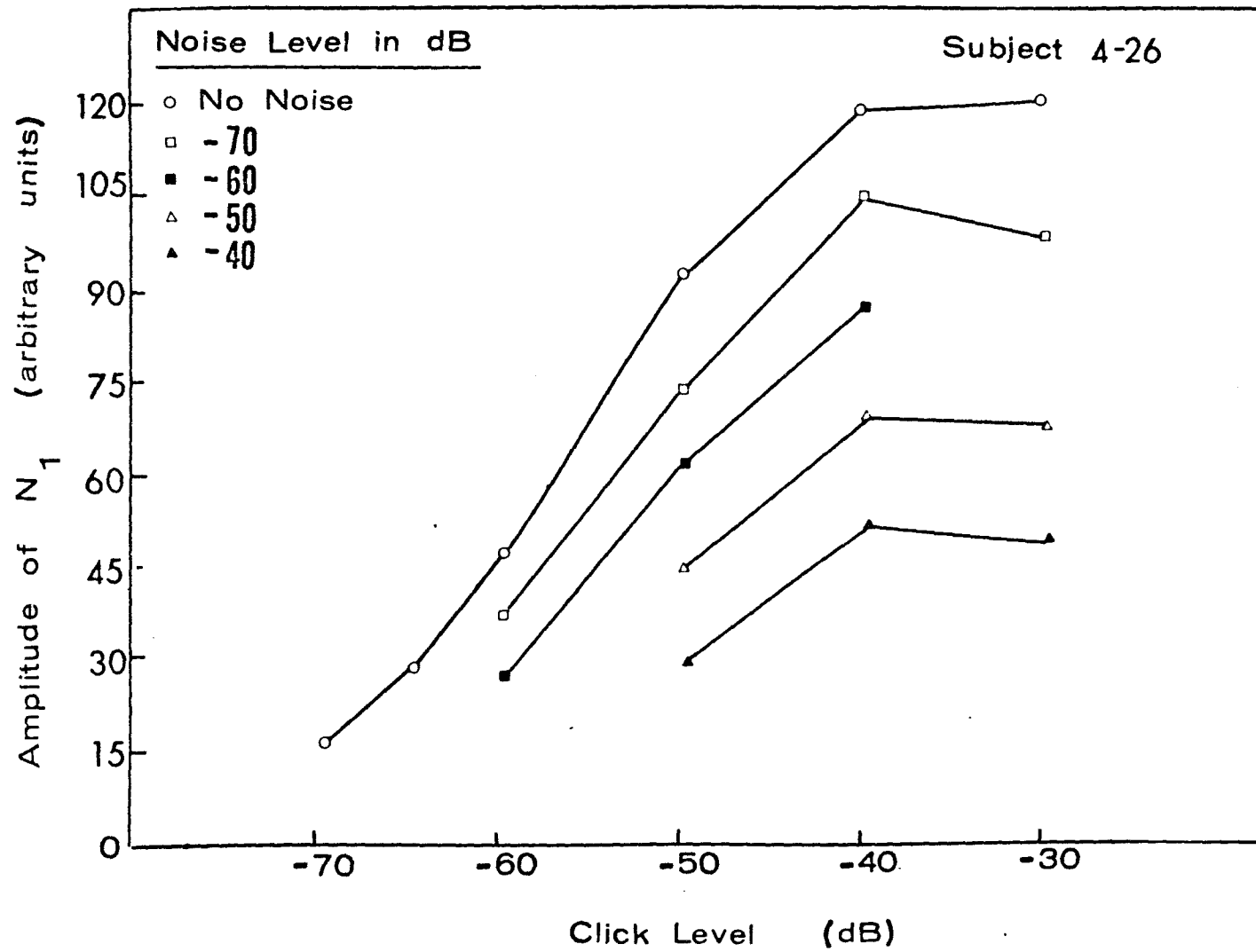


Figure 21

Intensity function for clicks presented with and without noise background. The peak-to-peak amplitude of the N_1 response is plotted as a function of click intensity. The parameter is the noise level in dB re 55mV across the earphone. Each data point is based upon approximately 30 responses to a click of fixed intensity. Subject 4-26.



functions.

(b) Neural amplitude distributions for four animals (1-31, 3-22, 3-14, and 3-06) are plotted in Figures 22 through 25. Examination of these figures reveals that, for each intensity of click, the addition of noise decreases N_1 amplitude (see above) and transforms obviously skewed distributions of amplitude into more symmetrical ones.

Elimination from the histogram of the long, left hand tails has two effects of interest. First, the form of the amplitude distributions is altered; second, variability of amplitude is diminished. To investigate the effect of changes in symmetry as such on physiological DLs, the following analysis was performed.

The percentage of correct responses in the two-alternative, forced-choice (2AFC) experiment is readily available from the difference distribution when the "I" and "I + Δ I" distributions are normal. (The difference distribution for this case is also normal.) No derivation was immediately available for the forced-choice procedure involving markedly skewed distributions. Accordingly, a graphical method was employed to obtain an approximate value for the percentage of correct responses, $P(c)$, for a case involving skewed distributions. This method is based upon the theorem (see Green and Swets, 1966, pages 45-47) that the area under the receiver operating characteristic curve (ROC) equals $P(c)$ in 2AFC procedures.

For the case of two overlapping normal distributions whose

Figure 22

Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. For each histogram approximately 130 responses were measured. Subject 1-31.

