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THE STUTTERING LARYNX: AN ELECTROMYOGRAPHIC STUDY OF LARYNGEAL MUSCLE ACTIVITY ACCOMPANYING STUTTERING

by

Frances Jackson Freeman

A dissertation submitted to the Graduate Faculty in Speech and Hearing Sciences in partial fulfillment of the requirements for the degree of Doctor of Philosophy, The City University of New York.

1977

This manuscript has been read and accepted for the Graduate Faculty in Speech and Hearing Sciences in satisfaction of the dissertation requirement for the degree of Doctor of Philosophy.

March 24, 1977
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ABSTRACT

Laryngeal muscle activity during fluent and stuttered utterances was investigated using multichannel electromyography. Analysis revealed that stuttering was accompanied by high levels of laryngeal muscle activity and disruption of normal reciprocity between abductor and adductor forces. The results demonstrate the existence of a laryngeal component in sutttering and show a strong correlation between abnormal laryngeal muscle activity and perceived moments of stuttering.

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Although this is chronologically the first page in this manuscript, it is the last to be written. Throughout the work involved in this endeavor I have been sustained by the knowledge that in the end I would have earned this opportunity to publicly acknowledge my indebtedness to the many individuals who have contributed both to the research and to my life as represented in this research.

The commitment of the four gentlemen who served as subjects for the series of experiments, entitles them to a full share in any value or merit the research may have.

I gratefully acknowledge the invaluable contributions made by the chairman and members of the thesis committee, Katherine S. Harris, Norma Rees, Oliver Bloodstein, Gerald McCall, and Irving Hochberg.

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Much of my approach to the disorder of stuttering has been influenced by the man who introduced me to the field and thus motivated this research, Charles E. Palmer.

Two women, by their influence and example have changed the course of my life. Irma Stockwell Taylor made me wish to be a speech pathologist and Katherine Stafford Harris made me wish to be a scientist. If I should ever achieve anything of merit in either area it will be entirely due to these two extraordinary teachers.

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My husband, Charles C. Freeman, is a full partner in this research. He served as a subject for the pilot experiments and supported me through the final draft. We jointly thank our daughters Jacke Lee and Denise for the contributions and the sacrifices which they have made to this project.

In 1970, when I believed that there was no possible way for me to pursue doctoral studies, I prayed for such an opportunity. In 1972 when it appeared that I would not successfully complete this research

I prayed again. Both prayers were answered promptly and beyond my expectations. I would be less than honest if I did not gratefully acknowledge His kindness in allowing me to pursue this work.

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INTRODUCTION

INTRODUCTION

This research investigated physiological events which occur in conjunction with moments of stuttering. Its primary aim was to describe the laryngeal muscle activity which accompanies stuttering. It should not be interpreted as a theory of the "cause of stuttering" in the traditional sense of an ultimate etiology of the disorder, though it was hoped that the findings would yield insights into the proximal causes of individual moments of stuttering.[1]

The need for such a study emerged from a survey of relevant literature. The conclusions drawn from the literature may be summarized as follows:

1. For over one hundred and fifty years hypotheses concerning a laryngeal component of stuttering have been proposed and discussed, but have not been tested experimentally.

2. Within recent years an increasing number of studies have indirectly implicated the phonatory mechanism (or phonatory failure) in stuttering.

3. A small number of physiological studies have offered direct evidence of abnormal laryngeal activity during moments of stuttering.

4. The technology which makes electromyography a feasible method for investigation of intrinsic laryngeal muscle activity

has been developed and utilized in a series of studies of normal speakers. These investigations, which have expanded the understanding of laryngeal speech physiology, form a normative body of data for comparison with pathological cases.

5. Electromyography has the potential for providing new information pertinent to a number of questions relating to stuttering, including: (a) questions related to states of muscle tension and (b) questions related to coordination of muscle activity.

The following review of literature is presented in five sections which correspond to, and indeed, generated these conclusions.

NOTES

1. Two types of "theories of stuttering" are discussed by Bloodstein (1969). He differentiates between theories which account for "the conditions under which the disorder comes about," and those theories concerned with "the nature of discrete instances of stuttering behavior." It is in relation to this latter type of "theory" that the present study is felt to have greatest relevance.

CHAPTER I

REVIEW OF THE LITERATURE

1. Theories of Laryngeal Involvement in Stuttering

Glottal Spasm[1] Theories

As early as 1800, a New York physician, Yates (Klingbell, 1939), ascribed stuttering to a "spasm of the glottis." Arnott (1828), a Scottish physician also attributing the cause to spasm of the glottis, wrote, "The most common cause of stuttering, however, is not as has been universally believed. . . . but where the spasmodic interruption occurs behind or beyond the mouth, viz., in the glottis so as to affect all the articulations."

Muller (1833), a noted German physiologist and comparative anatomist, investigated the anatomy of the larynx. Agreeing with Arnott, he concluded, "the immediate cause of stammering is a spasmodic affection of the glottis."

Deleau (1829), described by Klingbell (1939) as a specialist in maladies of the ear, distinguished three types of stuttering, one being characterized by spasm of the glottis.

Schulthess (1830) believed that spasm of the glottis, extending through nerve associations to other organs of speech, was the main cause of stuttering.

Hoffman (1840) subscribed to the glottal spasm theory, and advocated physical relaxation of the throat, tongue and lips, and avoidance of effort in other parts of the body.

Although best known for his diagnostic description of ataxia, the German pathologist and neuropathologist, Romberg (1858) also investigated stuttering, placing the disorder among the "neuroses of mobility," under the subtitle of "vocal spasms."

Bristowe (1879) defined stammering as a "spasm of the vocal or articulating organs," which might occur at the lips, the point or the root of the tongue, or at the glottis.

Potter (1882) described stuttering as a spasmodic habit, which once established became automatic. He felt that the spasm occurred at various stop points in the vocal tract, including the larynx.

Thorpe (1900) described an interaction between air flow and air pressures and spasm of the glottis. He believed that a spasm limited to the glottis affected the vowels, but that spread of the spasm to the lips and tongue affected the consonants as well.

Although it may not be appropriately considered a fully developed theory, McCall (1974) has provided provocative material in his discussion of commonalities or possible connections between spastic dysphonia and the stuttering block. With his colleagues, Skolnick and Brewer, McCall (1973) investigated laryngeal muscle activity in spastic dysphonia. He hypothesized that similar research with stuttering should lead to a better understanding of the pathologic mechanisms underlying the block. He specifically suggests that research into the laryngeal muscle activity accompanying stuttering should devote particular attention to seeking evidence of (1) muscle spasms, (2) involuntary movements, particularly tremor, and (3) disturbed muscle tone or dystonia.

Inappropriate Abduction/Adduction

Theories

According to Klingbell (1939), John Howard of New York was a specialist in the treatment of defective speech who published a number of articles on stuttering between 1879 and 1881. Howard maintained that stuttering was a throat contracting habit. In his view the problem was contraction of the glottis on vowel sounds, even when the stuttering appeared to be on consonants.

Within this century, Kenyon (1943) has been the foremost proponent of a laryngeal closure theory of stuttering. He concluded that the primary abnormal muscular action in stammering was vocal fold adduction which prevented the passage of air between the vocal cords. In his opinion, secondary abnormal actions consisting of an articulatory struggle were superimposed, and resulted from the failure of appropriate laryngeal activity. He described the essential picture of each stammering act as, "the substitution of vocal cord adduction for the production of an individual speech sound."

In contrast to Kenyon's adduction theory, Schwartz (1974)[2] has proposed a model of the stuttering block in which phonation is interrupted or prevented by inappropriate opening of the vocal folds, resulting from vigorous contraction of the posterior cricoarytenoid, in response to the subglottal air pressures required for speech. In response to this, "inappropriate airway dilation reflex (ADR)," Schwartz theorizes that the individual can elect one or both of two major sites at which to exhibit muscular compensation. He may, "elect to forcefully contract any or all of the musculature capable of overcoming the powerful abductory response of the PCA, thus evoking primitive, gross, sphincteric responses of the larynx;" or he may, "elect to struggle supraglottally, displaying hypertense postures of the lips, tongue, or jaw as a means of attempting the release of the abducted vocal folds."

Tension and Coordination Theories

Serre d'Alais (1829) described two types of stuttering, one a chorea of the muscles of articulation, and the other a tetanus of the muscles of phonation and respiration.

Colombat de Pisere (1831) described one type of stuttering characterized by rigidity of the muscles of the larynx and pharynx. This rigidity, he felt, produced spasms of the glottis, which were characterized by sudden stoppages of the breath-current.

A number of writers have been concerned with problems of coordination in stuttered speech. Sir Charles Bell (1832), the Scottish anatomist, described stuttering as a form of chorea, in which the speaker has an inadequate capacity to properly coordinate the different actions required for fluent speech. He stressed the importance of the laryngeal role in speech articulation, and described the disordered breathing of stutterers.

Poet (1833) wrote that the problem consisted of the spasmodic action of the muscles of speech acting in opposition to those of phonation. Bishop (1851) believed that stuttering was induced most frequently by attempting to speak, "without producing vibrations of the vocal cords." He blamed imperfect training of the organs of articulation, and defects in the sympathetic association between the articulatory and vocal organs.

James Hunt (1869) concluded from his years of work with stutterers that "stammering essentially consists of the want of power to combine the different actions concerned in vocalization." He felt that disharmony between phonation and articulation was the chief source of stuttering.

Kussmaul (1877), a German physician and surgeon noted for his work on the pulse and respiration, defined stuttering as a "lallo-neurosis," a "disarthria syllabaris," in which there is a want of harmonious action between the muscles of respiration, vocalization and articulation.

