Speech: Behavior of Middle Ear Muscle during Stuttering

Abstract. *Behavior of the middle ear muscle during speaking was observed in five stutterers by means of the Zwislocki acoustic impedance bridge. Change in impedance did not always parallel precisely the changes in speech sound level. Impedance changed during the initiation and during the course of the stuttering block.*

The middle ear reflex, once considered almost exclusively a protective reaction to loud sound, has become the subject of considerable investigation, and many new modes of activity for the middle ear muscles have been discovered. Increased interest in this subject is in part due to the development of the acoustic impedance bridge, by which small movements within the middle ear can be observed.

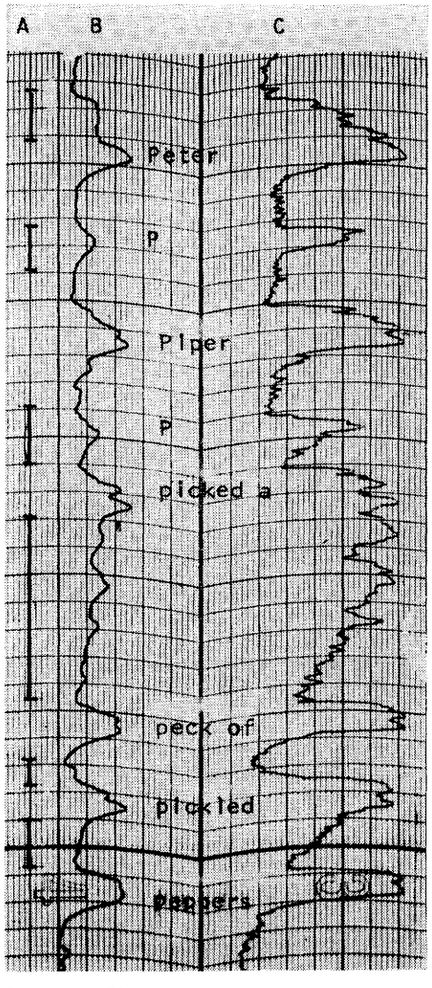

Fig. 1. Change in acoustic impedance during stuttering. Graphic lines show (*A*) stuttering periods, (*B*) speech signal, and (*C*) impedance change. Direction of the chart paper is from top to bottom.

Contraction of the stapedius muscle is directly linked to the initiation of speech and may actually precede the speech sound by a small fraction of a second (*1, 2*). Further, the middle ear reflex appears consistently even during whispered speech but not during random tongue and jaw movements; hence the production of speech rather than the motor movement itself is the key factor in the reflex. The question of muscle activity during stuttering is therefore particularly intriguing, for the stutterer is presumably caught between saying and not saying the word (*3*).

I have studied middle ear activity, during speech, in five stutterers, aged 18 to 22, who have normal hearing. Contractions of the middle ear muscle were observed by means of the Zwislocki acoustic impedance bridge (*4*). The instrumentation (*2*) may be briefly described as a microphone pick-up from the external ear canal, which records the subject's own speech as well as the carrier signal from the impedance bridge. The carrier signal is effectively separated from the speech by means of a tuned amplifier set at the carrier signal frequency.

Speech samples, which included some stuttering, were obtained by asking each subject to recite several familiar passages, after he had been informed that his speech was to be recorded for research purposes. The impedance bridge was held firmly in the ear canal; no gross head movements occurred during the recording.

Contraction of the middle ear muscle in the presence of stuttering and fluent speech was noted in all subjects. Although the relative amplitude of the middle ear reflex varied considerably among subjects (*5*), it appeared consistently both in voiced and unvoiced phonemes.

In Fig. 1, showing impedance change during several stuttering blocks on initial [p] sounds, it may be seen that impedance change did not always parallel the sound level of speech in a precise manner. This is particularly evident in the tracings for the words *Peter* and *pickled*. The graphic pattern preceding the word *peck* is also of particular interest, for it illustrates a much longer and more tonic block. In this instance the subject reported that the block became unexpectedly more severe and that he therefore made a conscious effort to relax prior to successful completion of the word. This description of the block appears to be reflected in the persistently high impedance tracing, followed by a gradual downward slope.

