

On the relations of intensity jnd's to loudness and neural noise^{a)}

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It is shown experimentally that, in contradiction of the fundamental concept of Fechner's law, the intensity jnd for auditory sinusoidal signals follows loudness, rather than its derivative with respect to sound intensity. The evidence is obtained by comparing the jnd's of a population with normal hearing to those of a population with hearing loss accompanied by loudness recruitment. Although the recruitment increases the slope of the loudness function, the jnd's of both populations were found to be practically equal when the loudnesses were equal. The phenomenon is accounted for mathematically by assuming that psychophysically relevant neural noise depends not only on the magnitude of loudness, but also on its derivative with respect to sound intensity. A related derivation accounts for the near miss to Weber's law.

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INTRODUCTION

The main purpose of this article is to describe a somewhat perplexing discovery made in the past but not yet published outside a Ph.D. dissertation (Jordan, 1962), according to which the intensity jnd's for sinusoidal signals are closely correlated with loudness, but are practically independent of its derivative with respect to sound intensity. The finding contradicts the fundamental assumption of Fechner's law and may appear paradoxical. According to the assumption, the relative jnd of sound intensity, the Weber fraction, is associated with a constant loudness increment, $\Delta L = k \Delta I / I$. The steeper the loudness function, the smaller should be the Weber fraction for a constant increment. The conclusion even transcends Fechner's law and applies to the now more acceptable Steven's power law. The difference form of this law is $\Delta L / L = \theta \Delta I / I$, where θ is the exponent of the power function. Obviously, for given ΔL and L , $\Delta I / I$ decreases as both θ and the slope of the loudness function increase. But our experimental results show unambiguously that $\Delta I / I$ is not correlated with the slope of the loudness function.

We performed jnd measurements on two populations of listeners, one with normal hearing and one with predominantly monaural hearing loss. The hearing loss was of sensorineural origin and was accompanied by loudness recruitment, in other words, a steepening of the loudness function. In spite of the slope difference, the jnd's were equal when the loudnesses were equal, except perhaps at the lowest SL's where the greater slope was associated paradoxically with a larger jnd. Loudness equality was achieved by the reliable procedure of loudness matching between the normal and pathological ears.

Jordan's dissertation was an outgrowth of the Lüscher-Zwislöcki (1948a,b) method for clinical detection of loudness recruitment by means of measurements of intensity jnd's and had as one of its purposes a formal validation of the method. As a related subject, "Zwislöcki's hypothesis was tested that two intensities producing equal sensations of loudness yield jnd values of equal magnitude." The hypothesis arose from clinical experience with the method.

For various reasons the dissertation was not summarized in a journal article until now. The present article, based on the data of the dissertation and some of its conclusions, has been prompted by recent confirmatory work of Hellman *et al.* (1985). Although, mainly for clinical reasons, the data were obtained with a modified psychophysical method of limits, which now must be considered antiquated, their agreement with contemporary results justifies their use. In particular, they clearly reflect the now well-established near miss to Weber's law, extending its validity to a population with hearing loss. In extension of the dissertation, both phenomena—the correlation of the jnd with loudness, not its gradient, and the near miss—are accounted for mathematically in the discussion section.

I. METHOD

The experiments were performed in an IAC booth with an ambient noise of 32 dB on the *A* scale. Subjectively, and according to a critical-band analysis, the noise was inaudible with test earphones in place.

The earphones consisted of TDH-39 transducers mounted in Grason-Stadler semiplastic, circumaural cushions. The sound-pressure outputs of the right and left earphones were balanced within ± 1 dB. The transfer functions were flat within ± 4 dB up to about 6 kHz, with a drop of 17 dB at 8 kHz.

^{a)} Presented in part at the 1985 Spring meeting of the Acoustical Society of America [J. Acoust. Soc. Am. Suppl. 1 77, S64 (1985)].

In the jnd experiments, a special modulator modulated the test tones, providing trapezoidally shaped increments with rounded corners. The onset and offset times were set at 50 ms, and the fundamental modulation frequency was 2.5 Hz. According to past experience, this modulation frequency produces about the lowest jnd's (Riesz, 1928; Lüscher and Zwislöcki, 1948a,b). Maximum modulation was 6 dB in terms of the jnd definition, $10 \log(1 + \Delta I/I)$. Below 2 dB, it could be varied in steps of 0.2 dB. Either the modulation or the test tone could be interrupted with a double-throw, manual switch without producing audible transients. The test tones were presented monaurally. In listeners with a strong monaural hearing loss, a narrow-band noise of 300-Hz bandwidth centered on the test-tone frequency masked the better ear, while the poorer ear was tested.

The jnd's were determined at the sound frequencies of 0.25, 0.5, 1.0, 2.0, 4.0, and 8.0 kHz in normal ears and at the frequencies of 0.25, 1.0, and 4.0 kHz in pathological ears. The order in which the frequencies appeared was randomized.