In several respects there exists a parallel between the "disharmony of actions," described by Hunt, and Van Riper's (1971) statements on the nature of stuttering. In his "attempted synthesis," Van Riper proposes that stuttering be considered a disorder of timing. In this framework he describes a block as, "a temporal disruption of the simultaneous and successive programming of muscular movements required to produce one of the word's integrated sounds, or to emit one of its syllables appropriately, or to accomplish the precise linking of sounds and syllables that constitutes its motor pattern." He concludes that the essence of the disorder is in the fracturing and disruption of the motor sequence of the word. Although a laryngeal component is not primary to the Van Riper hypothesis, he includes careful consideration of possible phonatory disruptions. He points out that in physiologic startle the laryngeal valve is suddenly and tightly closed, and that milder emotional states are often reflected in laryngeal tension (globus hystericus). He concludes that, "The delicate balancing of antagonistic muscle groups required for phonation can be altered by emotionally induced changes in muscular tension. If it is often difficult to speak under profound emotional stress, the reason may lie in the breakdown of either respiration or phonation or in the disruption of their synchronization."

In his discussion of the effects of emotionally produced tension on the timing of muscle activity in finely coordinated gestures, Van Riper suggests that much of the effect of emotional stress is probably caused by the mistiming produced by inappropriate changes in muscle tonus of the antagonistic muscle groups.

In his recent work, Bloodstein (1974) has proposed a model of the stuttering block in which he holds that stuttering consists primarily of "tension and fragmentation." He states his belief that the usual descriptions of stuttering--i.e., repetitions, prolongations, hard attack or silent intervals--relate to the surface aspects of the behavior. He concludes that on a deeper level, the stutterers' behavior consists of tensions and fragmentations in speech. He hypothesizes that the tensions and fragmentations result from the stutterer's belief in "the difficulty of executing the motor plan of some element of speech, perhaps momentarily sharpened by communicative pressures."

The views discussed by Adams (1974) have much in common with those expressed by Hunt (1961), Van Riper (1971), and Bloodstein (1974). Adams concludes a review of the contributions of respiration, phonation, and articulation to fluency with the statement that, "the vocal tract dynamics promoting fluency may be represented by the harmonious integrations of subglottic pressure, glottal resistance, and supraglottic pressures . . . . From this line of reasoning it follows that behaviors that prevent or upset the aforementioned integrations could generate stuttering. Stutterers should then have observable difficulties in managing respiration, phonation, and articulation, and in the coordination of these processes."

After reviewing the literature on speech production in stutterers, Adams concluded that stutterers possess a variety of behaviors that make prompt initiation, timing, and maintenance of airflow and phonation difficult or temporarily impossible to achieve. In this framework, he suggests that, "articulatory postures and gestures are prolonged and repeated as the individual strives to coordinate labial, mandibular, and lingual movements with a wavering airstream and vocal note that sometimes are not present when needed. Only when dependable airflow and phonation are restored, does fluent articulation appear."

Moravek and Langova (1967), building on their earlier work (Langova and Moravek, 1964 and Moravek and Langova, 1962), have developed a theory in which the central problem of stuttering is the prephonation or initial tonus (IT) period. By IT they designate "that phase of speech in which the patient starts to form voice and where the majority of the phonatory muscles are in action, but where the corresponding vocal effect is not achieved." They suggest that when voice is not achieved the stutterer increases his muscular effort, a reaction which further impedes his efforts to speak. These investigators describe the "basic pathogenic mechanisms, of stuttering as the disharmony between muscular activity and the proprioceptive feedback on one hand, and the central programme of the elementary articulation process on the other hand."

2. Research Implicating the Phonatory Mechanism in Stuttering

Implications of the Fluency Evoking Conditions

Among the controversies abounding in the literature on stuttering, a few phenomena have been reported with enough consistency to justify general acceptance. The ameliorative effects of certain speaking conditions are among these accepted "facts" (Van Riper, 1971; Bloodstein, 1969; and Beech and Fransella, 1968).[3]

In two related articles, Wingate (1969 and 1970) reviewed the literature on fluency-evoking conditions, and suggested a common factor underlying them. In the earlier article (1969) he dealt with a number of fluency evoking conditions, including choral reading, rhythm reading, and whispering. In the second article (1970) he considered two conditions of audition, DAF and masking, which have been demonstrated to effect reductions in stuttering. (Adamezyk, 1959; Chase, Sutton and Rapine, 1961; Lotzman, 1961; Soderberg, 1968; and Han and Steer, 1967).

In his conclusions, Wingate made a strong case for his theory that these fluency evoking conditions have the common property of effecting changes in the manner of vocalization. He hypothesized that, "in these circumstances which improve fluency, the stutterer is induced, in one way or another, to do something with his voice that he does not ordinarily do."

Underlying a portion of this hypothesis is Wingate's contention that the conditions of audition (masking and DAF) produce changes in vocalization. He cites increased vocal intensity and slowed syllable rate as the most obvious kinds of vocal changes.

Two recent studies (Adams and Hutchinson, 1974; and Contour, 1974) specifically tested this hypothesis as it relates to the masking condition. Both studies reported that vocal intensity levels were significantly higher and stuttering frequency significantly lower in the effective (louder) masking conditions.

Analysis of the rate effects was more complex. If the number of stutterings is reduced, then words per minute is increased. This increase, due to reduction of stutterings, interacts with any decrease which might be produced by slower syllable utterance rate. Thus, when Contour reports that the loudness of the noise did not significantly change reading rate; and when Adams and Hutchinson report that reading time was usually shorter in the noise condition, caution must be given in interpretation. Adams and Hutchinson point out these problems in discussion of their results.

In a related study, Reis (1974) compared the frequency of stuttering under four conditions: (1) normal vocal intensity, (2) below normal intensity, (3) above normal intensity and (4) under 75 dB white noise masking. While the normal condition resulted in significantly more stutterings, the other three conditions did not differ significantly from one another in their fluency evoking effectiveness. This research would tend to substantiate Wingate's hypothesis that a change in phonatory manner is the critical element in the fluency evoking conditions.

Implications of Adaptation Studies

Adaptation, as another recognized phenomenon of stuttering, has been "explained" in a variety of ways. The theories of adaptation in stuttering was reviewed by Bloodstein (1969).

Of these explanations, the "oral rehearsal" theory, as tested experimentally by Brenner, Perkins, and Soderberg (1972), was found to have special relevance to laryngeal functioning. Brenner and his co-workers asked the key question, rehearsal of what? In order to find an answer, they compared five rehearsal conditions, (1) silent rehearsals without lip movements, (2) silent rehearsals with lip movements, (3) whispered rehearsals, (4) aloud rehearsals, and (5) no rehearsals. When they found that only the aloud rehearsals produced adaptation, they concluded that, "the adaptation effect is a function, at least in part, of the voiced phonatory in conjunction with the articulatory components of speech."

Three studies, Adams and Reis (1971 and 1974) and Riemenschneider et al. (1974), have investigated the influence of the onset of phonation on the frequency of stuttering. Adams and Reis used two specially constructed paragraphs, one containing the normal combinations of voiced-voiceless sounds and one containing only voiced sounds. In the first study (1971) fourteen stutterers performed five "massed oral readings" of each paragraph. The authors reported (1) a significantly higher frequency of stuttering on the reading of the paragraph which contained the normal combinations of voiced-voiceless sounds, and (2) a significantly smaller adaptation effect on the readings of the paragraph containing the normal combinations of voiced-voiceless sounds. The paragraph containing the normal combinations of voiced-voiceless sounds was believed to be more difficult for the stutterer because it required more "on-off" phonatory adjustments.

In the 1974 replication study, the authors again found a higher frequency of stutterings on the readings of the paragraph with the normal combinations of voiced-voiceless sounds, but this time the differences did not reach the level of significance. However, their second finding, relating to the differences in rate of adaptation was confirmed. It appears that the rate of adaptation is negatively related to the number of "on-off" phonatory adjustments required.

Riemenschneider, et al. (1974), working with Adams, used a design which added a third paragraph to the original set. The new passage was composed entirely of voiced continuant sounds (in contrast to the original all-voiced passage which contained both stops and continuants). The overall differences in the amount of adaptation from passage to passage was significant beyond the .001 level, with the greatest differences between the paragraph with the normal combination of voiced-voiceless sound and the paragraph with only voiced continuants. These results supported the previously stated conclusion that adaptation increases as the number of requirements for prompt voice initiation decreases.

Implications of Studies of Phonation in Stuttering

Stromstra (1965) subjected to spectrographic analysis the recordings of the speech of thirty-eight children who had been brought to a speech clinic for suspected stuttering. He reported that the repetitive dysfluencies of those children who later became stutterers displayed abrupt stoppages of phonation which were absent in the repetitions of children who later became fluent speakers.

Agnello (1971) and Van Riper (1971) both report occurrences of "fry-like" phonation in stuttered speech. These may indicate an abnormal mode of vocal fold vibration in some moments of stuttering.[4]

Agnello (1974) further reports findings of longer voice onset times (VOT) and longer voice termination times (VTT) in the fluent utterances of stutterers when considered in light of comparable VOT and VTT measures of fluent speakers.

Adams and Hayden (1974) designed a study to test the hypothesis that stutterers have difficulties in rapid initiation of phonation. Using simple vowel phonation (a situation in which little stuttering normally occurs) they found that stutterers were significantly slower than nonstutterers in initiating and terminating phonation. Stutterers also differed from the normally fluent controls in the rate at which they improved their voice initiation and voice termination times with continued practice.