It is evident that the acoustic reflex during stuttering is not simply an indirect way of viewing speech sound but is rather the representation of a specific mode of activity within the speech process. Clearly, the tracings for impedance change and for speech sound are not entirely parallel. This observation applies also to normal speech, but it is much more readily demonstrated in stuttering.

It is conceivable that impedance change may afford a more precise method than sound recording for studying the initiation and course of the stuttering block; it may also provide greater objective information about this much researched but little understood speech disorder.

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Evoked Potential in Relation to Subsequent Alpha Frequency

Abstract. *A series of 24 visually evoked potentials was obtained from a normal human subject under a set of unvarying experimental conditions. The 24 trials were ordered according to the alpha frequency subsequent to presentation of the stimulus. The evoked potentials from the 12 higher- and 12 lower-frequency trials were averaged separately. These two average curves differed significantly at each of the six nodal points.*

For a given set of experimental conditions, considerable stability is shown by the average evoked potential (EP), the electrocortical response to a brief stimulus. This reliability over time has been found to hold over minutes (*1*), hours (*2*), weeks (*3*), and months (*1*). Whatever the interval, stability increases with an increase in the number of evoked potentials averaged. While the

average EP is relatively invariant, the EP varies considerably from trial to trial despite a constancy in the experimental conditions (*4*).

Since most research effort has been directed toward the average EP, the correlates of variability are largely unknown. In part, this reflects the methodological problem of identifying the EP in each trial, a problem which arises from the fact that the EP is generally small relative to the background electrocortical activity. If it is assumed, however, that the variability is not generated by a random process (*5*), any hypothesized correlate of variability can be investigated by a technique which involves the averaging of subsets of trials, with all trials within each subset selected on the basis of a different value of the hypothesized correlate. If these average selected EP's differ systematically, whether in amplitude, latency, waveform, or all three, the correlate may be considered as having been established. Further evidence of relationship would be given if each average selected EP is smoother, or shows less variance, than the average unselected EP, even though the former represents the average of fewer trials.

Since a subject's attention varies from trial-to-trial, and since experimentally induced attention affects the average EP (*6, 7*) and the alpha frequency (the modal frequency of the power spectrum of the electroencephalogram) subsequent to stimulus presentation (*8*), it seems reasonable to expect a relation between EP and subsequent alpha frequency. This relation, for which some indication exists (*9*), was investigated by averaging selected EP's on the basis of subsequent alpha frequency, a characteristic which is identifiable in each trial.

Data were obtained from one subject, a normal male adult (*10*). He was fitted with an electrode 2 cm anterior to, and to the right of, the inion. The a-c potential at this electrode, relative to one attached to the left ear lobe, was amplified and recorded on magnetic tape. The recording system had an accuracy of 1 percent of full scale, and a passband from 0.2 to 50 cy/sec (3-db cutoff points).

The subject was seated comfortably in a darkened room, facing a beaded screen at his eye level 4.5 m away. He was instructed only to attend to the center of a screen, 0.75 m high and 1.00 m wide. A modified motion picture projector was used to present randomly, at intervals of about 10 sec, 24 light flashes, each with a duration of 8.5 msec and a luminance of 172 mlam over the entire area of the screen. The projector was allowed to run for about 5 minutes before the first flash was presented. There was no change in sound intensity at the instant of light presentation.

Each of the 24 analog records was sampled 250 times a second at an aperture time of 100 μsec, converted by a method of successive approximations to 11-bit straight binary code, transferred to a general-purpose digital computer by a technique described previously (*11*), and written on digital tape. Each digital record covered a 4-second interval beginning with the onset of the light flash.