For several reasons, including a large number of parameter values, applicability of the same psychophysical method to jnd and loudness-level measurements, the use of naive subjects, and clinical implications, a modified method of limits was used. It consisted of three descending and three ascending series. The mean of all the series was accepted as the threshold value. The subjects signaled detection of modulation by means of a key activating a signal light. When low levels of modulation still elicited positive responses, the modulation was interrupted several times to ascertain if the subjects responded to the disappearance of modulation. In general, the experimenter manipulated the modulation interrupter so that the subjects compared the modulated signal to the nonmodulated signal rather than different degrees of modulation. Prior to test runs, a few test trials were administered at a sensation level of 40 dB and a sound frequency different from any of the frequencies used in subsequent measurements. These trials had the purpose of stabilizing the subjects' responses.

To obtain jnd measures in ears with hearing loss at the same loudness levels as in normal ears, dichotic loudness matches were performed. The sensation level in the good ear was preset at one of the levels used in jnd determinations on the subjects with normal hearing, and the sound intensity in the pathological ear was varied in descending and ascending sequences according to the method of limits. Five descending and five ascending sequences were administered, and the means of all sequences calculated for every subject. The jnd's in the pathological ears were determined at these mean sensation levels in the same way as in the normal ears.

Prior to jnd and loudness-level measurements thresholds of detectability in quiet were determined by means of the same method of limits, and three descending and three ascending series. This was necessary to ascertain if the subjects used in the determination of normal jnd's had normal hearing and to find out which sound frequencies in any given subject with predominantly monaural hearing loss were suitable for measurement of abnormal jnd's.

The population with normal hearing consisted of 26 col-

TABLE I. Means, medians, and standard deviations of the jnd's for the normal group at the indicated frequencies and sensation levels. All measurements are in decibels: $\text{jnd} = 10 \log [(\Delta I + I)/I]$.

Sensation level	Measures	Frequency in cps					
		250	500	1000	2000	4000	8000
10 dB	Mean	2.00	1.86	1.70	1.45	1.18	1.81
	Median	1.99	1.64	1.61	1.44	1.22	1.63
	σ	0.309	0.359	0.295	0.189	0.225	0.352
20 dB	Mean	1.48	1.41	1.28	1.12	0.954	1.33
	Median	1.43	1.40	1.25	1.17	0.986	1.38
	σ	0.241	0.194	0.181	0.194	0.184	0.189
40 dB	Mean	0.915	0.877	0.862	0.738	0.677	0.862
	Median	0.850	0.828	0.826	0.768	0.640	0.820
	σ	0.187	0.148	0.141	0.158	0.167	0.194
60 dB	Mean	0.600	0.558	0.512	0.454	0.354	0.531
	Median	0.596	0.581	0.558	0.419	0.342	0.568
	σ	0.148	0.141	0.138	0.109	0.089	0.161
80 dB	Mean	0.300	0.315	0.288	0.254	0.227	0.288
	Median	0.300	0.314	0.290	0.263	0.220	0.286
	σ	0.063	0.067	0.063	0.054	0.052	0.077
90 dB	Mean	...	0.250	0.238	0.180	0.212	
	Median	...	0.250	0.230	0.188	0.207	
	σ	...	0.044	0.063	0.031	0.051	

lege students with a median age of 21.5 years and a range from 19 to 27 years. They all had hearing levels equal to or better than 5 dB relative to the old audiometric zero (ASA, 1951) at octave frequencies from 250 to 8000 Hz. The population with abnormal hearing consisted of 14 subjects, aged 20 to 73 years with a median of 42 years. They had predominantly monaural, sensorineural hearing loss that was accompanied by loudness recruitment. They satisfied the following criteria at the test frequencies: hearing levels no greater than 10 dB in the better ear and between 30 and 75 dB in the worse ear. Only six of the 14 subjects satisfied the criteria at all test frequencies. The remaining eight satisfied

TABLE II. Means, medians, and standard deviations of the jnd's for the pathological group at the indicated frequencies and loudness levels. All measurements are in decibels: $\text{jnd} = 10 \log [(\Delta I + I)/I]$.

Loudness level	Measures	Frequency in cps		
		250	1000	4000
10 dB	Mean	2.600	2.430	1.630
	Median	2.500	2.480	1.250
	σ	0.583	0.440	0.344
20 dB	Mean	1.500	1.44	0.87
	Median	1.57	1.425	0.825
	σ	0.134	0.149	0.170
40 dB	Mean	1.02	0.82	0.58
	Median	1.000	0.783	0.575
	σ	0.223	0.183	0.105
60 dB	Mean	0.600	0.47	0.41
	Median	0.616	0.45	0.356
	σ	0.126	0.078	0.071
80 dB	Mean	0.33	0.26	0.29
	Median	0.33	0.266	0.30
	σ	0.070	0.054	0.083
90 dB	Mean	...	0.22	0.23
	Median	...	0.212	0.23
	σ	...	0.044	0.071