Agnello's work on VOT and VTT in stutterers prompted Di Simoni

(1974) to study other timing relationships in the speech of stutterers. He examined "the fluent speech of stutterers for the effects of final consonant on duration of the preceding vowel and of final vowel on the duration of the preceding consonant." He found that the vowel durations achieved by stutterers were "greatly different" (124 to 201 milliseconds longer) than those previously reported for normal speakers (House and Fairbanks, 1953). However, the essential vowel-duration relationships due to the nature of the final consonant were retained. He also found that the consonant durations of the stutterers were much greater ($p< 0.05$) than those found for a group of nonstutterers matched for sex and age. Stutterers also showed a great deal more variability in consonant duration control than did nonstutterers.

In the consonant experiment the subjects recorded 25 repetitions (tokens) of each utterance type. Spectrograms were made, and the duration measurements for repetitions 2-6 were compared with the duration measurements for repetitions 19-24. The author had hypothesized that the stutterers would show disturbed timing relationships on the first repetitions and more nearly normal relationships on the last trials. Instead, the converse proved to be the case. The stutterers' first trials (although significantly longer in duration than those of the normally fluent controls) exhibited normal patterns of differential durations related to the final vowel. Their last repetitions (while still significantly longer than the normals) did not demonstrate the normal pattern of differential durations. Di Simoni describes this finding as unexpected and worthy of further research.

Webster (1975) studied the characteristics of phonatory onsets in ten stutterers. In five hundred speech samples from stutterers he found 373 samples in which the initial portion of phonation showed irregularity in the pitch periods, and 127 samples in which the distribution of pitch periods was judged abnormal (by comparison with 100 speech samples from 10 nonstutterers). In addition to the irregular periods, these waveforms were lower in amplitude than those associated with normal phonation and were frequently accompanied by an audible, strained voice quality. In some instances, the acoustic signal was reported to be barely audible even though the oscilloscope clearly showed the phenomenon. Webster goes on to note that when compared with the samples of the normal speakers, many of the stutterers' speech samples displayed faster amplitude rises during the first several cycles.[5]

In discussion of these findings Webster states, "It is likely that during these unusual oscillations deviant tensions were occurring in laryngeal muscles responsible for voicing. The irregular phonation seen in nonnormal samples could also result from excessive or unstable tensing of vocal folds."

3. Physiological Studies Offering Direct Evidence of Abnormal Laryngeal Activity

Chevrie-Muller (1963) utilized the glottalgraph to study twenty-seven stutterers, and reported abnormal laryngeal activity which included (1) arhythmic vocal fold vibrations, (2) unpredictable glottal openings, and (3) partial or complete absence of voicing during rapid glottal activity.

Fujita (1966) took posterio-anterior X-rays of a Japanese stutterer, reporting (1) irregular or inconsistent opening and closing of the pharyngolaryngeal cavity, and (2) asymmetric tight closure of the glottis which extended upward and included closure of the pharyngeal cavity.

Ushijima, Kamiyama, Hirose, and Niimi (1965) produced a film "Articulatory Movements of the Larynx During Stuttering." Using a thin fiberscope, which inserted through the nasal passage and yielded a superior view of the larynx, they photographed stuttered and fluent utterances. The film reveals abnormal laryngeal activity during stuttering, including (1) irregular, unpredictable glottal openings and (2) very tight closure of the laryngeal aperture, which included adduction of the vocal folds, adduction of the ventricular folds, and in some instances, anterior-posterior closure at the level of the arytenoids and the tuberculum of the epiglottis.

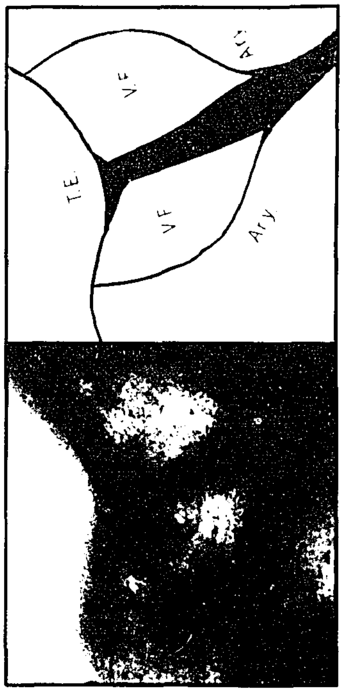
Freeman and Ushijima (1975) produced still photographs from sequential frames of the film. The four frames reproduced in Fig. 1 illustrate the sequence typical of the tight laryngeal closure of some moments of stuttering. Each frame is shown in its original form, and in a tracing of the tissue outline. Frame 1 shows the true folds in phonatory position. In Frame 2 the ventricular folds can be seen as they are adducted and partially occlude the glottis; while in Frame 3 the adduction of the ventricular folds is almost complete. Frame 4 shows anterior-posterior closure at the level of the arytenoids and the tuberculum of the epiglottis. Even with this tight closure of the larynx, the subject is still attempting (unsuccessfully) to phonate.[6]

Contour, Brewer and McCall (1974) also utilized the fiberscope in their study of six stutterers. These authors considered laryngeal behavior patterns in relation to two dysfluency types--sound-syllable repetitions and prolongations. They reported that most sound-syllable repetitions were characterized by the vocal folds being held in a partially-abducted position. While in this position, the vocal folds were frequently observed to oscillate, a small degree laterally and then back again a small degree medially. An exception to this general pattern was noted when some subjects produced monosyllabic whole word repetitions; in these cases, laryngeal adduction was observed. Within their limited samples of sound prolongations they observed examples of both abnormal abduction and adduction. In discussion of their experiments these authors state that their observations on sound-syllable

FIGURE 1

Laryngeal closure sequence during stuttering block. Individual frames[6] from the film "Articulatory Movements of the Larynx During Stuttering" produced at the Research Institute of Logopedics and Phoniatrics, University of Tokyo (1965). Producers, Ushijima, T., G. Kamiyama, H. Hirose, and S. Niimi.

1



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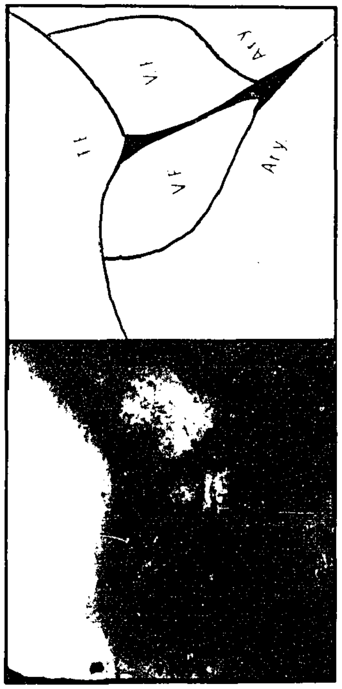

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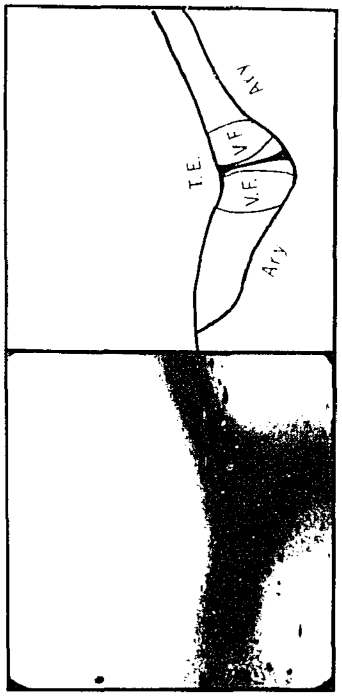

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repetitions are suggestive of a disturbance in the smooth, reciprocal interplay between agonist and antagonist intrinsic laryngeal muscles. They state, "it is as though the laryngeal adductors and abductors are simultaneously and/or alternatively contracting in a manner that results in a fluency-disruptive, laryngeal 'tug-of-war' between these two opposing muscle groups."

4. Electromyographic Investigation of Intrinsic Laryngeal Muscle Function in Speech

The processes of human speech can be investigated on one or more of several levels. Vocal tract dimensions, and certain articulator movements may be inferred from analysis of the acoustic signal or from air pressure and flow measures. In contrast to these "indirect" approaches, direct observations and measurements of movements can be made utilizing fiberoptic or radiographic photography (Sawashima and Hirose, 1968; Sawashima and Ushijima, 1971; and Landman, 1970). Electromyography, however, monitors yet a different level of function--that is, the signals which accompany the muscle contraction and lead to the articulator movement. As a research technique electromyography allows study of the dynamics of speech production, not only in the descriptive terms of which muscles contract and when, but also in terms of the relationships between the component gestures of speech and their linguistic counterparts. These, in brief,are the philosophical constructs underlying the use of electromyography in the study of human speech and language.

In the preface to the third edition of Muscles Alive, Basmajian (1974) makes the following observations about the "state of the art" of electromyography: "Electromyography has now come of age. Technique has caught up with enthusiasms. Investigators no longer are forced to choose the easiest way, but more and more they choose the best electrodes, and best analytic methods. Inserted electrodes, multichannel apparatus, multifactoral recordings, computer technologies all are becoming routine."

Techniques for electromyographic investigation of the intrinsic laryngeal muscles in humans have been reviewed and described by Faaborg-Anderson (1957), Cooper (1965), Hirano and Ohala (1969), Gay and Harris (1970) and Hirose (1971a).

Although a number of questions relating to laryngeal muscle function in speech remain unresolved, electromyographic evidence exists on a number of points. The findings relating to the muscles of interest in this research may be summarized as follows:

1. Cricothyroid (CT). Contraction of the CT acts mechanically to diminish the distance between the cricoid arch and the thyroid lamina. This action increases the distance between the thyroid cartilage and the vocal processes of the arytenoid cartilages, modifying the length and/or tension in the vocal folds. The function of CT has been studied in both singing (Gay, Strome, Hirose, and Sawashima, 1972; Shipp and McGlone, 1971) and in speech (Erickson, 1976; Collier, 1975; and Atkinson, 1973). In both activities increases in fundamental frequency were found to correlate with increases in CT activity. Although other muscles may act for pitch lowering, and other muscles may participate or assist in pitch raising, the CT is considered the primary pitch raiser.