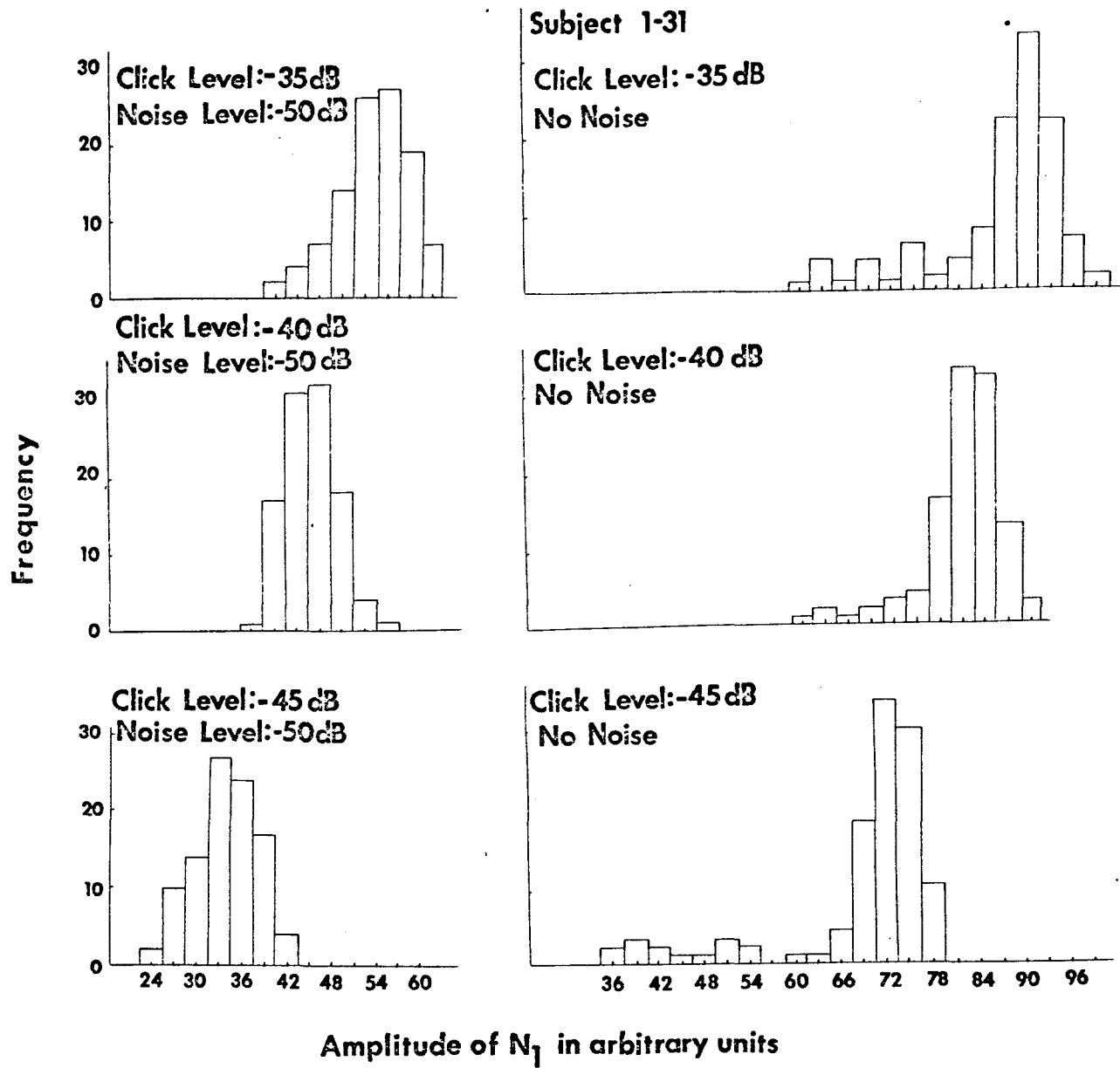


Figure 23

Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. For each histogram approximately 130 responses were measured. Subject 3-22.

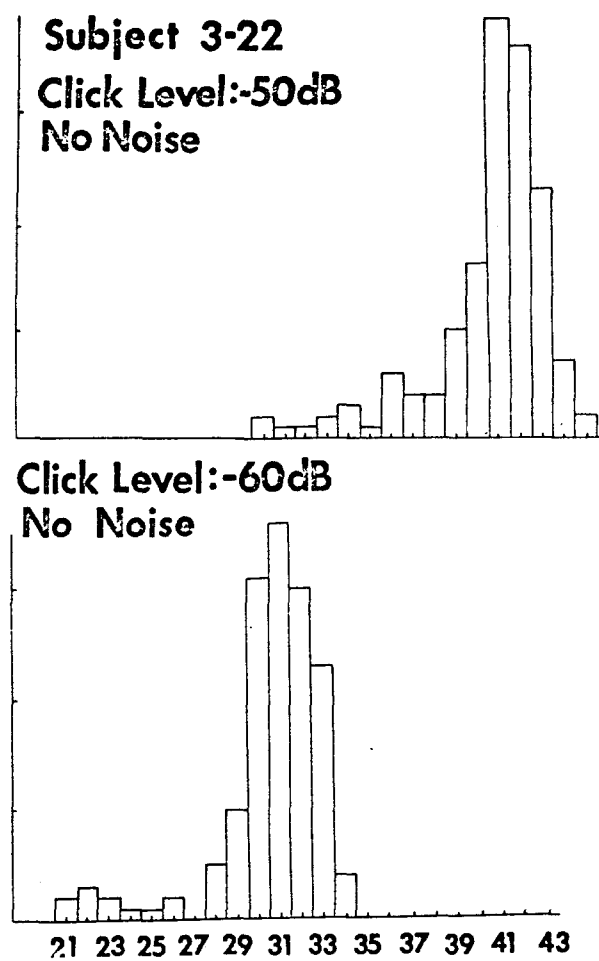
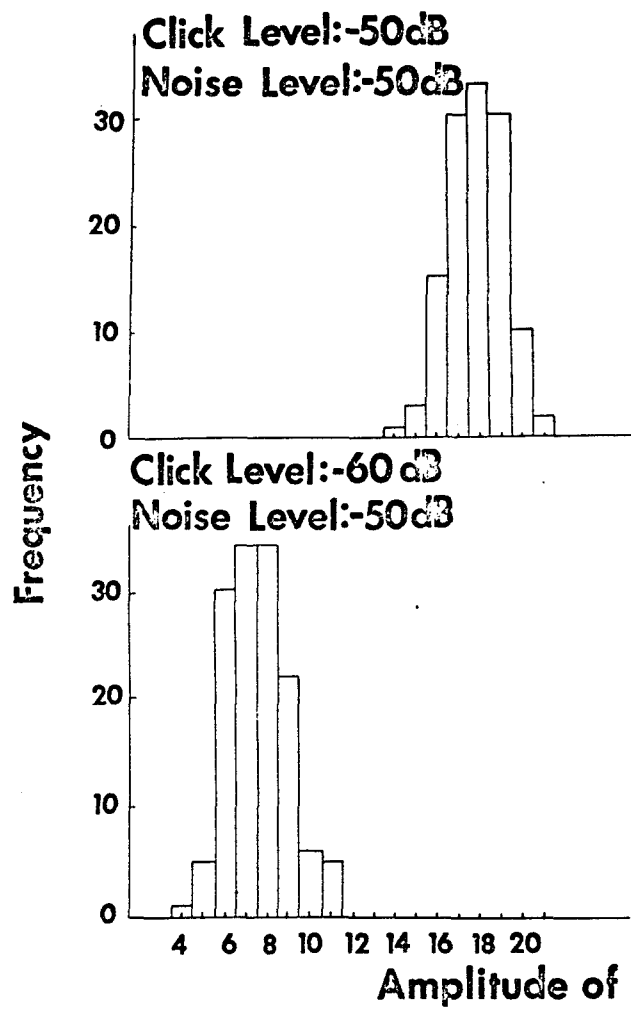
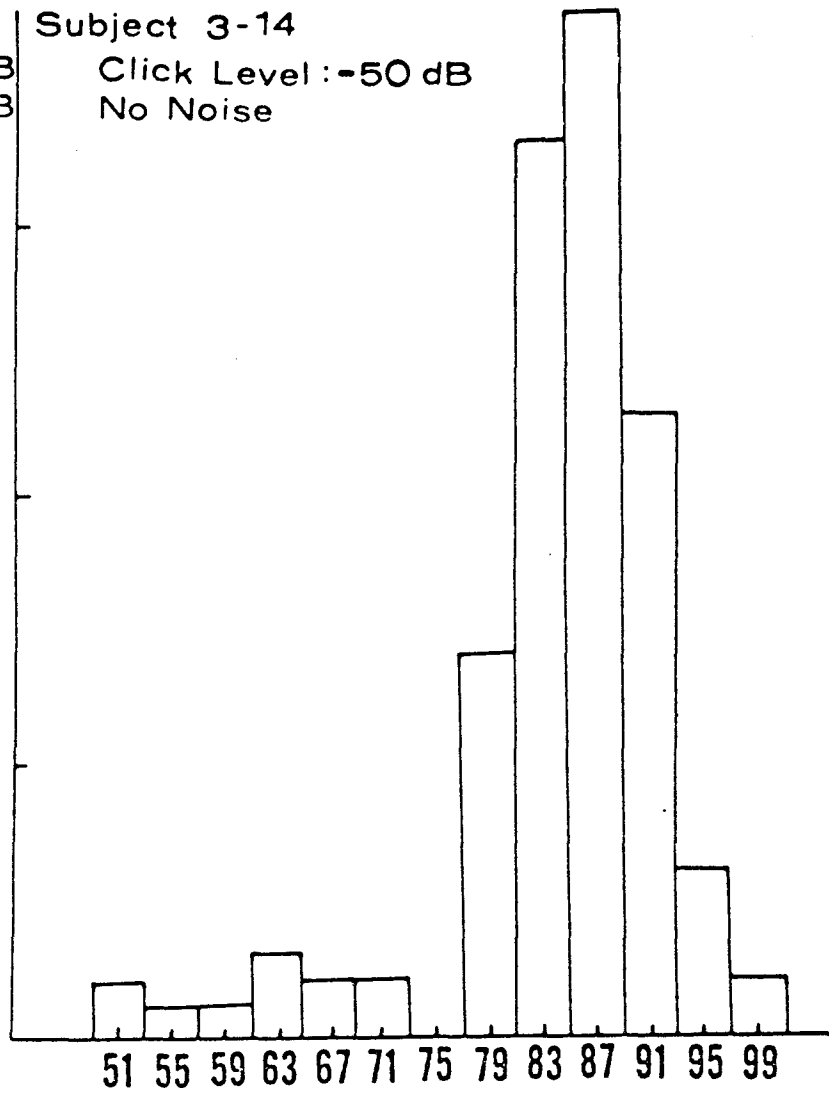
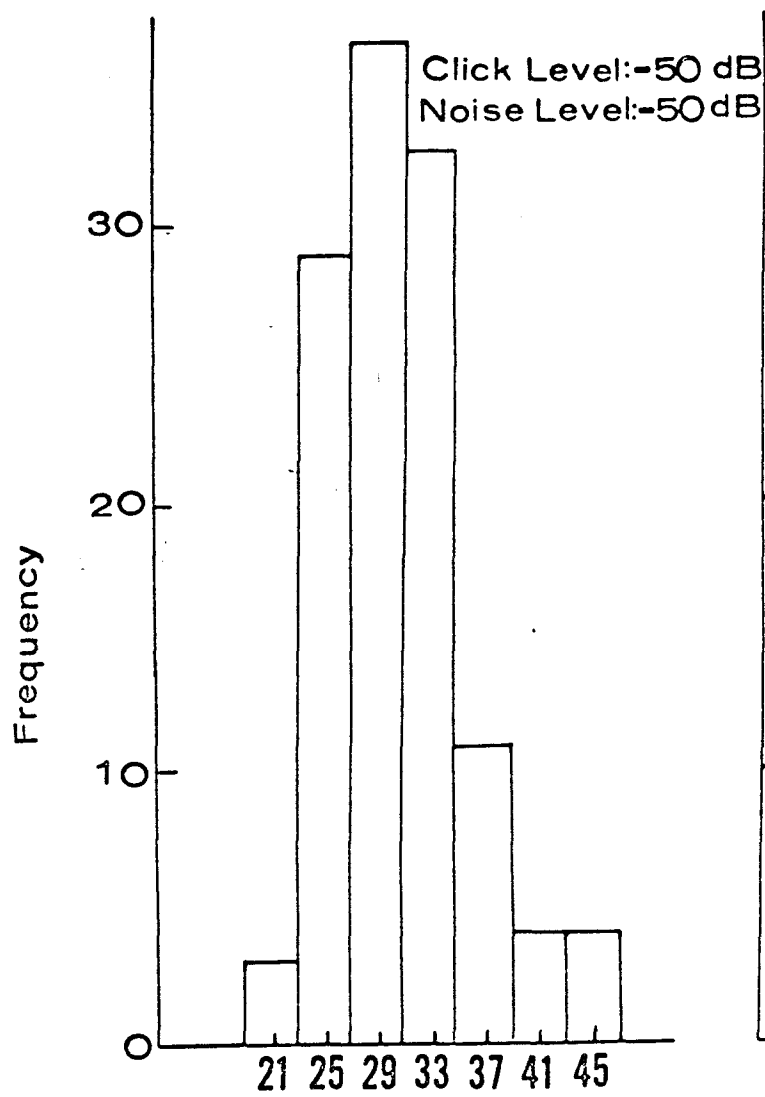