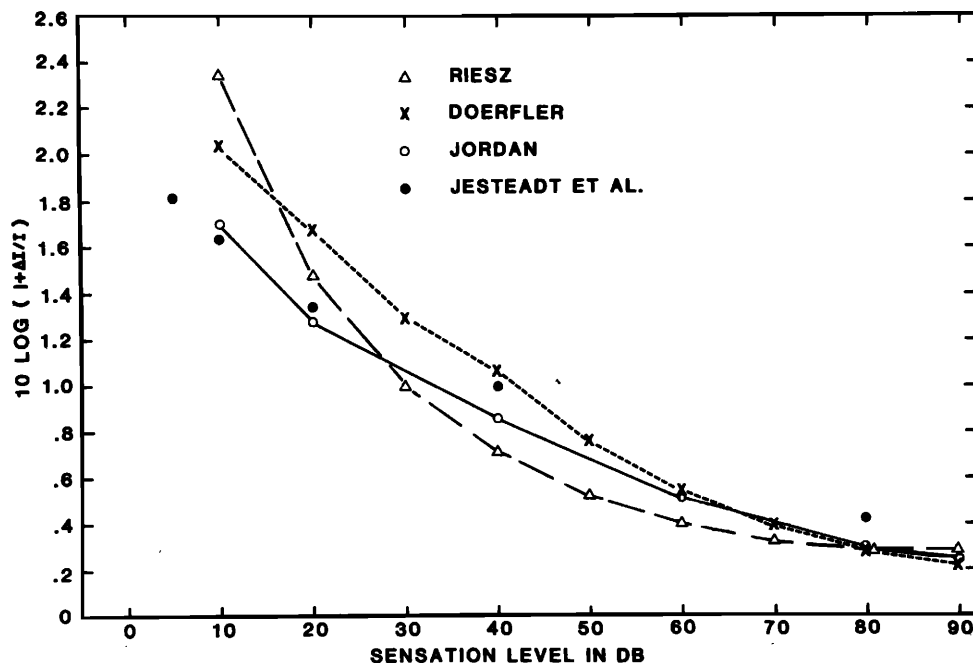


FIG. 1. The jnd as a function of sensation level at 1 kHz. Mean data from four studies. In the measurements of Riesz, Doerfler, and Jordan, amplitude modulation was used; in those of Jesteadt *et al.*, tone bursts.

them on some but not all of the frequencies. The audiograms of these subjects could be combined, however, so as to obtain four complete frequency sets. As a consequence, 10 rather than 14 complete sets of jnd measurements in pathological ears were obtained.

II. RESULTS

The results of jnd measurements are summarized in Tables I and II and Figs. 1 to 4. They are expressed in terms of $10 \log(1 + \Delta I/I)$, where I means the pedestal intensity and ΔI the intensity increment. Table I contains all the means,

medians, and standard deviations for the normal population. Table II does the same for the pathological one. No jnd's were measured at 90 dB SL at 0.25 or 8 kHz because of equipment limitations. Because comparative jnd data on the pathological population were obtained only at 0.25, 1, and 4 kHz, the jnd's obtained at the additional frequencies of 0.5, 2, and 8 kHz on the normal population are excluded from further consideration. Also, since the means and medians are nearly equal, only the means are used in the figures and the computations that follow.

In Fig. 1, the jnd's determined at 1 kHz on the normal population are compared with corresponding results of three

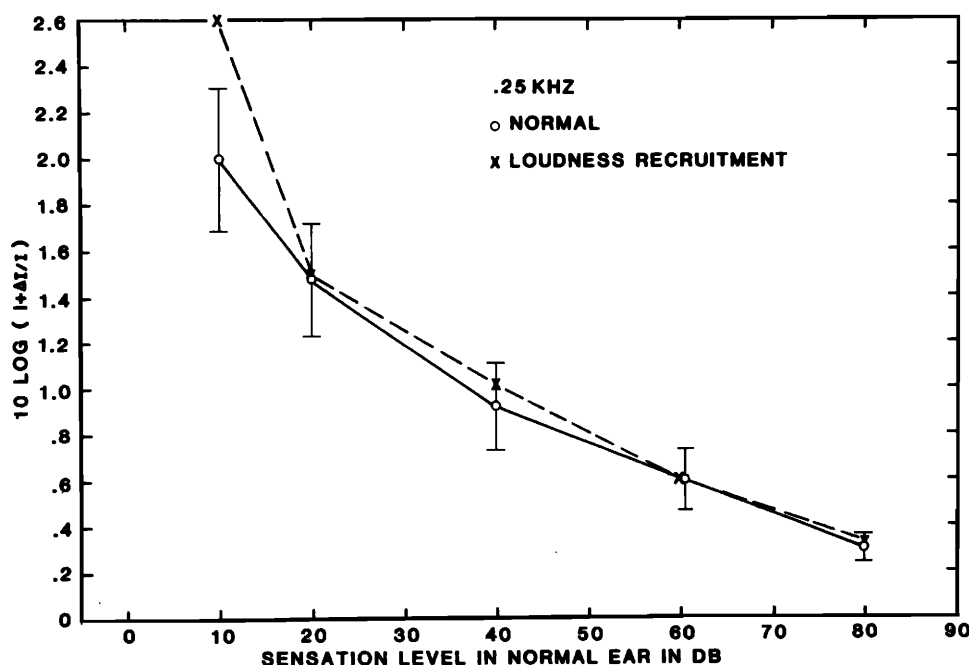


FIG. 2. The jnd as a function of sensation level in the normal ear at 0.25 kHz for two populations, one with normal hearing and the other with predominantly monaural hearing loss accompanied by loudness recruitment. Points plotted at the same sensation levels correspond to equal loudnesses.