In nontone languages fundamental frequency changes contribute to the perception of the suprasegmentals--intonation and stress. Atkinson (1973) and Collier (1975) both attribute a primary role in intonation control to the CT. Collier studied a number of different intonation contours in Dutch, and found that apart from one exception (when fundamental frequency was low and falling gradually), the CT always showed a straightforward correlation with the fundamental frequency.

Hirose and Gay (1972) found an increase in CT activity for stressed vowels, but no participation in the segmental (voiced-voiceless) distinction. These findings, which relate CT activity to the production of the suprasegmentals--intonation and stress--rather than to segmental articulation appears consistent in studies of normal speakers of English.[7]

Two other aspects of CT function deserve mention. First, in vocal fry (or pulse register) phonation, McGlone and Shipp (1971) have reported CT suppression. Second, during tight glottal closure as in swallow, CT activity is generally suppressed.

2. Posterior Cricoarytenoid (PCA). Contraction of the PCA acts mechanically to increase the distance between the vocal processes of the arytenoid cartilages. As the primary muscle of vocal fold abduction,[8] it is active for inspiration and strongly active for deep inspiration (Suzuki and Kirchner, 1969).

Hirose and Gay (1972) reported on intrinsic laryngeal muscle activity in contrasting utterances of voiced-voiceless stops and voiced-voiceless fricatives. They found consistent increases in PCA activity for voiceless consonant production, with suppression of PCA activity for voiced segmentals. They reported that the PCA does not show a simple "all-or-none" pattern of activity, but rather shows a pattern of fine adjustment. These findings were verified in later studies (Hirose, 1974 and Hirose, Lisker and Abramson, 1972). In a recent investigation of Japanese (Hirose and Ushijima, 1974) the relationship between PCA activity and glottal gestures for voiceless sounds was studied through correlation of the electromyography with frame analysis of fiberoptic films of the glottis. The investigators stated that, "Our results further indicate that the degree and timing of PCA activity are directly responsible for determining the size and the temporal course of the glottal opening for voiceless segments, although the suppression of the adductors may also have to be taken into consideration for a complete description of voiceless segment production."

One other point made in each of these studies deserves comment. When observing PCA activity Hirose and his colleagues consistently found a clear pattern of reciprocity between the PCA and the interarytenoid (INT). Shipp (1975), in his laryngeal studies, has observed this same pattern.

3. Interarytenoid (INT). Contraction of the INT acts mechanically to diminish the distance between the arytenoid cartilages. Anatomy texts traditionally group the INT with the thyroarytenoid (TA) and lateral cricoarytenoid (LCA) as the glottal adductor group (Dickson and Maue, 1970; Zemlin, 1968; Kaplan, 1960). However, EMG studies suggest functional differentiation among the adductor muscles (Hirose, 1971b and 1974). Because of the previously mentioned pattern of

reciprocity between the PCA and INT, Hirose and Gay (1972) have considered the INT to be the pure adductor of the vocal folds. They found INT activity for all voiced segments, both vowels and consonants, with INT suppression accompanying all voiceless segments. INT activity is apparently lower for voiced consonants (at least in the case of the voiced fricatives and stops studied) than for vowels. Further, INT activity for a vowel following a voiceless stop is higher than activity for a vowel following a voiced stop. Since Sawashima et al. (1970) demonstrated that glottal width is greater for voiceless than for voiced consonants, it is reasonable that activity responsible for adduction of the vocal folds should be greater after a voiceless consonant.

The INT operates in synchrony with the other adductor muscles (TA and LCA) in sphincteric glottal closure, in swallowing, gagging or breath holding. High levels of INT activity accompany reflexive glottal closure.

4. Lateral Cricoarytenoid (LCA). Contraction of the LCA acts mechanically to diminish the distance between the vocal processes of the arytenoid cartilages. Although grouped with the INT as a glottal adductor, the LCA (and the TA) show activity patterns which differentiate their function from that of the INT. Like the INT, the LCA and the TA show lower activity during voiceless segments, demonstrating general reciprocity with the PCA (Hirose and Gay, 1972 and 1973).

However, these same studies found that in English,[8] all voiced segmentals were not accompanied by TA and LCA activity. In stead, the LCA and TA were active for vowels, but showed lower levels for consonants (in this case stops and fricatives), regardless of the voiced-voiceless distinction. This pattern contrasts with that of the INT which showed activity for all voiced segments. Both the LCA and the TA showed higher levels of activity for stressed (as compared with unstressed) vowels, a finding which seems to suggest participation in control of suprasegmental pitch variation.

The LCA participates in sphincteric glottal closure, showing high levels of activity in swallowing and breath holding. High levels of LCA activity also occur in glottal stop production and in so-called glottal or hard attack (Hirose and Gay, 1973). Consideration of this general pattern led Hirose (1974) to speculate that the LCA is responsible for medial compression, and that this is its primary contribution to glottal adduction.

5. Thyroarytenoid (TA). The TA forms the main mass of the vibrating vocal folds (Zemlin, 1968). Mechanically, its contraction may produce different effects, depending upon the forces which are simultaneously acting upon the laryngeal structures to which it attaches. Potentially it may act to (1) thicken the vibrating mass of the vocal folds, (2) increase vocal fold tension and (3) adduct the vocal folds. Traditionally, it has been viewed as playing a dual role as an adductor (with the LCA and INT) and as a tensor (with the CT) (Dickson and Maue, 1970). Like the INT and LCA its activity levels are low for voiceless segments, and thus it is suppressed during peak periods of PCA activity. Like the LCA it was active for vowels, but showed no

consistent pattern of activity for consonant segments (stops and fricatives) regardless of the voiced-voiceless distinction.[9] Like the CT, the TA showed higher levels for stressed vowels than for unstressed vowels (Hirose and Gay, 1972). Particularly high levels of TA activity were found to accompany vocal fry (pulse register) phonation (McGlone and Shipp, 1971).

Hirose (1974) has speculated that the TA is responsible for antero-posterior tension increase in the vocal folds, and that this is its primary contribution to glottal adduction. In general, it must be noted that the activity patterns of the LCA and the TA are quite similar.

5. Electromyographic Investigation of Tension and Coodination

Since both excessive tension and disrupted coordination were implicated in the hypothesis of phonatory disruption in stuttering, consideration of the EMG correlates of these factors was deemed appropriate.

EMG Correlates of Tension

Investigations of the relation of EMG to tension are reviewed by Basmajian (1974). A series of experiments (Lippold, 1952; Bigland, Lippold, and Wrench, 1953; Bigland and Lippold, 1954a, 1954b) have demonstrated that during a voluntary contraction the tension is proportional to the measurable electrical activity both under isometric and under isotonic conditions.

Kewley-Port (1974) discusses the relationship between the integrated EMG potentials and muscle tension with specific reference to the integration system utilized in this study. For a given subject, for a given electrode site, on a given experiment, the microvolt levels of the integrated EMG signal was felt to be a valid representation of the relative strength of muscle contraction (i.e., tension). These values (in microvolts) cannot be compared across subjects, across experiments, or even across electrode sites for the same subject on the same experiment. Such comparsions are impossible because the number of motor units sampled are not consistent across insertions. It is possible, however, to compare variations in microvolt values from the same site, during any given experiment, and thus draw conclusions about variations in muscle tension levels.

EMG Correlates of Tension in Stuttering

Most of the early EMG studies conducted with stutterers were designed to investigate basic neurophysiological differences between stutterers and nonstutterers (Travis, 1934; Morley, 1937; and Steer, 1937). They offer little information about muscle activity accompanying individual moments of stuttering.

More closely related to the present study is the work of Sheehan and Voas (1954). They investigated action potentials in the masseter muscle during moments of stuttering, reporting that muscular tension appears to build during stuttering, reaching its peak near the termination of the block.

Shrum (1967) specifically set out to study the effects of tension, especially during the prephonation period, through the use of electromyography. Using silver disk surface electrodes, he recorded from several sites including two (bilateral) masseter (jaw) muscle sites, two (bilateral) platysma (neck) muscle sites, and one leg muscle site. Ten stutterers and ten nonstutterers read a connected passage and a list of twenty-five words. When the stuttered words of the stutterers were compared with the normally spoken words of the nonstutterers, significant differences were found for the "duration of increased tension in the jaw, neck, and chest muscles before speaking."

The stutterers were compared with themselves on three conditions, (1) words spoken normally, (2) words on which they expected to stutter, and (3) words on which they stuttered. The "duration of increased tension" was shortest for the normally spoken words, longer for the words on which stuttering was expected, and longest for the stuttered words.

From these results Shrum (pgs.81-82) concludes that, "When the stutterers stuttered, they tensed significantly earlier before speaking than did nonstutterers," and "when the stutterers reported they 'expected' to stutter, they increased their duration of anticipatory tension over that of their normally spoken words, and when they stuttered, they markedly increased the duration of anticipatory tension over the lines when they 'expected' to stutter."

In elaborating on these conclusions, he states that, "They (stutterers) do seem to differ from nonstutterers when they stutter as to how soon in anticipation of talking they begin to tense, with the stutterers tending to tense earlier than the nonstutterers."

It would appear that Shrum's conclusion is only one of two possible interpretations of his data. The duration from point A (where muscle activity is increased over the resting state) to point B (where initiation of phonation was recorded) could be longer because the stutterer began to tense earlier (as concluded by Shrum); or it could be longer because initiation of phonation was delayed. The case for the second explanation is strengthened considerably by Agnello's (1974) findings of longer voice onset times in stutterers, and Adams and Hayden's (1974) finding that stutterers are significantly slower than nonstutterers in initiation of phonation.