Figure 24

Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. For each histogram approximately 130 responses were measured. Subject 3-14.



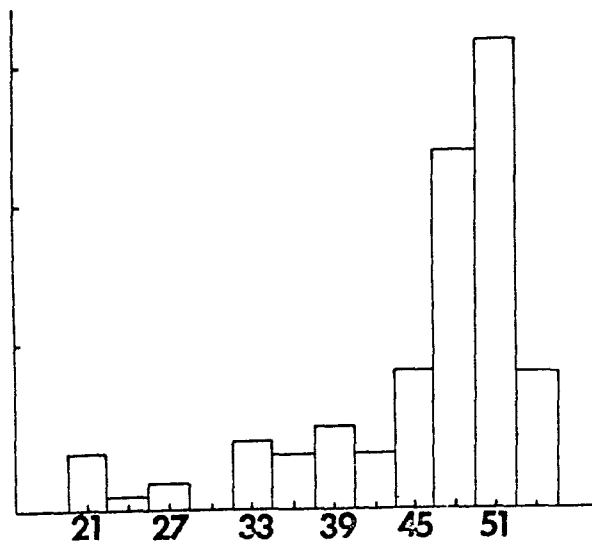
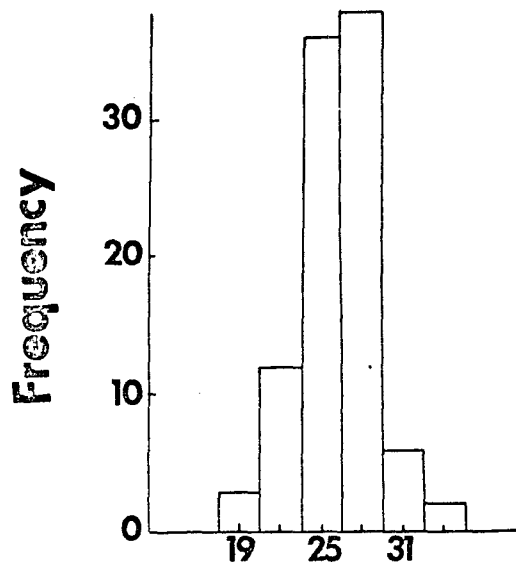
Amplitude of N_1 (arbitrary units)

Figure 25

Histograms of the peak-to-peak amplitude of the N_1 response with and without a noise background. For each histogram approximately 130 responses were measured. Subject 3-06.

Click Level:-40dB
Noise Level:-50dB

Subject 3-06
Click Level:-40dB
No Noise



Amplitude of N_1 in arbitrary units

means are one sigma apart ($d' = 1$), $P(c) = 76.0\%$. For a case of two markedly skewed distributions separated by one sigma, two chi-square (d.f. = 3) distributions were "overlapped" and the ROC curve was plotted from values obtained by moving a criterion along the abscissa (see Figure 26). The ROC curve was plotted on a 100 X 100 grid, and the area under the curve was approximated by counting boxes. The percentage of correct responses equaled 80.7%--a small increase from the value of 76.0% for the case of two normal distributions. It appears that discrimination is only slightly more accurate when the underlying distributions are markedly skewed.

As a check on the accuracy of this graphical method, two normal distributions were "overlapped" ($d' = 1$) and the resulting ROC curve was plotted. The graphical method yielded $P(c) = 75.85\%$; this agrees closely with the value of 76.02% obtained from tables of the normal distribution.

(c) Examination of the amplitude distributions plotted in Figures 22 through 25 reveals the effect of noise on N_1 variability. Specifically, variability of response decreases with the addition of noise. Sigma-functions obtained from four animals (1-31, 3-22, 4-08, and 4-26) are presented in Figures 27 and 28. It can be seen that the addition of a noise background decreased N_1 variability--the more so, the higher the noise level.

Special note should be taken of the similarity in form of the sigma-functions and the physiological Weber functions

Figure 26

Graphical computation of the percentage of correct responses in the two alternative-forced choice experiment for asymmetric distributions of the decision variable. (A) Two chi-square distributions (d.f. = 3) whose means are separated by one standard deviation unit. (B) Receiver operating characteristic curve (ROC) obtained by moving a criterion through the distributions of (A). The ordinate and abscissa are, respectively, the probabilities of correct detections and of "false alarms." The area under the ROC curve equals percentage of correct responses (see page 8.)