The first step in the analysis necessitated an ordering of the 24 trials according to alpha frequency subsequent to the EP. For each trial this determination included the data beginning 1 second after the light flash and ending after the 4th second and was based on that frequency at which the power was maximum over this 3-second interval. The computation of the power spectral density was based on a preliminary computation of autocorrelations determined at 125 time lags of 4 msec each.

After the 24 trials had been ordered according to subsequent alpha frequency, with the highest rank being associated with the highest alpha frequency, a rank-difference correlation was computed between this alpha frequency order and the trial presentation order. The resulting Spearman rho correlation was not significant.

The 24 alpha frequencies ranged from 9.3 to 11.0 cy/sec with 6 of the 24 alpha frequencies centered around 9.6. The subject's nonstimulus alpha frequency under the experimental conditions was also 9.6, as computed from a randomly selected 3-second interval preceding the first flash; a similar computation based on data following the last flash yielded an alpha frequency of 9.7.

The second step in the analysis required the division of trials ordered according to alpha frequency. The simplest possible division—a dichotomy—was employed: the 12 lower-frequency trials were placed in one group and the 12 higher-frequency trials in the other. The 12 selected trials within each group were averaged, and the result was plotted as a computer output. Figure 1 shows these two outputs, as well as their combination.

The 12 lower-frequency trials yielded an average EP with positive deflections at about 40 msec and, there-

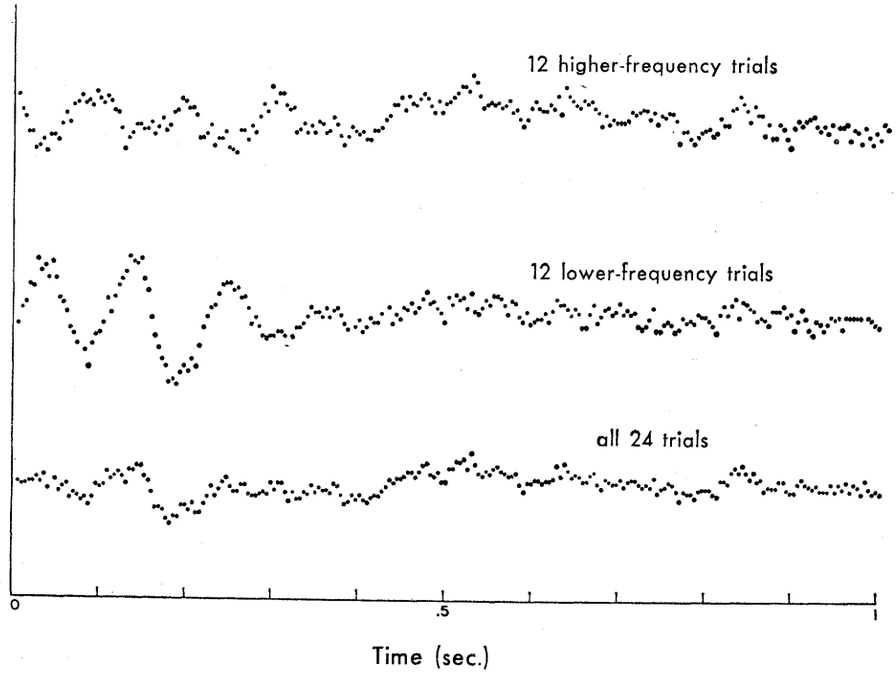

Fig. 1. Average evoked potentials of three sets of trials. The three curves are plotted to the same scale. A positive potential at the recording electrode is upward for each curve. The stimulus was presented at $t = 0$.

after, at increments of about 100 msec, and with negative deflections evenly spaced at intermediate points. Relative to this curve, the 12 higher-frequency trials yielded an average EP whose deflections are not only opposite in sign but are also attenuated. The average for all 24 trials yielded an attenuated copy of the average EP obtained from the 12 lower-frequency trials, as might be expected. The first three positive and negative peaks of the 12 lower-frequency trials differ significantly at the .01 level from the six time-associated points of the 12 higher-frequency trials.