Shrum also reports on the amplitude measurements taken from these muscle sites. He found that, "the stutterers resembled the nonstutterers even when the stutterers were stuttering." From these and other findings, Shrum concluded that: (1) "The stutterers, when they were not stuttering resembled the nonstutterers with respect to their levels of anticipatory tension, the muscle areas in which anticipatory tension first increased, the patterns of muscle tensions used before and during speaking, and the spread of muscle tension during speaking." and (2) "With the exception of a few individuals among the stutterers, the occurrence of stuttering, or the reported 'expected' stuttering, was not accompanied by changes in patterning, muscle areas where anticipatory tension first increased, or spread of muscle tension during speech."

Critique of Shrum's work suggests three possible conclusions:

(1) Stutterers, when stuttering, do not exhibit higher levels of muscle tension than they do when speaking fluently.

(2) The amplitude measurements used by Shrum were not sufficient-

ly sensitive to demonstrate these differences.

(3) The muscles whose activity was monitored are not the muscles in which critical tension occurs.

EMG Correlates of Coordination

In terms of muscle activity, "good coordination" occurs when muscles and muscle groups work together to produce the desired effects with a minimum of wasted effort. As described by Sherrington (1909), "reciprocal inhibition" facilitates coordinated movement by the agonist muscles through the relaxation of antagonist muscles. As demonstrated by Travill and Basmajian (1961) the antagonist in a muscle pair usually relaxes completely while the agonist is active. One exception to this rule is noted (Barnett and Harding, 1955; Basmajian, 1957 and 1959; Bierman and Ralston, 1965), at the end of a whip-like motion of a hinge joint, a sharp burst of activity occurs in some antagonists. This activity is interpreted as serving a protective function, to brake the force of the agonist and avoid damage to the joint (Basmajian, 1974). Within this framework, synergist muscles show simultaneous activity, and antagonist muscles are reciprocally active.

Exceptions to the normal pattern of reciprocal antagonist activity have been investigated. In general, these exceptions occur in motor acts which may be described as inefficient or "poorly coordinated." Specifically, co-contraction of antagonist muscles has been found to occur under the following conditions:

(1) In stress: Gellhorn (1947) found that with very strong static flexion of the wrist some activity appeared in the antagonist.

He reported three stages of recruitment varying directly with the stress; in the first two stages the synergists were recruited and in the third (with excessive straining) the antagonist was recruited. Patton and Mortensen (1970) found that increasing the load increased co-contraction of agonist and antagonist in both flexion and extension of the elbow. Hirshberg and Dasco (1953) reported simultaneous activity in agonists-antagonists in "strenuous motion or in tense experimental subjects."

(2) In rapid movements: Goabel and Bouisset (1966) studied low, middle and rapid rates of flexion and extension of the elbow and found partial overlapping of agonist and antagonist activity at high speeds.

(3) In unskilled or untrained subjects: Bratanova (1966) found that co-contraction was common in subjects who were in the early learning stages for performance of a motor task. Reciprocity of antagonist activity was characteristic of the same subjects' later (post-training) performance. Interestingly, Person (1958, 1965 and 1969) has demonstrated that relaxation (or tension) in an antagonist muscle can be trained to increase or decrease.

(4) In infants and young children: Fenges, Gergely and Toth (1960) studied flexion reflexes in normal young children and observed that both agonists and antagonists contract in what they term a "co-reflex phenomenon." Missiuro (1963) found a spread of electrical activity to other muscles of the same extremity in children. Gater (1967) found that as infants mature the excessive co-contraction typical of childhood diminishes progressively.

(5) In neurological impairment: Landau and Clare (1959) studied the "up-going toe" of upper motor neuron lesions and found it to be the result of an exuberant overflow of activity to the great toe extensors with co-contraction of the flexors. Kenney and Heaberlin (1962) studied locomotion in cerebral palsied children and reported an abrupt onset of agonist activity and a rapid response of the agonists with sufficient power to be obstructive.

(6) In nonrhythmic activities: Kozmyan (1965) demonstrated that the latency of antagonist inhibition with agonist excitation (resulting in co-contraction) varied most frequently during movements in response to nonrhythmic stimulation. With rhythmic repetitive movements, the latencies as well as dissociation of reciprocal inhibition is diminished. Thus, inhibition of the antagonist muscles was to be expected in rhythmic activity with any element of supraspinal control or learning.

NOTES

1. Dorland's (1974) defines spasm as "1. a sudden, violent, involuntary contraction of a muscle or a group of muscles, attended by pain and interference with function, producing involuntary movement and distortion. 2. a sudden but transitory constriction of a passage, canal, or orifice." They define a phonatory spasm as a spasm of the tensors of the vocal bands, and a glottic spasm as a laryngospasm. In a separate entry Laryngospasm is defined as a spasmodic closure of the larynx.

2. See Freeman, Ushijima, and Hirose (1975) and Schwartz (1975a) for a discussion of the ADR theory. Schwartz (1975b) has modified his earlier theory, giving a less prominent role to the ADR, and placing more emphasis on adductive laryngeal response to various kinds of stress.

3. There are however marked differences of opinion as to why stuttering is reduced by the "fluency-evoking conditions". Distraction has long been discussed as a key factor in these conditions.

4. McGlone and Shipp (1971) studied fry phonation, reporting high levels of TA and LCA activity with low levels of CT activity. Freeman (1974) investigated EMG correlates of fry phonation in one stuttering subject, reporting high levels of TA and LCA activity and low levels of CT activity. Implications relating to stuttering were discussed.

5. Oscillograms of the stuttered speech of the subjects of the present study showed patterns like those reported by Webster (1975).

6. Blurring of the images in frames three and four results from a change in the distance between the tip of the scope and the vocal folds, as the larynx is raised during the tight closure of the block.

7. Dixit and MacNeilage (1974) have reported evidence of CT activity in the production of segmental distinctions between members of the complex stop categories of Hindi.

8. Technically, the PCA is a glottal dialator, since relaxation of the adductor forces results in opening the glottis. There is also evidence that lowering the larynx will tend to abduct the vocal folds (Kirchner, 1975).

9. In recent studies of Danish, Fischer-Jørgensen and Hirose (1974a and 1974b) reported results on the LCA and TA which were not

consistent with the patterns previously observed in English and Japanese subjects. They state that they are not sure whether these inconsistencies relate to language differences or speaker differences. However, the four subjects of the present study (when not stuttering) showed TA and LCA patterns consistent with those reported by Hirose and his collegues for other speakers of English.

CHAPTER II

METHODS AND INSTRUMENTATION

Electromyographic Experimental Procedures

The development and current status of electromyographic techniques in physiological research are reviewed by Basmajian (1974). The philosophical considerations, objectives, merits, limitations, and technological problems in the application of electromyographic techniques to research on speech production are discussed by MacNeilage and Sholes (1964), Cooper (1965), and Harris (1970).

The electrodes and insertion techniques employed are essentially those described by Hirose (1971a).

Electrode Preparation

With one exception,[1] hooked-wire electrodes were used for the recordings. The wire employed was a 0.002 inch diameter platinum (90%)-iridium (10%) alloy with isomel polyester coating (Consolidated Reactive Metals, P-91).

The electrodes were made in essentially the same manner described by Basmajian and Stecko (1962) and Hirano and Ohala (1969). The free ends of a length of wire sufficient to serve as a pair of electrodes were threaded through a hypodermic needle and pulled through the needle until only a small loop of wire remained at the tip of the needle. This loop was cut, leaving two short hooks.

For the peroral insertions to the posterior cricoarytenoid and interarytenoid muscles a specially designed probe was used. Illustrated in Fig. 2, this probe consists of an L-shaped metal rod with the shaft of a 26-gauge needle cut and epoxy-bonded to the end of its shorter arm. The free ends of the wire were threaded through the needle carrier and through the thin polyethylene tube bonded along the rod.

General Procedures

In order to inhibit salivation, seven to ten drops of tincture of belladonna were administered by mouth prior to the beginning of the experiment. For the peroral insertions, a topical anesthesia (Cetacaine[2] spray) was administered to the pharynx and larynx. This was followed by a gargle or instillation of 2-3 ml. of 2% Xylocaine.[3] The percutaneous insertions were preceded by a topical administration of 2% Xylocaine without epinephrine through a Panjet-70 airjet (Panray)[4] at the site of the needle insertion.

During the insertion and verification procedures, an oscilloscope and an amplifier speaker system were used for monitoring the pertinent muscle activity. After insertion the electrode-bearing needle was withdrawn leaving the electrodes hooked into the target muscle.

The external wires were taped to the skin to make sure an accidental pull did not inadvertently dislodge them. The recordings were always made with the subject in an upright sitting position.

The correct placement of an electrode in a specified muscle

Figure 2

An L-shaped probe used for peroral insertion of the hooked-wire electrodes into the posterior cricoarytenoid and the interarytenoid. The shaft of a 26-gauge hypodermic needle is epox-bonded to the shorter arm. This illustration is from Hirose (1971) pg. 74.

was verified by monitoring the activity occurring during performance of specified gestures and/or maneuvers. The difficulties in this type of verification procedure were discussed by Shipp, Deatsch, and Robertson (1968), Harris (1970), and Hirose (1971a). Principle among the problems cited was the lack of normative EMG data for the phonatory-articulatory muscles. The rapidly expanding body of research applicable to the function of the muscles in question -- Fink, Basek, and Epanchin (1956), Faaborg-Anderson (1957), Bowden and Scheuer (1960), Rattenborg (1961), Hirano and Ohala (1969), Suzuki and Kirchner (1969), Hirano, Ohala and Vernard (1970), Shipp and McGlone (1971), McGlone and Shipp (1971), Hirose (1971b), Gay, Strome, Hirose, and Sawashima (1972), Murakami and Kirchner (1972), Hirose and Gay (1972 and 1973), Fukuda, Sasaki, and Kirchner (1973), Atkinson (1973), Hirose (1974), Hirose and Ushijima (1974), Collier (1975), and Erickson (1976) -- has provided information of value in defining verification procedures. However, the intra- and inter-individual ranges of variation in muscle function are not established, and must be considered.