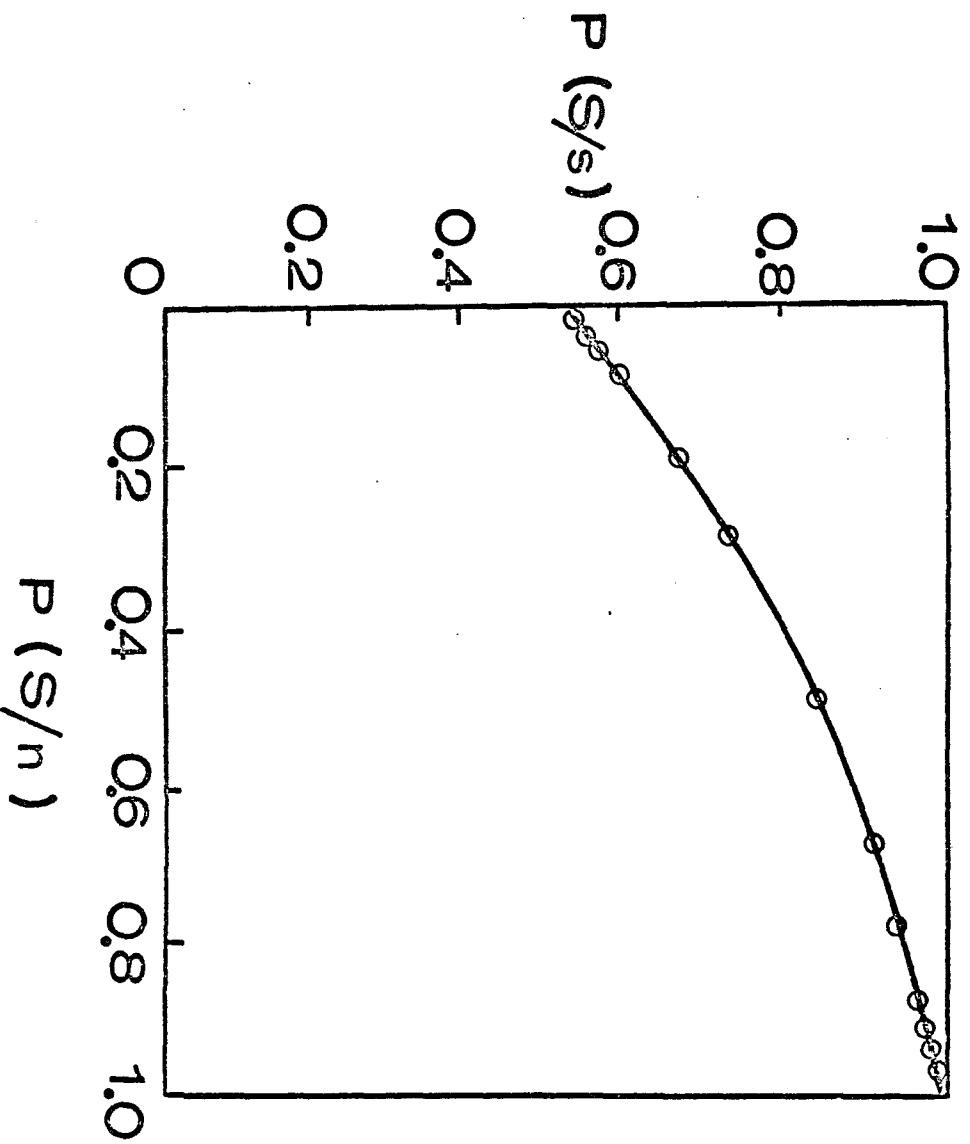
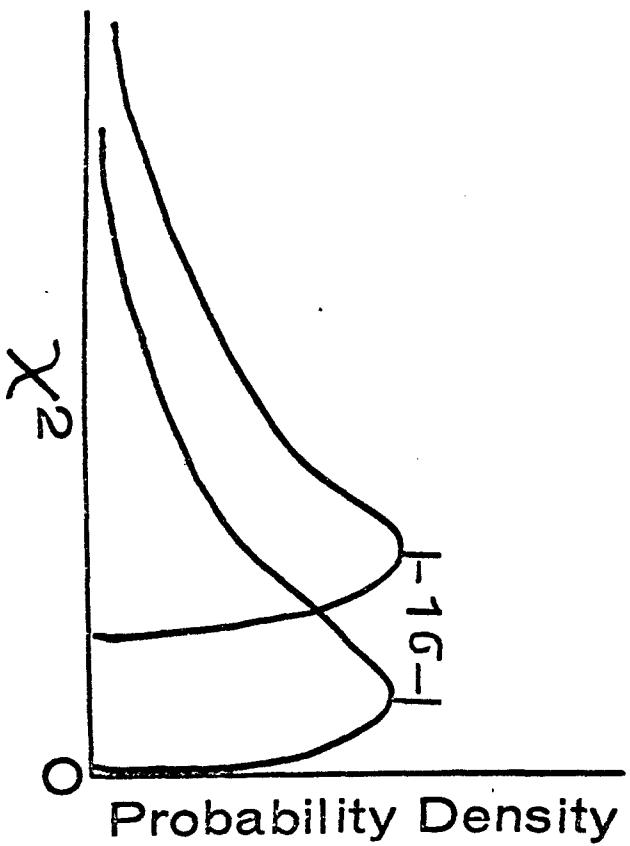


Figure 27

Standard deviation of N_1 amplitude as a function of click intensity with and without a noise background. Each data point is based upon approximately 150 responses to a click of fixed intensity. (A): Subject 1-31. (B): Subject 3-22.