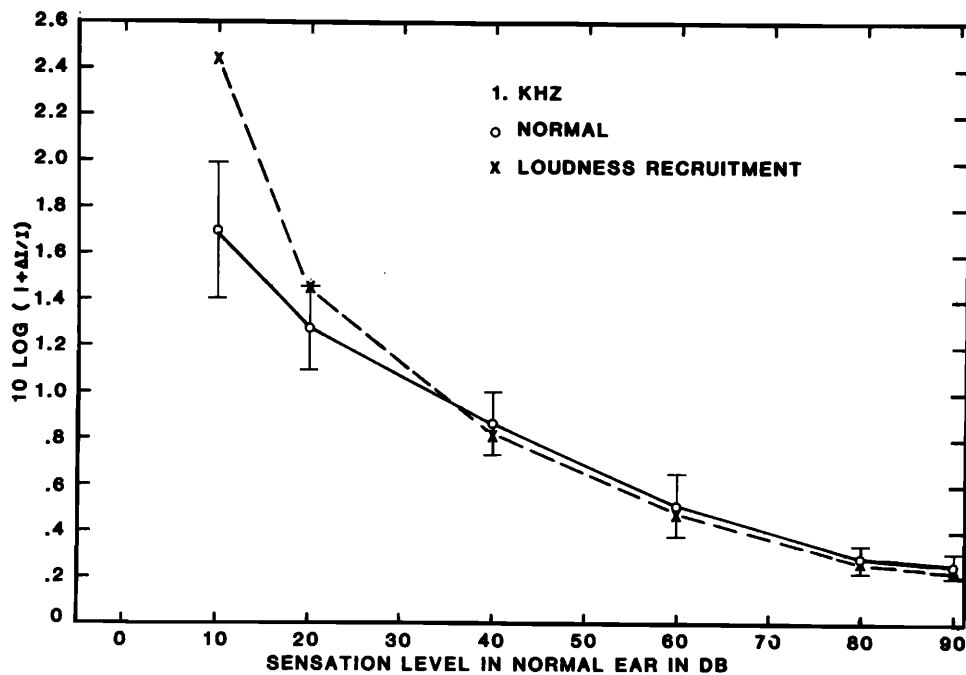


FIG. 3. Same as Fig. 2. for 1 kHz.

other studies. In two of these studies amplitude modulation was used, and the data are directly comparable with ours, although the modulation envelopes were nearly sinusoidal rather than trapezoidal. In the third study, that of Jesteadt *et al.* (1977), the jnd's were derived from discrimination of tone bursts of unequal intensity. The investigators used an adaptive two-interval forced-choice method. It is of some interest that our data for the frequency of 1 kHz agree best with those of Jesteadt *et al.*, especially at low SL's, in spite of the methodological differences. The main difference in the data lies in the rate with which the jnd's decrease as SL is

increased. However, the data of Jesteadt *et al.* trace probably the flattest jnd curve published for sinusoidal stimuli. The somewhat less extensive results obtained by Schacknow and Raab (1973) and Penner *et al.* (1974) are more in line with ours, although their stimulus paradigm was more like that of Jesteadt *et al.* These comparisons would seem to indicate that the amplitude modulation produces the same jnd results as discrimination of tone bursts, at least at 1 kHz. A similar conclusion was reached very recently by Long and Cullen (1985). However, our data deviate more from those of Jesteadt *et al.* at 0.25 and 4 kHz, indicating a greater frequency