The maneuvers used for insertion verification may be divided into two categories:

(1) Physiological (nonspeech) maneuvers:
    a. respiration
    b. swallowing
    c. coughing
    d. gagging
    e. breath-holding
    f. chewing
    g. jaw opening and closing

h. head movements
i. lip smacking
j. tongue protrusion
k. smiling
l. laughing

(2) Phonatory (speech-like) or speech maneuvers

a. sustained phonation
b. sustained fry phonation
c. intermittent phonation [a--a--a--a]
d. scale singing
e. high pitch (falsetto) and low pitch (chest) contrasts
f. series of glottal stops [ʔaʔaʔaʔa]
g. series of boiced stops [babababababa]
h. series of voiceless stops [papapapapapapa]
i. series of glottal fricatives [hahahahahahaha]
j. sustained voiced fricative
k. sustained voiceless fricative
l. whispering
m. speaking loudly and speaking softly
n. stressed and unstressed syllables [əpʌ]

The physiological (nonspeech) maneuvers are in some respects preferable for verification judgments, since, with the exception of laughing and smiling, they can be studied in nonhuman species. Table 1 lists the critical test maneuvers used, and presents a profile of the anticipated activity patterns against which each insertion was verified.

A two-step verification method was utilized. First, after each insertion, the oscilloscope and amplifier-speaker systems were utilized for monitoring the pertinent muscle activity. If the observed patterns differed from the anticipated, appropriate patterns, the electrode was removed and a new insertion was made for that muscle. Secondly, recordings were made as the subject performed the critical maneuvers. Using the recordings, final verification was based upon examination of the simultaneous activity patterns from each insertion site. In most

TABLE 1

Summary of activities used in verification of electrode placement for laryngeal muscle insertions.

											Speech Activities		
MUSCLE	Inspiration	Swallowing	Breath holding	Phonation	Fry phonation	Ascending scale	Descending scale	Jaw opening (resistance)	Head raising (supine)	[h a]	[p a]	[b a]	[? a]
PCA	+x	-#	-	-	-	-*	*-			+ -	+ -	- -	- -
INT	-x	+x	+	+	+	++	++			- +	- +	+ +	+ +
LCA	-	+x	+	+	+	++	++			- +	- +	- +	x++
TA	-	+x	+	+	+x	++	++			- +	- +	- +	+ +
CT	-	-	-		-x	-+x	+-x	-x	-x				
SH						**x	**x	+x	+x				

\+ indicates relatively higher levels of activity

\- indicates relatively lower levels of activity or suppression

x indicates a particularly characteristic pattern of activity

# indicates that the maneuver calls for suppression followed by activity

* indicates that at the upper extremes of the subject's singing range activity may occur.

** indicates that activity occurs only at the upper and lower extremes of the subject's singing range.

cases, each maneuver was recorded at the beginning of the experimental session, and again at the end. By comparing the initial and final recordings, the experimenter sought to detect changes in the muscle action patterns indicative of significant movement of the electrodes during the experiment.

The verification techniques, as described thus far have been developed and used in the Haskins Laboratories electromyographic research on normal speech production (Hirose, 1971a,b; Gay, Strome, Hirose, and Sawashima, 1972; Hirose and Gay, 1972; Gay and Hirose, 1973; Hirose and Gay, 1972; Hirose, 1974; Hirose and Ushijima, 1974; Atkinson, 1973; Collier, 1975; and Erickson, 1976).

Special verification issues are introduced by an investigation of pathological speech production. Although deviation from normal activity patterns was anticipated as a possible component of the moment of stuttering, it was also possible that "abnormal" or "atypical" activity patterns might occur (1) during the physiological (nonspeech) activities, (2) during speech-like phonatory activities and/or (3) during the nonstuttered speech activities. This problem was essentially ignored during the first phase of verification. Standards for verification were the same as those used for normals, and the electrodes were removed if appropriate activity patterns did not occur.

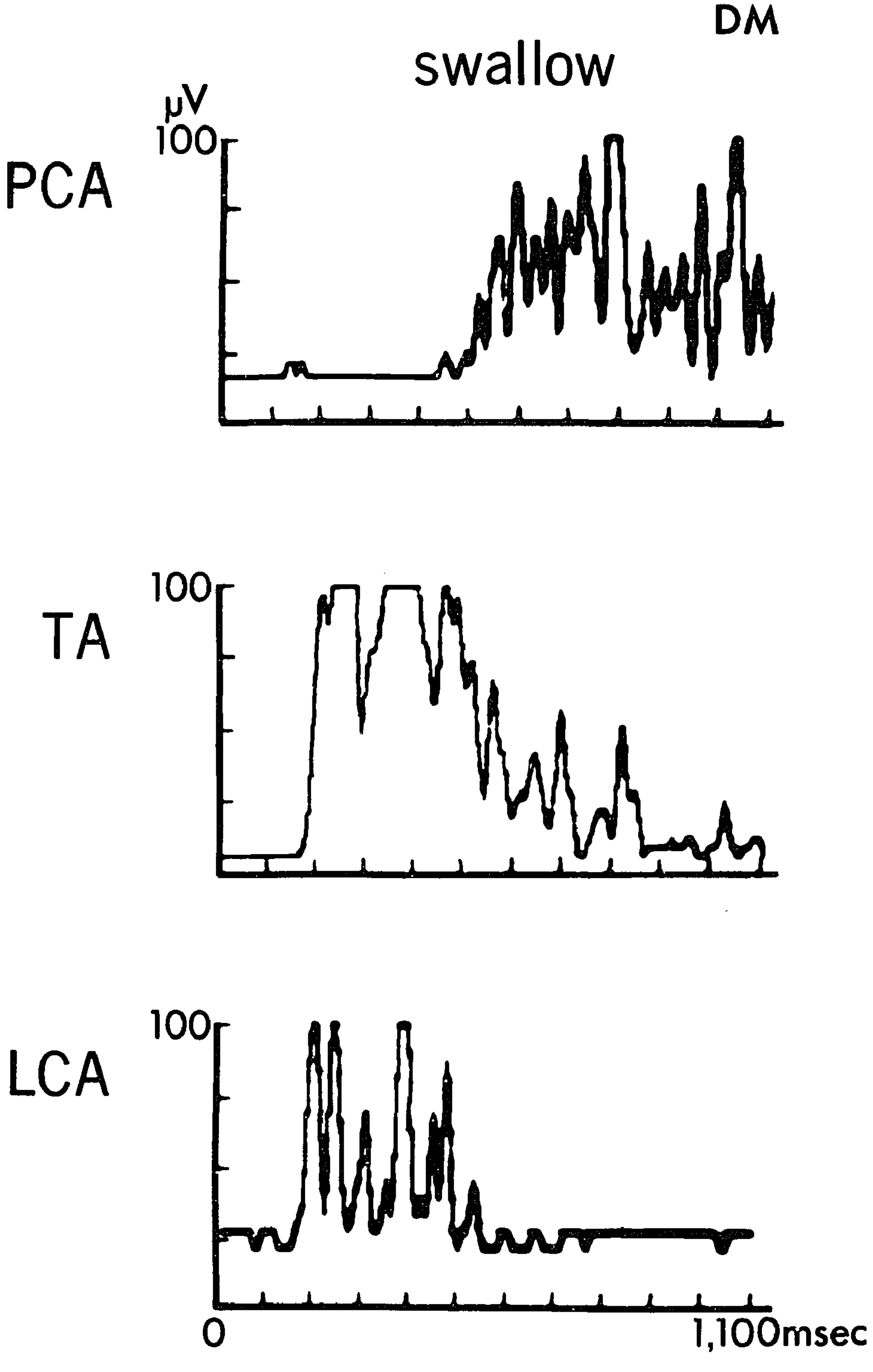
During the second step in verification--examination of the recorded pattern--the possible occurrence of atypical patterns during nonspeech maneuvers was considered. Therefore, multiple tokens of each nonspeech maneuver were examined, and the insertion verified if most, but not necessarily all, of the tokens demonstrated appropriate patterns. When a recording from one site was very similar to the pattern from another insertion site, special care was devoted to finding examples of nonspeech maneuvers which differentiated between the two sites in question. For example, when it was found that during stuttering moments for subject D.M., the activity patterns from the LCA and PCA were very similar, the nonspeech activity recordings were carefully re-examined. When recordings of inspiration showed high levels of PCA activity and no activity in the LCA, it was determined that the LCA was not contaminated by the PCA. When recordings of swallow (Fig. 3) showed high levels of LCA activity, with suppression of the PCA, it was determined that the PCA was not contaminated by the LCA. In these cases, verification was based on demonstrated differentiation between the two sites in question. Figure 3, which shows the rectified integrated EMG tracing for three laryngeal muscles during swallow (subject D.M.), illustrates the normal pattern of adductive activity for protective closure; and as such exemplifies recordings used for verification. If an insertion site could not be verified according to the criteria described in this section, the recordings from that insertion site were excluded from the body of data reported in the results.

A 300 microvolt calibration signal was recorded at regular intervals during the experiment. These were compared in order to verify the reliability of the recording and playback equipment.