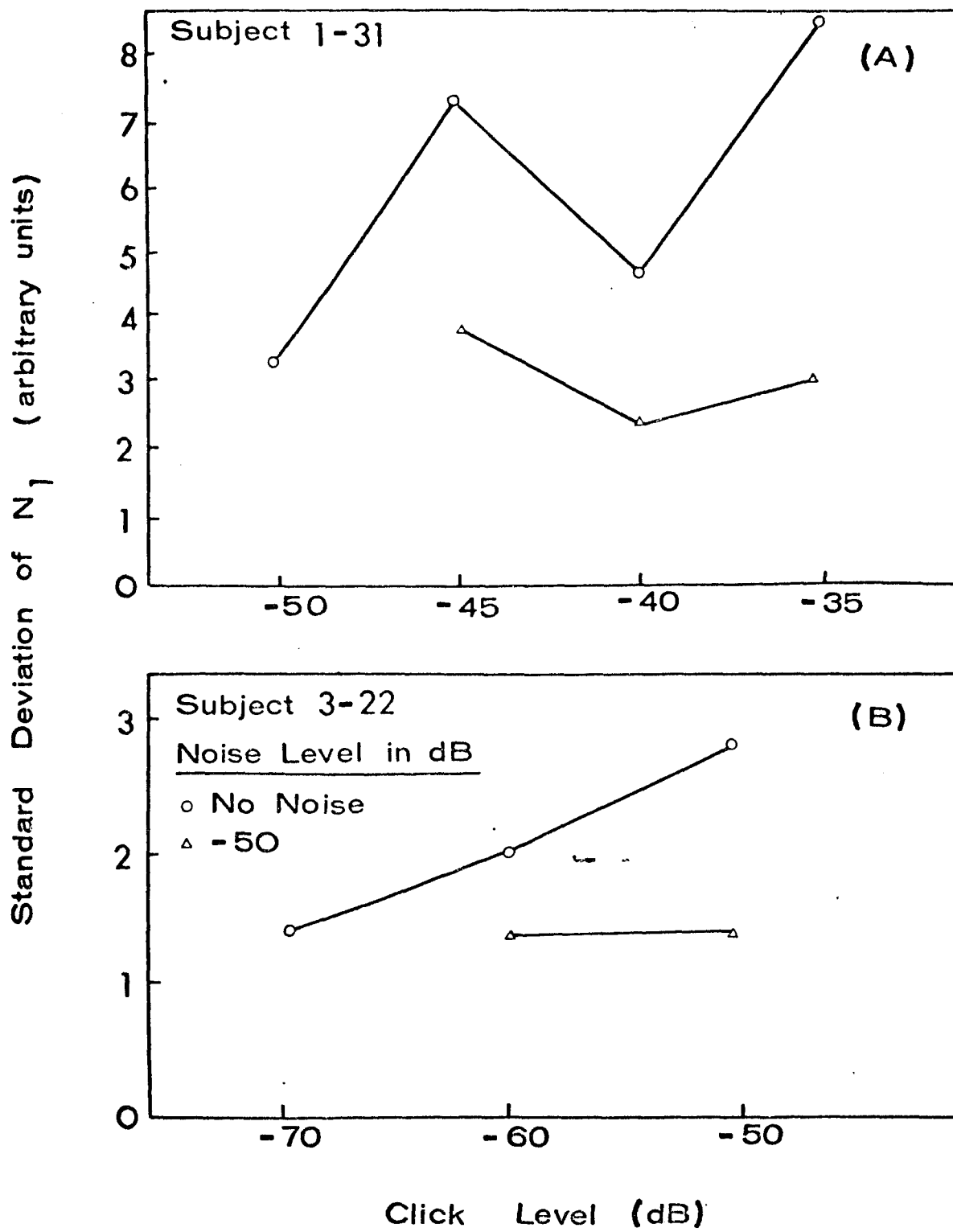
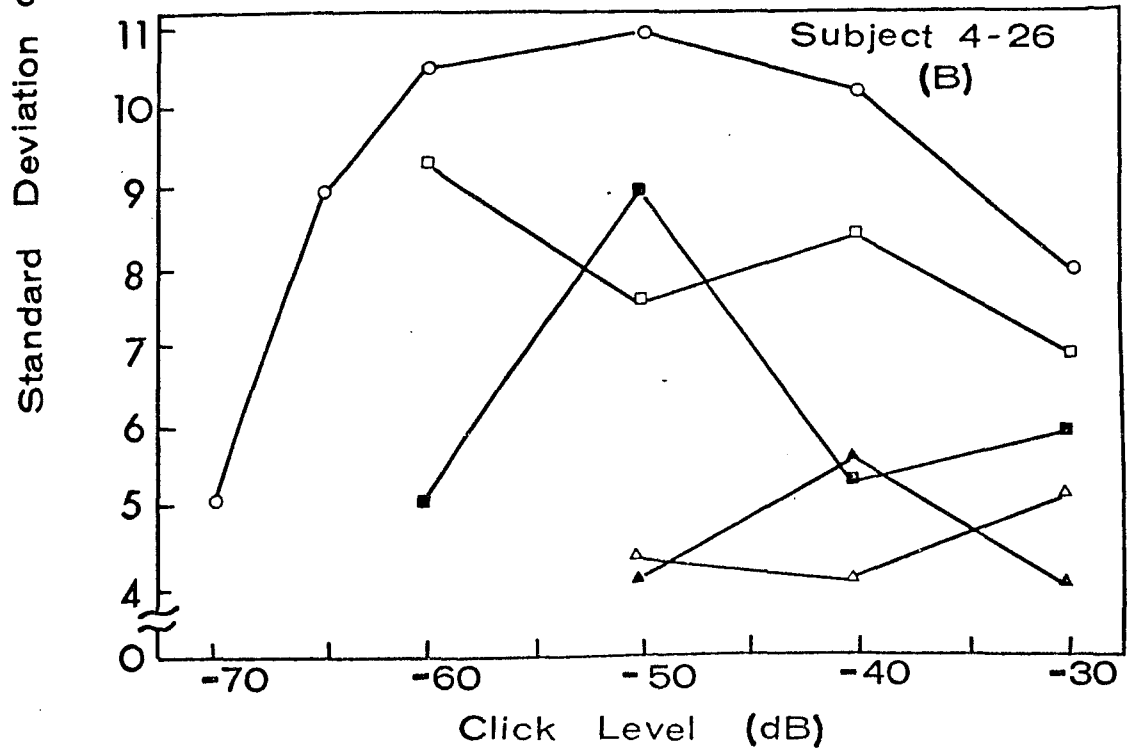
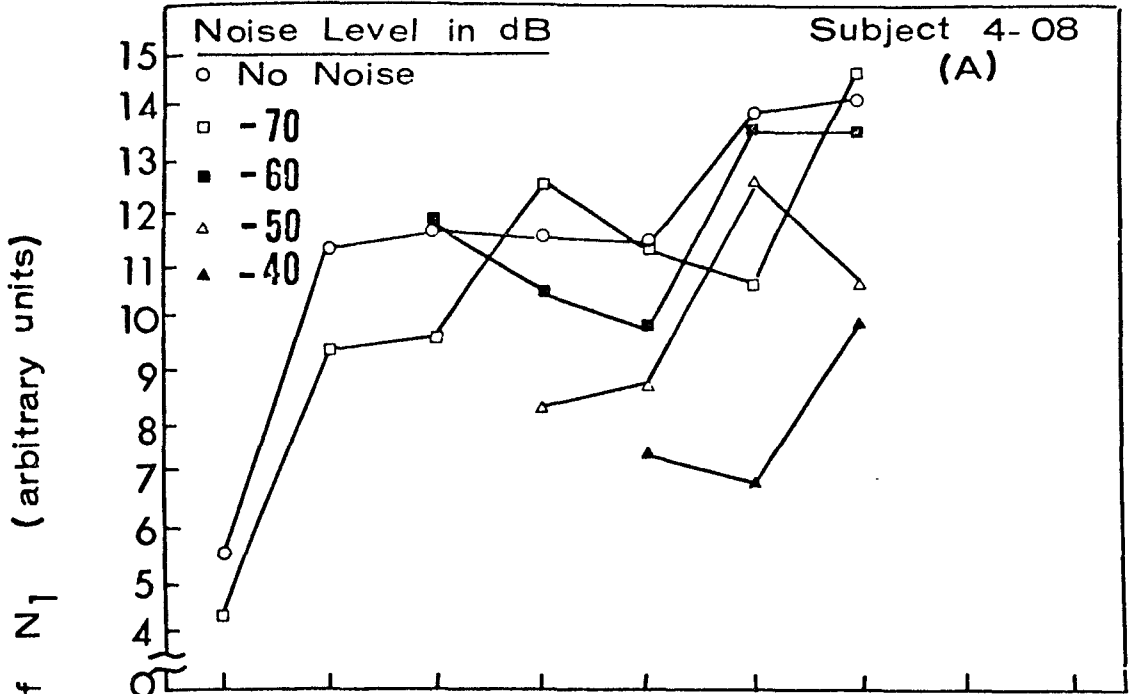


Figure 28

Standard deviation of N_1 amplitude as a function of click intensity with and without a noise background. Each data point is based upon approximately 40 responses to a click of fixed intensity. (A): Subject 4-08. (B): Subject 4-26.



presented in the preceding chapter. Adding noise decreases both N_1 variability and the physiological DL.

Quantitative analysis of the effects of noise can be made in the case of three animals. The analysis, which is presented in Table 2, is identical to that performed on page 90 to predict the effect of click intensity on the physiological DL. The masked Weber fraction is predicted from its unmasked value by appropriately decreasing the relative DL (in dB).

Two factors combine to contribute to the reduction of the DL. First, the effect of noise on N_1 variability is given in column (C) of the table. Second, the masked and unmasked intensity functions have slightly different slopes in the region of the DL (see column D of the table). This was evaluated by making measurements at intervals of 2 dB. (Measurements at 5 dB intervals were made in the case of animal 1-31.)