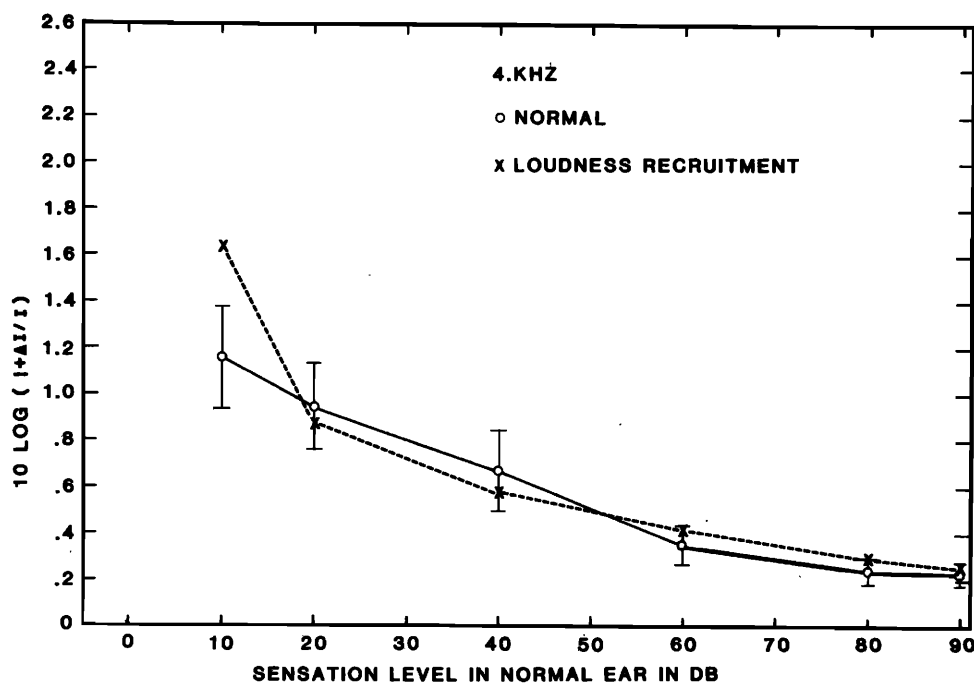


FIG. 4. Same as Fig. 2 for 4 kHz.

dependence. Similar differences have been found by others (e.g., Harris, 1963; Fastl and Schorn, 1981). The differences between the jnd's obtained by means of modulation and tone-burst methods become most apparent in the presence of auditory pathology, as has been shown already by Lüscher and Zwislöcki (1951) and later by Fastl and Schorn (1981). Because of these differences, our results reproduced in Figs. 2 to 4 may apply only to the modulation method. Nevertheless, the similarity of the near miss to Weber's law between our results and those obtained by others with the tone-burst method has induced us to apply to the phenomenon a mathematical analysis resembling that of McGill and Goldberg (1968) and Jesteadt *et al.* (1977). Their derivations were applied to the latter method.

The jnd values obtained by Doerfler (1948) are somewhat higher than ours except at the highest two sensation levels. They follow a similar although somewhat steeper course along the intensity axis. Riesz's data follow a curve with more curvature than do the remaining entries in Fig. 1. They have the highest values at 10 dB SL and the lowest in the mid-SL range. At 80 and 90 dB, all the modulation data are nearly identical. It is difficult to explain the deviant pattern of Riesz's results, since he used an amplitude envelope similar to that of Doerfler's.

The jnd's measured on the populations with normal and pathological hearing are compared in Figs. 2 to 4. They are plotted as functions of the sensation levels in normal ears. However, the pathological jnd's were not determined directly at these levels but rather at the pathological-ear levels that produced loudness equality with them. As a consequence, the normal and pathological jnd's plotted over the same abscissas correspond to equal loudnesses. The circles joined by solid lines indicate the normal jnd values; the crosses and the dashed lines indicate the pathological ones. The vertical lines show double the intersubject standard deviations of the normal jnd's. The standard deviations of the pathological jnd's were nearly the same.

The principal result evident in the figures is that the normal and pathological jnd's are approximately equal when the loudnesses are approximately equal, except perhaps at the lowest sensation levels. At these levels, the pathological jnd's appear to be somewhat larger than the normal ones. However, the deviation may be artifactual and due to slight loudness inequality resulting from a bias in loudness matching. At the very low sensation levels, the jnd's depend critically on loudness, as is evident in Figs. 2 to 4. Results suggesting an opposite bias were obtained previously in a small clinical study by Neuberger (1950) and led to ambiguous conclusions.

We should note here that, because the hearing loss varied among the listeners and test frequencies, the sensation-level ranges over which loudness recruitment took place differed. When the hearing loss amounted to only 30 dB, the loudness recruitment was practically completed at a 60-dB equivalent sensation level in the normal ear. A hearing loss of 75 dB extended the recruitment range to 90 dB. Clearly, all the subjects with hearing loss had steep loudness functions at the equivalent sensation levels of 10, 20, and 40 dB at all sound frequencies tested. Most had such functions at 60

dB, fewer at 80 dB. In spite of the steepened loudness functions in the whole or at least in one part of the population with hearing loss, its mean jnd's were approximately the same as in the subjects with normal hearing. The same was true for the statistical jnd distributions (Jordan, 1962), as reflected in practically equal standard deviations. If the jnd's depended on the slope of the loudness functions, the means would have to be lower and, at sensation levels at which only a part of the population exhibited loudness recruitment, the statistical distributions would have to be broader.

We should also note that loudness recruitment was practically completed in all subjects with hearing loss at the equivalent sensation level of 90 dB. As a consequence, not only the loudnesses but also the slopes of the loudness functions were practically the same as in the normal population. The equality of jnd's obtained under these conditions means that both groups used the same detection criterion.

Another result of potential significance is the relation between the size of the jnd and its intersubject standard deviation, evident in Figs. 2 to 4. As the jnd becomes smaller, so does the standard deviation. The functional relationship is illustrated in Fig. 5, where the standard deviation is plotted as a function of the mean jnd. Because of homogeneity of the variances involved, all the data obtained at the frequencies of 0.25, 1, and 4 kHz on the normal and pathological populations are pooled together. The data points appear to cluster around a straight line on the log coordinates of the figure, except for an apparent dip in the vicinity of 1.5 on the abscissa axis. This relation is further considered in the discussion section.