The raw EMG tracings were examined visually in order to check

Figure 3

Electromyographic recording of a swallow as performed by subject D. M.


DM
swallow
µV
100
PCA
100
TA
100
LCA
0
1,100msec

for (1) changes in the level of recording from any given muscle and (2) the presence of movement artifacts. Some changes in recording levels were found to occur during the first few minutes of a given experimental session. In these cases, data recorded prior to the change was not included in the final analysis of results. One muscle (the CT for subject P.N.) showed a recording level change between experimental conditions (about half-way through the experiment), and was subsequently excluded from the data analysis. Recordings which contained nonphysiological artifacts were excluded in whole or in part from the data analysis. Fortunately, this was not a problem in most of the recordings.

Table 2 summarizes the insertions attempted with each subject during each experiment and reports the success rate in achieving verifiable quality recordings from each muscle.

Procedures for Each Muscle

Posterior Cricoarytenoid (PCA)

The insertions into the PCA were made perorally by indirect laryngoscopy using the L-shaped carrier (Fig. 2). Using this approach the needle is inserted parallel to the alignment of the muscle fibers. During the insertion the subject sat in an upright position and phonated a sustained vowel in order to open the hypopharyngeal lumen for easier access to the site of insertion. The insertion ,performed under inspection with a laryngeal mirror, was made into the belly of the muscle on the cricoid cartilage through the hypopharyngeal mucosa (Fig. 4). If the insertion is placed too far cranially there is spatially a

TABLE 2

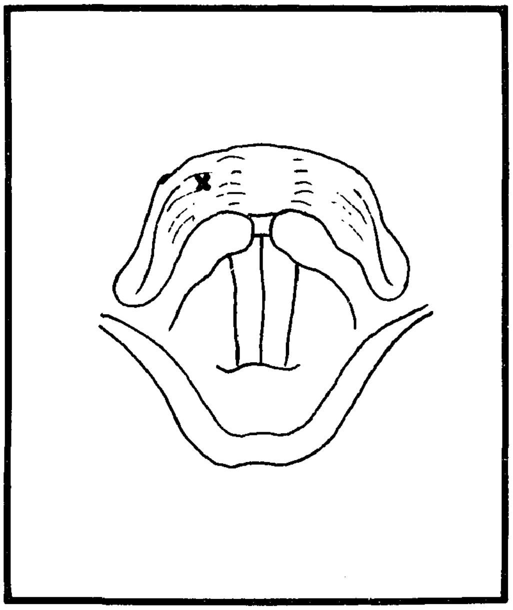
Verified insertions for each subject and for each muscle over the series of experiments.

SUBJECT	Laryngeal Muscles						Upper Tract Articulators				TOTALS*		
	PCA	INT	LCA	TA	CT	SH	IL	SL	GG	OO	Laryn-geals	Upper Tracts	Combined
D.M.	X		X	X			X	X	X	X	3	4	7
P.N.		X	X	X					X	X	3	2	5
G.G		X	X	X	X	X		X		X	5	2	7
C.D.	X	X		X				X		X	3	2	5
TOTALS	2	3	3	4	1	1	1	3	2	4	14	10	24

* The Haskins Laboratories multichannel electromyographic recording and processing system provides for simultaneous processing of recordings from eight channels. In all cases eight insertions were attempted. For this series of experiments 32 insertions were attempted, and of these 24 resulted in successful, verifiable recordings. Two PCA insertions and one INT insertion were impossible because of the Subjects' anatomy and gag reflexes; one GG, one LCA, and three CT recordings were rejected because (1) they could not be verified, or (2) they did not result in good quality recordings, or (3) they exhibited evidence of movement artifacts.

Figure 4

A diagrammatic view of the larynx during sustained phonation by indirect laryngoscopy. A cross (x) indicates one point of needle insertion into the posterior cricoarytenoid. This illustration is from Hirose (1971a) pg.77.


x

possibility of contamination by the interarytenoids. Since the activity patterns of these muscles are quite distinct, verification by functional differentiation is possible, by having the subject repeat short periods of vowel phonation interspersed with deep inspiration. The insertion was accepted if the muscle showed activity during inspiration and suppression during phonation. Studies of both animals and humans agree in reporting increased PCA activity for inspiration; while studies of speech are equally consistent in reporting PCA suppression and INT activity for vowel phonation (see Chapter 1).

Interarytenoid (INT)

The insertions into the INT were made perorally, employing the same technique and equipment used in the PCA insertions. The insertion was made at the midline between the two arytenoid prominences (Fig. 5).

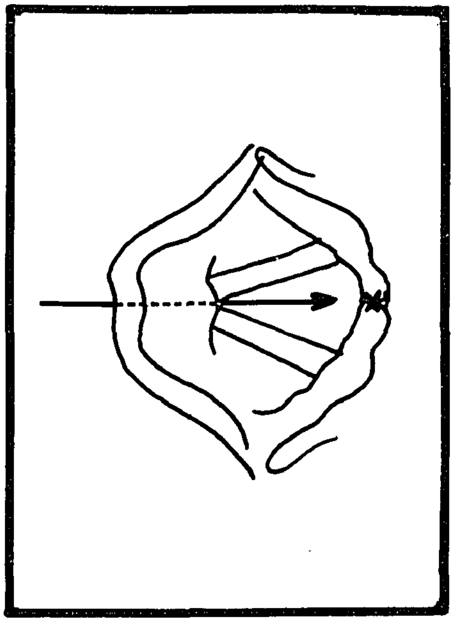
Spatially, the only possible contaminant is the PCA. The placement was verified if the muscle was active during phonation and suppressed during inspiration. This pattern of reciprocity with the PCA has been found to be very characteristic for the INT (see Chapter 1).

Cricothyroid (CT)

Percutaneous insertion into the cricothyroid was made at a point above the cricoid ring, and approximately 1 cm lateral to the midline. With the subject in the supine position, the needle was directed posteriolaterally and slightly superiorly, aiming at the lower edge of the thyroid lamina. Using this insertion technique it is possible to have a misplacement of the electrode either in the

Figure 5

A diagrammatic view of the larynx during quiet respiration by indirect laryngoscopy. An arrow indicates the direction of a needle inserted percutaneously into the subglottal space towards the interarytenoid area. A cross indicates one point of needle insertion into the interarytenoid by the peroral route. This illustration is from Hirose (1971a) pg. 79.



lateral cricoarytenoid (LCA) if the placement is too deep, or in the sternohyoid (SH) if the insertion is too superficial.

Verification required the performance of a series of activities including: (1) singing a scale, (2) breath-holding, (3) swallowing, (4) series of glottal stops, (5) jaw opening against resistance, and (6) raising the head. The insertion was accepted if the muscle showed marked activity for singing an ascending scale, but relatively low levels of activity for breath-holding, swallowing, glottal stop production, jaw opening, and head raising. Although the LCA and SH may show activity changes with pitch changes, the LCA and SH will not show the clear linear relationship to pitch change characteristic of the CT. Differentiation between LCA and CT can be made during swallow and breath-holding. Both of these maneuvers are accompanied by marked LCA (and TA) activity, while CT activity is suppressed. The same pattern holds for "fry" phonation (McGlone and Shipp, 1971). During jaw opening against resistance, the laryngeal strap muscles (including the SH) show marked activity, while the CT shows no characteristic pattern of involvement.

Thyroarytenoid (TA)[5]

The insertions into the TA were made percutaneously, with the subject in a supine position attempting sustained phonation. The skin was pierced at a point close to the midline at the level of the cricothyroid space. The needle was then directed cranially and slightly laterally, penetrating the cricothyroid membrane to reach the muscle from its inferior surface (Fig. 5). The needle does not pass through the subglottal space, but rather through the submucous tissues near the anterior commissure. Three possible sources of contamination must be considered. First, if the insertion is not deep enough CT contamination is possible; second, if the insertion is too lateral LCA contamination is possible; and finally, if the insertion is too medial, contamination by vibration of the vocal folds is possible.

Verification was made by asking the subject to (1) sing a scale, (2) produce sustained fry phonation, (3) swallow, (4) alternate phonation and inspiration. The insertion was accepted if the muscle showed activity for the scale, the fry, the swallow, and the phonation, with suppression during inspiration. Swallow and fry phonation differentiate between the CT and the TA. Differentiation between LCA and TA is more difficult, since these two vocal fold adductors show similar patterns for many maneuvers. Both are active for glottal stop, but the work of Gay and Hirose (1973) found earlier and stronger activity for the LCA. Both are active for fry phonation, but McGlone and Shipp (1971) show slightly stronger TA activity for this maneuver. Both show higher levels of activity for high pitches and lower levels for low pitches, but the TA activity pattern (similar to that of the CT) shows a more linear relationship to pitch in scale singing. No verification technique presently known will clearly and consistently distinguish between the LCA and TA insertions. This problem is acknowledged, and considered in interpretation of the data.

Movement artifacts related to cord vibration are recognizable by their regularity and periodicity. If these occurred during normal phonation, as monitored on the oscilloscope, the electrode was removed and a new insertion was made. However, examination of the recordings showed one subject (P.N.) whose high intensity (very loud) utterances resulted in TA recordings which were slightly contaminated by vibratory artifacts. These occurred rarely, and were essentially ignored in later data analysis.

Lateral Cricoarytenoid (LCA)

Insertions into the LCA were made at almost the same point as the CT insertions. In this procedure, however, the needle was directed laterally and slightly cranially penetrating the cricothyroid membrane at a point anterior to the inferior tuberculum of the thyroid cartilage and deeply enough to reach the LCA (Fig. 6). Possible contamination from the TA was minimized by keeping the direction of the insertion lateral and less cranial than the TA insertion, and following the contour of the cricoid ring.