For each of the three animals the masked Weber fraction obtained by the on-line voting procedure is smaller than that predicted by analyzing the effects of noise on distributions of N_1 amplitude. These discrepancies probably arise from the fact that N_1 variability for masked clicks is actually smaller than the measured standard deviations. The cochlear microphonic of the background noise--although small--added differently to the N_1 deflection on each trial, contributing additional variance to the measurements of amplitude that were made by reading the filmed oscillograms. This additional variance, which affected on-line voting only very rarely, was assessed in a special

Table 2
 Prediction of the Weber Fraction in Noise from its Unmasked Value

Animal No. and Stimulus levels	(A)	No Noise	(B)	(C)	(D)	(E)	(F)	Noise	(G)	(H)
	$\Delta I/I$		$10 \log \frac{I + \Delta I}{I}$	$G_{\text{noise}} / G_{\text{no noise}}$	$\frac{\text{Slope noise}}{\text{Slope no noise}}$	(C) / (D)	(B) x (E)	$10 \log \frac{I + \Delta I}{I}$	$\Delta I/I$ (Predicted)	$\Delta I/I$ (Obtained)
1-31 Click: -40 dB Noise: -50 dB	1.24		3.50 dB	0.586	1.605	0.365	1.28 dB		0.34	0.18
3-06 Click: -40 dB Noise: -50 dB	1.63		4.20 dB	0.383	1.580	0.242	1.02 dB		0.26	0.23
3-14 Click: -50 dB Noise: -50 dB	0.55		1.90 dB	0.455	1.610	0.283	0.54 dB		0.13	0.07

experiment using animal 4-26. A Hewlett-Packard 3722A Pseudo-Random Noise generator was used to produce masking waveforms that were identical from trial to trial. The N_1 responses evoked by clicks synchronized to the "frozen" noise background were measured for four combinations of click and noise level. Standard deviations of amplitude were computed and found to be approximately two-thirds of the corresponding sigmas obtained with random noise (see Appendix B).

The reduced variability of N_1 amplitude in this experiment is a consequence both of the coherence of the microphonic waveform and the N_1 deflection and of the elimination of variations in stimulus energy. Although, the latter effect is probably the lesser, it cannot be evaluated quantitatively without knowledge of the effective stimulus duration.

It is clear from the foregoing that insofar as discrimination between click intensities is based on N_1 responses to these stimuli, the effect of masking noise on discrimination can now be understood. Introduction of a noise background diminishes N_1 amplitude, presumably by reducing the mean number of elements fired by the click. Of significance for differential sensitivity, however, is the fact that trial-to-trial variations in the number of excited elements is also reduced. This reduction in response variability occurs in the face of some increase in the variability of stimulus energy.

Although the continuous background is a noise whose addition to the click stimuli can only increase variability of acoustic

energy in the region of each click, it seems to function as a steady stimulus-- removing from the pool of potentially excitable elements a more or less constant number. That is to say, the fluctuations of stimulus energy are not accompanied by an increase in N_1 variability. We have already noted (see page 94) that noise shifts the intensity function downward about equally over the intensitive range. The effect of this "line-busy" phenomenon on N_1 variability is, however, difficult to specify quantitatively. We would need to know for all elements such factors as the time course of recovery (after having been fired by the noise) and the form and sigma of the threshold probability distribution at each instant during recovery. As Frishkopf (pp. 61-63) has already noted, a complete model of the "line busy" effect would have many free parameters. To the extent that these parameters are "free", the usefulness of the model is limited.

CONCLUDING REMARKS

Further comment should be made on the discrepancies between the physiological DLs obtained on line and those "predicted" from distributions of neural events. In studies such as this, in which electrophysiological events are related to psychophysical data, the physiological methods assume great importance. We have already noted (see pages 18 and 19) the sampling problem inherent in the comparison of behavioral Weber functions with functions derived from the firing patterns of

single nerve cells. This problem may be partially solved when a gross electrode is used to record compound action potentials.

A sampling problem of another sort arises when DLs are computed from distributions of neural effect. The percentage of correct discriminations between two stimulus intensities (I and $I + \Delta I$) can only be calculated if the two neural distributions are completely specified. Such specification may well require measurements of more neural responses than can be obtained during the course of one experiment on an acute preparation.

The two means are given by the intensity function, whose slope in the region between I and $I + \Delta I$ must be known. Interpolation in the transfer function between widely separated data points is fraught with danger. To resort to theory (see, e.g., Crozier) to define the intensity function is to beg the question.

The forms and standard deviations of the two distributions must also be estimated from measurements. The sigmas are obviously of critical importance, and we have examined (see page 104) one instance of the effect of skewness on discrimination. Again, resort may be made to theory in order to define the neural distributions (cf. McGill); again, however, the theory must be validated.

The on-line voting procedure employed in this thesis is simple, straightforward and distribution-free. On each trial, the two click-intensities were "discriminated" by means of their

N_1 responses. Physiological DLs were then obtained by a blocked-trials staircase procedure without reference to the parameters of the underlying neural distributions. "Validity" for the method is provided by the finding that physiological and psychophysical DLs were similarly affected by changing click intensity and by adding masking noise.

It now seems worthwhile to attempt trial-by-trial comparisons--in individual subjects--of N_1 potentials and discrimination responses. The techniques for implanting gross electrodes in the brains of trained animals are well established. Evoked responses are routinely recorded from the central nervous systems of responding subjects. Too often, however, the search for evoked-response correlates of discrimination and discrimination learning has proved fruitless. A more modest effort--with electrodes located in more peripheral structures--may well turn out to be more profitable.

Chapter VI

SUMMARY POINTS

Differential intensity sensitivity for acoustic clicks was the principal concern of this thesis. Weber functions were obtained from human observers using a blocked-trials variation of the two-interval-forced-choice procedure. In addition, a novel on-line procedure was employed to obtain "physiological DLs" based on the compound action potential of the guinea pig's auditory nerve (N_1).

(1) The form of the Weber function for clicks differs significantly from the Weber functions usually reported in hearing. Specifically, differential sensitivity for click intensity deteriorates markedly in the mid-range of intensities.

(2) This unusual result is also found in the DLs obtained from the guinea pig.

(3) Addition of a continuous background noise produces a second unusual result: differential sensitivity is improved, the more so, the greater is the intensity of the noise. So long as the click is audible in the presence of the masking noise, intensity discrimination is improved.

That discrimination between "statistic-less" pulses is improved by the addition of noise is unexpected. Indeed, theories of discrimination that are based on fluctuations of stimulus

energy ("ideal observer" theories) would predict exactly the opposite result.

(4) The paradoxical effect of masking noise was also found in physiological experiments.

The parallelism between the psychophysical and the electrophysiological results lead us to propose that the former are based on the latter. Accordingly a model of discrimination was proposed which is based on the parameters of the N_1 response. Experiments were then performed to determine (1) the slope of the intensity function, (2) the form of the neural amplitude distributions and (3) the variability of N_1 amplitude.

(5) It was found that the changes in differential sensitivity with click intensity are based only on changes in N_1 variability--the slope of the intensity function and the form of the amplitude distributions are more or less unaffected. Specifically, increased DLs as click intensity is increased from threshold were accompanied by increased variability of N_1 amplitudes.

(6) The addition of noise had several effects on the N_1 response. First, the slope of the intensity function is slightly increased. Second, neural amplitude distributions--which are markedly skewed in the absence of noise--are transformed into more or less symmetrical form. Third, variability of N_1 amplitude is diminished.

These three effects were evaluated quantitatively, and some advantages of treating gross electrode responses in on-line fashion were considered.

Appendix A

PEAK-TO-PEAK READER

The problems involved in on-line comparison of N_1 amplitudes have been discussed in Chapter III. The difficulties were resolved by using the peak-to-peak reader presented in Figure 29.

The output signal of the Grass P-5 physiological pre-amplifier was introduced into a power amplifier (Pilot SA-260). The power amplifier provided a gain of 30 times; the large voltage swings that resulted were important in minimizing the small non-linearities in the characteristic of the silicon diode. In addition, the power amplifier served as a low-impedance generator (source impedance = one ohm) to drive the diode-capacitor combination.

The first component of the N_1 response at the output of the power amplifier is negative-going. This is passed by the diode whose cathode is connected to the source. The diode continues to conduct, negatively charging the $0.1 \mu\text{F}$ capacitor until the waveform begins to go positive, point C of Figure 30A. At this moment the diode disconnects and the voltage across the capacitor equals the peak negative voltage at point C.

Until the waveform reaches point C, the signals at points 1 and 2, which are the inputs to the differential amplifier

Figure 29

Schematic diagram of the peak-to-peak reader. Points 1 and 2 are the differential inputs to the oscilloscope.