III. DISCUSSION

Our experimental results show unambiguously that the intensity jnd's for sinusoidal stimuli of the same frequency are constant when the loudnesses are constant, independent of the slopes of the loudness functions. The relationship ap-

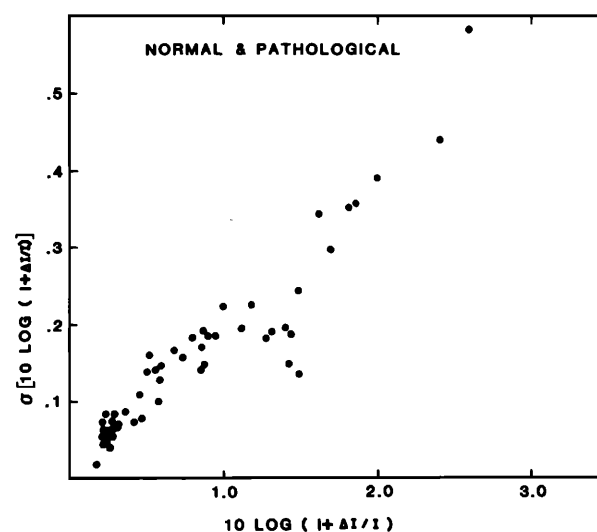


FIG. 5. Intersubject standard deviations of the measured jnd values as a function of the mean jnd values. The normal population and the population with hearing loss are pooled together.

pears to be robust since it is preserved even in the presence of cochlear pathology. Can it be explained within the framework of our present knowledge and concepts? We think that it can.

On the assumption that the primary perceptual correlate of an increment in sound intensity is a loudness increment, we have two pertinent relations at our disposal. One stems from the theory of signal detectability and is equivalent to the definition of d' ,

$$D = \Delta L / \sigma_L, \quad (1)$$

where D has the meaning of d' in the loudness domain, ΔL is the just detectable loudness increment, and σ_L , the standard deviation of the loudness variable. The other relation is an unspecified monotonic loudness function with sound intensity as the independent variable:

$$L = F(I). \quad (2)$$

The difference form of this equation is

$$\Delta L = F'(I) \Delta I. \quad (3)$$

Substituting for ΔL in Eq. (1) its expression of Eq. (3), we obtain

$$D = F'(I) \Delta I / \sigma_L \quad (4)$$

or, after rewriting,

$$\Delta I = D \sigma_L / F'(I). \quad (5)$$

Clearly, ΔI appears to be inversely proportional to the derivative, or slope, of the loudness function, as would be expected from Fechner's or Stevens's laws. However, if we assume that σ_L is directly proportional to the derivative, ΔI becomes independent of the derivative.

If

$$\sigma_L = B F'(I), \quad (6)$$

then

$$\Delta I = D B F'(I) / F'(I) = D B. \quad (7)$$

According to our mathematical derivation, the independence of the intensity and of the slope of the loudness function requires the standard deviation to be directly proportional to the derivative of the function. Whether this proves to be true, in fact, must remain for future experimental work to determine. Such proportionality would be plausible on the condition that σ_L originates in the auditory periphery, before the transformation $L = F(I)$ takes place. Under such conditions, $\Delta L = F'(I) \Delta I$ and $\sigma_L = F'(I) \sigma_I$, if σ_I , the standard deviation in the peripheral domain, is of the same order of magnitude as ΔI . This seems to be true.

Next, we address the question of the near miss to Weber's law. For this purpose, we transform the data of Figs. 2 to 4 shown in terms of $10 \log(1 + \Delta I / I)$ by expressing them in terms of $10 \log(100 \Delta I / I)$. The transformed data are shown in Fig. 6 by filled circles and crosses for the normal population, and by the unfilled symbols for the pathological one. For the purpose of approximation by a regression line (solid line), all the data for the normal population are pooled together. The same is done for the pathological population (intermittent line). The line for the pathological population has a somewhat steeper slope, but the difference is small and it is ignored in the following discussion. Since the correlation coefficients are $r = 0.97$ for the pathological population, and $r = 0.99$ for the normal one, the straight-line fits are quite satisfactory. Both the straight-line fits and the slopes of 0.1265 and 0.1157, respectively, are in good agreement with the results of Schacknow and Raab (1973), Penner *et al.* (1974), and Jesteadt *et al.* (1977).

According to Fig. 6 and the results of others mentioned above, the Weber fraction as a function of sound intensity may be expressed as