These results indicate that the process of averaging over all trials may be misleading if it is assumed that the trials are drawn from a homogeneous population. Walter has noted regular serial changes in the EP in a given run, (*12*), Brazier has demonstrated systematic changes between successive averages of 60-trial blocks (*13*), and my study shows reliable nonserial changes within a single 24-trial block. These results suggest that, where averaging is employed, variance should also be considered.

In Fig. 1 each point in the curve of the 12 higher-frequency trials is the average of 12 values. In general, the variance of these 12 values is greater than the variance of the same time-associated values comprising the lower-frequency curve, a result which is consistent with the greater dispersion of points in the higher-frequency curve of Fig. 1. A similar result is obtained when the 24 trials are divided into three groups of 8 trials each, or four groups of 6 trials each, again according to subsequent alpha frequency. In each case, the variance increases with an increase in the alpha frequency of the trials.

In addition to Fig. 1, further evidence of the relation between EP and subsequent alpha frequency is revealed by the fact that, in general, the variance at each time point tends to decrease with a decrease in the alpha frequency range of each group. Of course, associated with this decrease in range is an increase in the number of groups and a decrease in the number of trials in each group. Despite this decrease in the number of trials, the standard error of the mean at each time point often shows a decrease, especially for the lowest-frequency group.

Inasmuch as the subject used in this study was not selected on the basis of his evoked potential and alpha frequency characteristics, characteristics which analysis revealed as normal, it is assumed that the relation between evoked potential and alpha frequency occurs in the general population. Nevertheless, the validity of this assumption has yet to be confirmed.

Finally, I have assumed that the relation between the evoked potential and subsequent alpha frequency is based on trial-to-trial variation in attention. However, since attention was not measured independently in this study, alternative explanations, such as that based on the phase of alpha at the time of stimulus presentation, cannot be discounted.

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28 March 1966

Statistical Analysis in Toxicity Experiments

H. W. Norton [*Science* **152**, 671 (1966)] makes three criticisms of Lindsay and Kullman's "Pentobarbital sodium: variation in toxicity" [*ibid.* **151**, 576 (1966)].

To the first paragraph of his letter I would reply as follows. Survival rate varied as much as ninefold between consecutive time periods on a given day, or, at the other extreme, did not vary at all between as many as four consecutive periods. Nevertheless, the question of whether dependence of toxicity on time varies from experiment to experiment is not of primary importance. The major purpose of the paper is to indicate that a simple quadratic may not be sufficient to explain the dependence of toxicity on time.

The comment that the comprehensive test of interaction reaches only the probability of 0.092 may be irrelevant. I believe it is possible for interaction not to be significant in the comprehensive test and yet be real in individual tests.

Although the arcsin transformation does, as Norton notes, alter the form of the toxicity curve slightly as sensitivity changes, I do not believe that this contributes significantly to interaction. The effect of this transformation would be to flatten the curves lying near the extremes of the ordinate more than others. However, near the lower extreme (the only extreme approached) the curves are already rather flat; in the experiment in which survival was lowest, for example, one animal survived in each of four successive periods. The straight-line portion of this curve is not distorted by the transformation, yet it contributes strongly to interaction. It should be noted that flattening would tend to hide fluctuations in the curve and thus lessen the chance of their detection in tests of significance. We think, however, that a variance-stabilizing transformation is needed and that the arcsin is the best available for the data.

Concerning the parenthetical remark in Norton's second paragraph, I would say that having begun this line of reasoning Norton should have extended it to include calculation of the probability of both the quadratic and septic effect occurring by chance in the same sample; the probability would be <0.04 at the 5-percent level, and should the actual levels be used (0.005 and 0.032, respectively), much less. Furthermore, in testing whether the curve is more complex than a quadratic (as indicated by the septic effect significant at the 0.032 level), only tests of the higher-order effects need be considered. A comprehensive test of cubic through