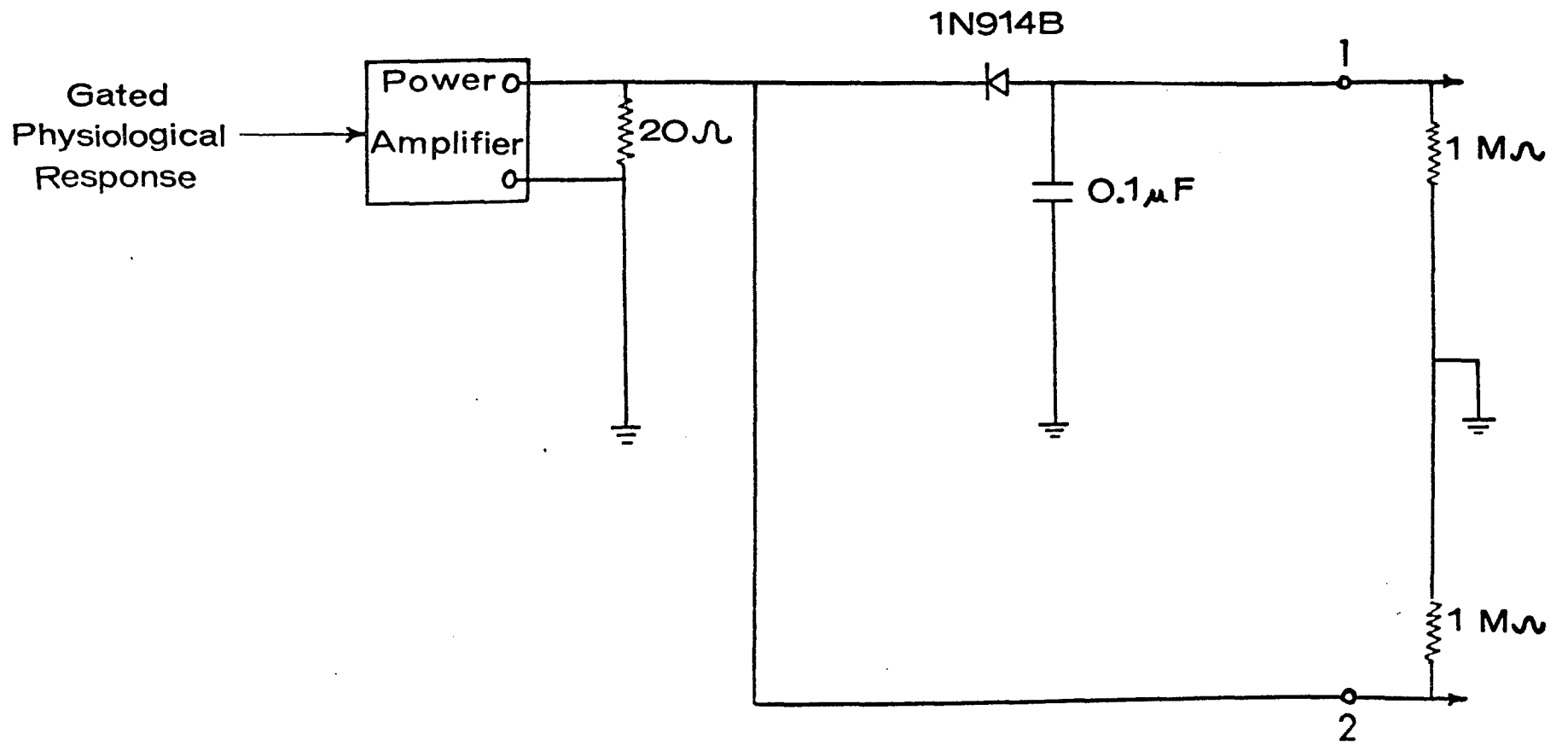
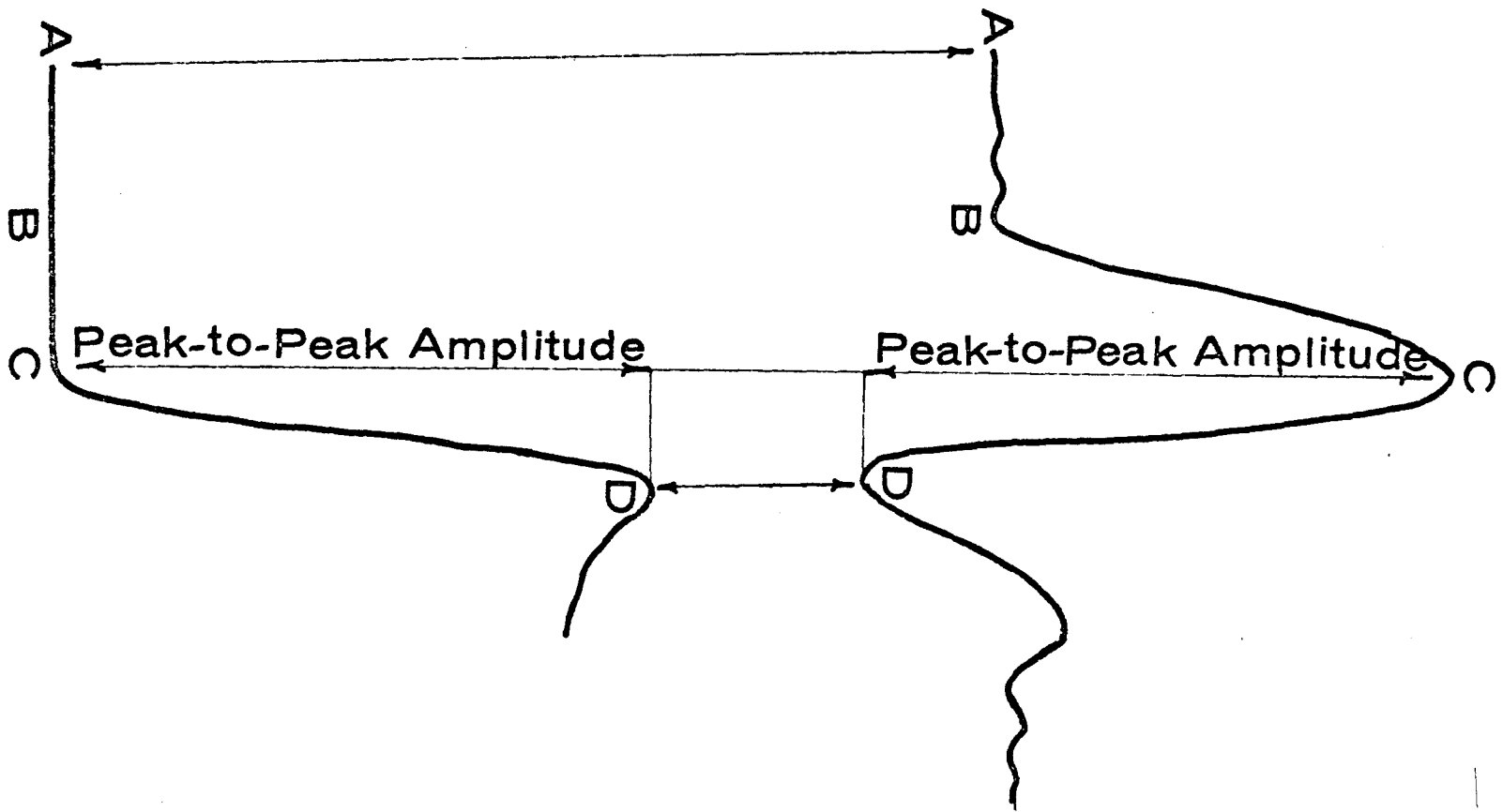


Figure 30

- (A) Schematic N_1 response, indicating peak-to-peak amplitude. Negativity at the round window electrode and at the output of the power amplifier is plotted as an upward deflection.
- (B) Readout of the peak-to-peak amplitude.



of an oscilloscope (Tektronix 502A), are exactly the same. (The diode has a negligible forward resistance.) The oscilloscope trace, which displays the output of the peak-to-peak reader, is at zero volts (see Figure 30B). After the N_1 waveform starts to go positive, the signals presented to the input terminals of the differential amplifier are no longer identical. Input 1 reads the voltage imposed on the capacitor (equal to the peak negative voltage of N_1) while input 2 continues to read the N_1 waveform which is now positive-going. The output waveform of the peak-to-peak reader is then a function of the moment-to-moment difference between the voltage at 1 and at 2. The amplitude of the displayed waveform (Figure 30B) equals the peak-to-peak amplitude of the N_1 waveform (Figure 30A).