$$\Delta I / I = C I^{-\mu}, \quad (8)$$

where C and μ are positive constants. Rewriting Eq. (8) in the form

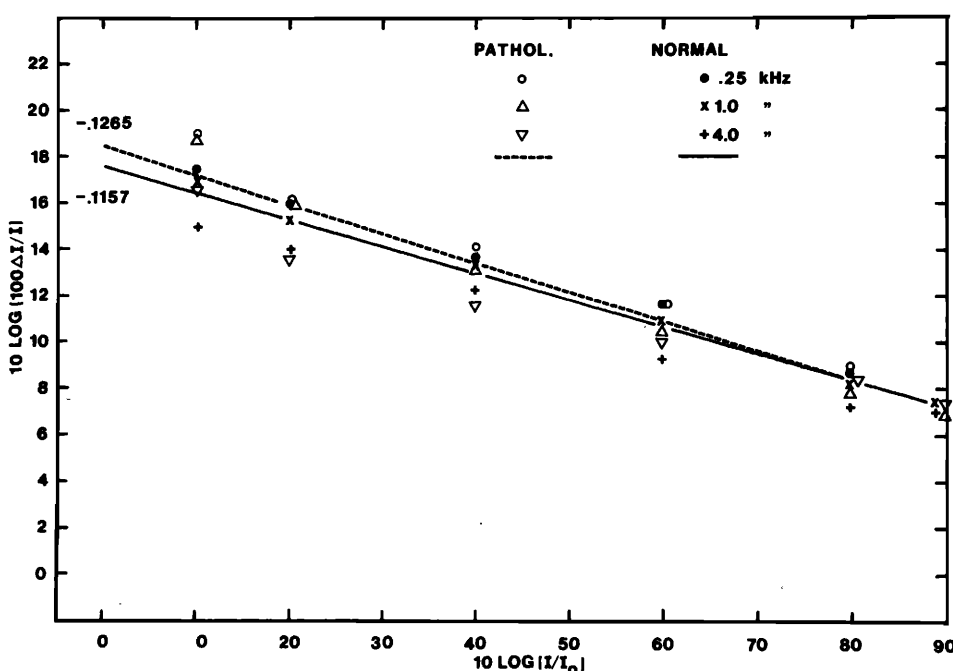


FIG. 6. Measured values of jnd expressed in terms of $10 \log(100 \Delta I / I)$ as functions of sensation level in normal ear expressed as $10 \log(I / I_0)$ with I_0 —threshold intensity. The lines are least-squares regression lines fitted to the pooled data of the normal and pathological populations.

$$\Delta I = CI^{1-\mu}, \quad (9)$$

and replacing ΔI by its expression of Eq. (5), we find

$$D\sigma_L/F'(I) = CI^{1-\mu} \text{ or } \sigma_L = (C/D)I^{1-\mu}F'(I). \quad (10)$$

According to this equation, the near miss to Weber's law is satisfied when the standard deviation of the loudness variable increases as the $(1 - \mu)$ power of sound intensity and in direct proportion to the derivative of the loudness function. The latter condition is consistent with the condition for the jnd independence of the slope of the loudness function.

Interesting insights are gained by introducing Stevens's power law as the loudness function,

$$L = kI^\theta. \quad (11)$$

Under these conditions,

$$F'(I) = \theta k(I^\theta/I). \quad (12)$$

When $F'(I)$ in Eq. (10) is replaced by its expression of Eq. (12), we obtain

$$\sigma_L = (C/D)\theta kI^{\theta-\mu}. \quad (13)$$

Introducing for θ its generally accepted approximate numerical value of 0.3, and for μ , a value of 0.15 which is only slightly larger than that obtained by Penner *et al.* and agrees reasonably well with the results of Doerfler (1948), we come to the relations

$$\sigma_L = (C/D)\theta kI^{0.15} \text{ or } \sigma_L = (C/D)\theta kI^{\theta/2}. \quad (14)$$

Since $L^{1/2} = k^{1/2}I^{\theta/2}$, Eq. (14) can be written in the form

$$\sigma_L = (C/D)\theta k^{1/2}L^{1/2}. \quad (15)$$

In this form it shows that the loudness variable has a Poisson-like distribution. If we make $(C/D)\theta k^{1/2} = 1$, which we can since k is arbitrary, we obtain

$$\sigma_L^2 = L \quad (16)$$

as required by the Poisson distribution. This is of interest because the temporal distribution of neural spikes, which must underlie more or less directly the loudness sensation, is often regarded as a Poisson process (e.g., McGill and Goldberg, 1968).

Another interesting case is for $\mu = 0$ (Weber's law). Then, according to Eq. (13),

$$\sigma_L = (C/D)\theta kI^\theta \text{ or } \sigma_L = (C/D)\theta L. \quad (17)$$

Such conditions prevail in the presence of Gaussian acoustic noise as a stimulus (Miller, 1947; Moore and Raab, 1975; Raab and Goldberg, 1975). In this noise the power is equal to the variance and, after quadratic rectification, the standard deviation should be equal to the mean amplitude, as in Eq. (17). The hair cells appear to approximate half-wave quadratic rectifiers (Zwislocki, 1973; Goodman *et al.*, 1982). Thus Gaussian noise as a stimulus would appear to impose its statistics on the nervous system.

However, the situation is by no means simple. Even a sinusoidal stimulus can produce results conforming to Weber's law under a variety of conditions. When its frequency is very high (Florentine, 1983), when it is accompanied by a band of Gaussian noise not necessarily overlapping in frequency (Viemeister, 1971), and even when it is accompa-

nied by a discrete spectrum of sinusoids (Green and Mason, 1985). It is not clear at all how under these diverse conditions a direct proportionality derived for the relation between the loudness variable and its standard deviation can be established, but such a proportionality appears necessary if d' serves as the detectability criterion. The situation is further complicated by the finding of Teich and Khanna (1985) that the variance, not standard deviation, of the firing rates of single 8th-nerve fibers is directly proportional to the mean firing rate, even in the presence of acoustic Gaussian noise as a stimulus. Several models have been proposed in the past to reconcile these relationships (see Teich and Lachs, 1979 for their model and a review of preceding models). However, the simplifying assumptions made in these models are not entirely in agreement with what we presently know of the peripheral neuronal characteristics.