On each trial, the two readout deflections (from the zero-volt baseline) were compared. If the greater deflection was evoked by the more intense click stimulus, a correct "neural vote" was recorded.

The value of capacitance ($0.1 \mu\text{F}$) employed in the peak-to-peak reader was chosen to satisfy several considerations.

(a) The charging time of the capacitor must be short compared with the rise-time of the N_1 response. The charging time-constant is the product of the internal impedance of the power amplifier and the $0.1 \mu\text{F}$ capacitor. Since the source impedance of the amplifier equals one ohm, the charging time-constant equals 10^{-7} sec, which is considerably less than the rise-time of the N_1 potential. The latter is approximately 5×10^{-4} sec.

(b) The discharge time of the capacitor must satisfy two considerations. First in order that the capacitor lose negligible charge during the positive-going deflection, the discharge time-constant must be large relative to the time between the negative and positive peaks (C and D). Second, the capacitor is to be completely discharged by the end of the one-second interval separating click responses. Since the capacitor is connected across the 1 M input resistance of the oscilloscope, the discharge time-constant equals 0.1 sec. This is 200 times the interval from the negative to the positive peak; at the same time, the one-second interval between responses equals ten time-constants.

It was necessary to gate the input to the peak-to-peak reader to pass only the N_1 response. Otherwise, cardiac potentials whose amplitude is several times larger than the N_1 response, would have saturated the circuit. Gating was accomplished by means of a mercury-wetted relay connected at the input to the power amplifier and pulsed to open a 10-msec "time window" with each click stimulus. On rare occasions, irrelevant potentials coinciding with the acoustic response and passed, therefore, by the relay would contaminate the response. On those trials, no attempt was made to read the peak-to-peak display.

The output of the peak-to-peak reader was compared with the N_1 response by photographing both from the face of a dual-beam oscilloscope. Calibration runs of 150 responses were made at -50 dB and at -20 dB. A Grass kymograph camera was employed

to make permanent records. The peak-to-peak amplitudes and their corresponding readouts were measured and compared. The Pearson product-moment correlation for the run at -50 dB was .99; at -20 dB, $r = 0.96$.

Appendix B

CLICK RESPONSES MASKED BY PSEUDO-RANDOM NOISE

A Hewlett-Packard 3722A Noise Generator was used to produce masking waveforms that were identical from trial to trial. An external time base (frequency = 131071 Hz) was used to supply clock pulses to the noise generator. With the "sequence length" switch set at 131071, noise waveforms lasting one second and repeating once each second were generated. The Pseudo-Random Noise Generator replaced the Grason-Stadler Generator in the stimulating system (see Figure 4).

The stimulus click was always presented at the same point of the noise waveform. This was accomplished by means of the synchronizing trigger produced by the noise generator.

For each combination of click and noise level, approximately 35 responses were photographed and measured. Standard deviations for "frozen" and random noises are presented in the table below. Random noise was obtained from the same generator by setting the "sequence length" switch to "infinite".

Table 3

Variability of N_1 Amplitude for Clicks Evoked by
Random Noise and by Pseudo-Random Noise. (Animal 4-26).

Stimulus Condition				
Click Level (dB); Noise Level (dB)	-50; -50	-40; -50	-50; -40	-40; -40
Random Noise	11.38	7.29	7.79	6.68
Pseudo-Random Noise	5.65	5.13	6.04	4.33

Appendix C

Difference limens ($\Delta I/I$) for click intensity with and without a continuous noise background. Three to four limens were obtained for each combination of click and noise level.

Table 4: Subject A.I.

Table 5: Subject H.T.

Table 4

(Subject A.I.)

Click Level (dB SL) Noise Level (dB SPL)	10	20	30	40	50	60	70	80	88
	No Noise	0.51 0.69 0.55	0.38 0.35 0.38	0.95 1.08 1.00	1.13 1.39 0.95	0.90 0.82 0.90	0.70 0.55 0.82	0.78 0.38 0.59 0.59	0.29 0.26 0.23
15	No Data	0.51 0.44 0.44	0.94 0.69 0.70	0.62 0.80 0.95	0.70 0.64 0.70	No Data	No Data	No Data	No Data
25	<u>15 dB SL</u> 0.54 0.51 0.66	0.29 0.38 0.39	0.73 0.54 0.73	0.62 0.90 0.70	0.82 0.62 0.54	0.70 0.59 0.62	0.62 0.48 0.54	0.20 0.28 0.32 0.28	0.35 0.28 0.38
35		<u>25 dB SL</u> 0.38 0.51 0.45	0.45 0.44 0.35	0.55 0.48 0.45	0.70 0.62 0.74	0.46 0.74 0.62	0.55 0.56 0.62	0.32 0.26 0.35	0.41 0.32 0.29
50				0.48 0.38 0.39	0.38 0.38 0.38	0.55 0.62 0.59	0.48 0.51 0.55	0.39 0.32 0.38	0.44 0.45 0.32
65					0.20 0.26 0.26	0.17 0.23 0.22	0.29 0.28 0.32	0.27 0.20 0.25	0.51 0.48 0.36

Table 5

(Subject H.T.)

Click Level (dB SL) Noise Level (db SPL)	10	20	30	40	50	60	70	80	88
	No Noise	0.38 0.38 0.47	0.38 0.35 0.29	1.29 0.95 1.09	1.13 1.00 1.57	0.91 1.00 0.75	0.78 0.78 0.70	0.55 0.70 0.70	0.28 0.28 0.23
15	No Data	0.32 0.38 0.35	0.72 0.59 0.64	0.78 0.91 0.86	0.78 0.91 0.95	No Data	No Data	No Data	No Data
25	<u>15 dB SL</u>	0.26 0.38 0.38	0.62 0.74 0.45	0.82 0.73 1.23 0.95	0.59 0.95 0.95	0.82 0.79 0.73	0.66 0.59 0.73	0.28 0.23 0.29	0.28 0.41 0.38
		<u>25 dB SL</u>	0.55 0.62 0.47	0.35 0.38 0.38	0.54 0.48 0.44	0.73 0.66 0.66	0.86 0.95 0.51	0.26 0.23 0.28	0.41 0.38 0.35
				0.47 0.51 0.62	0.29 0.45 0.54 0.41	0.90 0.54 0.54	0.48 0.41 0.32	0.31 0.20 0.23	0.32 0.38 0.41
65					<u>55 dB SL</u>	60 65 dB SL			
					0.44 0.38 0.51	0.35 0.62 0.38 0.55 0.41 0.54	0.29 0.20 0.23	0.23 0.20 0.22	0.32 0.28 0.45

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AUTOBIOGRAPHICAL STATEMENT

The author received a Bachelor of Arts degree from Brooklyn College in 1963. He entered the graduate program in Psychology at Lehigh University that same year. While at Lehigh University the author was awarded a graduate teaching fellowship. In his second year of graduate study he was awarded a research fellowship at the Pratt Psychological Laboratory. During that year he was elected to Psi Chi, the national honor society in psychology. He received the Master of Science degree in June of 1965.

In September, 1965 the author entered the doctoral program at The City University of New York (Brooklyn College). During his term at Brooklyn College he was awarded a graduate assistantship for research in the Psychoacoustic Laboratory under the direction of Dr. David Raab.

In 1967, the author was elected to associate membership in the Society of Sigma Xi.

While at Brooklyn College the author received an appointment as lecturer in the School of General Studies.

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