The last point we would like to discuss concerns the intersubject variability of our jnd determinations. The standard deviation of $\Delta I/I$ is shown in Fig. 7 as a function of mean $\Delta I/I$ for the normal and the pathological populations. The line indicated by the long dashes represents the straight-line least-squares fit to the data of the normal population; the other dashed line does the same for the pathological population. As is evident, the difference between the two lines is small, and it appears justified to represent the pooled data of both populations by one line. Such a representation is provided by the solid line.

Although the fits of the component lines must be considered as good on the basis of the associated correlation coefficients—0.96 and 0.94, respectively—the patterns of the dots suggest a slight curvature concave downward. This means that $\sigma(\Delta I/I)$ grows somewhat more slowly than in direct proportion to $\Delta I/I$. Although it appears farfetched to think that the intersubject standard deviation is directly related to the intrasubject noise limiting the detectability of intensity increments, we should point out that this growth is consistent with the growth of the standard deviation derived from the near miss to Weber's law. Intrasubject standard deviation would be expected to be more closely related to the near miss, but we felt that the small number of repeat jnd measures at our disposal did not permit its sufficiently reliable determination.

IV. SUMMARY OF CONCLUSIONS

Measurements of intensity jnd's for sinusoidal stimuli on listeners with normal hearing and with predominantly monaural hearing loss accompanied by loudness recruitment show that (1) the jnd's depend little if at all on the slope of loudness as a function of sound intensity and are approximately equal when the loudnesses are equal within any given sound frequency, (2) our jnd values obtained by means of amplitude modulation are grossly similar to the jnd values obtained by means of tone bursts of unequal intensity, and (3) the near miss to Weber's law is roughly the same for our amplitude modulation results as for the tone bursts.

Our empirical results can be accounted for theoretically

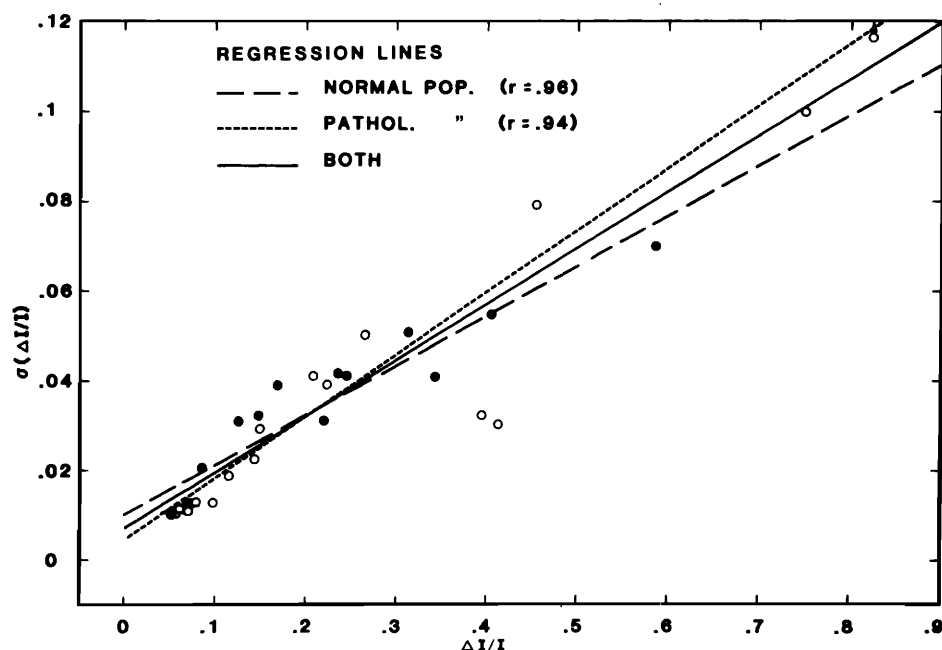


FIG. 7. Standard deviations of the jnd values of Fig. 5 replotted in terms of $\sigma(\Delta I/I)$ vs $\Delta I/I$. Filled circles are for the normal population, unfilled, for the pathological population, and the dashed lines show the corresponding least-squares fits. The solid line shows the regression line for the pooled data.

on the basis of d' as the detectability criterion, if (1) the standard deviation associated with the loudness variable is directly proportional to the derivative of the loudness function, and (2) the standard deviation increases with sound intensity more rapidly than the square root of loudness but less rapidly than loudness.

The required relations of the standard deviation to loudness and its derivative with respect to sound intensity are satisfied if (1) the neural noise underlying the standard deviation originates in the auditory periphery, before the transformation controlling the form of the loudness function takes place, and (2) the noise statistics lie between the Poisson statistics, where the variance is equal to the mean, and Gaussian statistics of acoustic noise, where the variance is equal to the noise power.

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