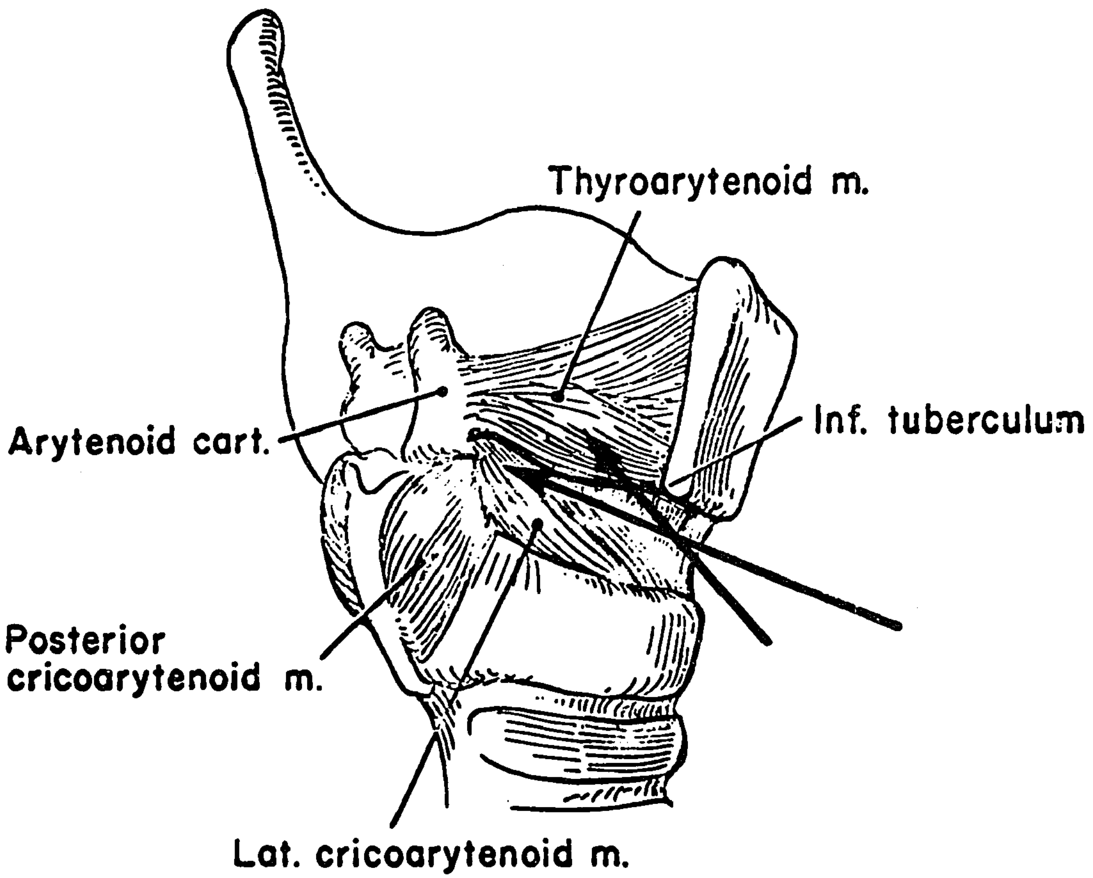
Verification was made by having the subject (1) hold his breath, (2) swallow, (3) produced glottal stops[ʔaʔaʔaʔa], and (4) sing a scale. The insertion was accepted if the muscle showed marked activity for the first three maneuvers. Other details related to functional differentiation of the LCA from the CT and the TA have already been discussed.

Sternohyoid (SH)

The insertion into the SH was made at the level of the thyroid

Figure 6

A diagrammatic view of the larynx with the right thyro ala removed. Arrows indicate the direction of needle insertion into the thyroarytenoid (A) and into the lateral cricoarytenoid (B). This figure, a modification of Hirano and Ohala, (1969) is from Hirose (1971b) pg. 81.


Thyroarytenoid m.
Arytenoid cart.
Inf. tuberculum
Posterior cricoarytenoid m.
Lat. cricoarytenoid m.

lamina. The muscle was palpated by placing the subject in a supine position and asking him to raise his head. As the subject raised his head from the supine position, the needle was inserted lateral to the midline parallel to the alignment of the muscle fibers. It is possible for this insertion to be contaminated by other strap muscles or by the CT. However, none of the arguments made in this thesis rest on differentiation of the laryngeal strap muscles. Since SH activity occurs only at the upper and lower extremes of the subject's singing range, while the CT shows gradual increases in activity throughout an ascending scale, the singing of ascending and descending scales was used in addition to the differentiation procedures already described on the CT.

Genioglossus (GG)

Insertions to the GG followed a percutaneous approach. The needle was inserted perpendicularly to the surface of the skin at the midpoint between the hyoid bone and the mandibular ridge in the paramedial line. The needle was then directed deep enough to be palpated by the physician's finger inserted onto the floor of the mouth of the subject.

Verification activities included (1) tongue protrusion and (2) swallowing. The insertion was accepted if vigorous muscle activity accompanied both of these maneuvers. When these procedures are followed, contamination is not felt to be a major problem.

Superior Longitudinal (SL) and
Inferior Longitudinal (IL)

Insertion into the SL was made to the upper surface of the anterior 1/4 of the tongue, approximately 2 cm from the tip and 1 cm lateral to the midline. The needle was directed obliquely to the surface and the tip was kept superficial so that the electrodes were left immediately underneath the superior surface of the tongue. The insertion was accepted if activity accompanied (1) tongue protrusion, and (2) production of the interdental fricatives [θaθa] and [ðaða]. If this insertion is too deep the possibility of contamination exists, because the fibers of other intrinsic muscles and the GG fibers intermingle with those of the superior longitudinal (Miyawaki, 1974).

For the inferior longitudinal, insertion was made into the lower surface of the anterior 1/3 of the tongue about 1 cm from the lateral edge. The needle was directed superficially in order to place the electrodes close to the mucosal surface. The insertion was accepted if the muscle was active for (1) tongue protrusion, (2) production of [θ] and (3) production of retroflex consonants [l] and [r]. Possibility exists for contamination from other muscles including the anterior portions of the styloglossus and the hyoglossus (Miyawaki, 1974).

To a large extent, both of these insertions rely on anatomical expectation, and the possibilities of contamination cannot be ruled out. However, for the purposes of the present research differentiation of the intrinsic tongue muscles was not crucial. The muscles reported in this investigation as the SL and the IL were tongue muscles anatomically defined by the sites of insertion, and showing activity for (1) tongue tip protrusion, (2) production of interdental fricatives, (3) production of retroflex consonants. Post-experimental examination of the recordings regealed that these muscles were active for the following English sounds: [θ], [ð], [s], [z], [l], [r], [t], [d], [n], [tʃ]. They showed no consistent pattern of activity in relation to vowel production.

Orbicularis Oris (OO)

The insertion techniques for the hooked-wire electrode recordings from the OO are described in Fischer-Jørgensen and Hirose (1974). For all subjects recordings were made from the orbicularis oris superior. Insertion into the OO was made at the vermilion border of the upper lip about 1 cm lateral to the midline. This is approximately at the position described by Leanderson (1972). For subject C.D. the paint-on electrode was applied to this same point (see footnote 1 of this chapter).

The insertion was accepted if EMG activity was found for (1) protrusion of the lips and (2) production of labial stop consonants.

Subjects

The subjects for the experiments were four adult males--D.M., P.N., G.G., and C.D. They were selected because they were anatomically suitable for laryngeal electromyography; and they were willing to undergo the procedures required for the experiments. The subjects used were the first four suitable individuals located.

G.G. and C.D. were considered mild to moderate stutterers, while D.M. and P.N. were considered severe.[6] In age they ranged from 22 to 47. All had begun to stutter in early childhood and each had received some form of therapy. P.N. and G.G. were currently in therapy.

All four were relatively free of conspicuous secondary mannerisms involving non-articulatory muscles (i.e., head jerks, strong grimaces or foot stamping).[7] P.N. and G.G. occasionally used lip smacking and/or tongue clicks, which are audible on the tapes.

D.M. and P.N. reported that they were seldom completely fluent. G.G. reported times when he was relatively fluent, and other times when he was able to control so well that listeners were not aware of his blocks. C.D. reported periods of freedom from stuttering. Further, he reported that he was fluent when reading aloud or performing, as in acting or reciting poetry. He was aware of specific words or phoneme combinations which were likely to precipitate blocks.

Procedures for Securing the Desired Behavioral Samples

The basic design of the study required that comparable fluent and stuttered tokens be obtained from each subject. Since stuttering is a behavior known to be highly variable, the experimental procedures were necessarily flexible.

In each case the subject repeatedly read aloud the "Quotations on Science" passage (Appendix A). The passage was felt to constitute a moderately difficult oral reading task for educated adults. D.M. read the entire passage; G.G. read paragraphs one, two and three; and P.N. and C.D. read only paragraphs one and two.

The repeated readings were used to promote adaptation and thus provide fluent tokens for comparison with stuttered tokens. The frequency and consistency of stuttering behaviors in subjects D.M., P.N. and G.G., made securing fluent tokens the primary problem. Conversely, C.D. had few audible blocks during the oral readings. In his case, special strategies were necessary in order to secure stuttered tokens.

Initially, two fluency evoking conditions (choral reading and rhythm reading) were used in order to secure samples of fluent utterance. When, in the course of the experiments, it became evident that electromyographic investigation might yield valuable information about the nature of the fluency evoking conditions, three additional conditions--

whispering white noise masking, and delayed auditory feedback (DAF)--were included in the data collection procedures. D.M. was recorded under the choral reading and rhythm reading conditions; G.G. was recorded under the choral reading, rhythm reading, and whispering conditions; P.N. was recorded under the choral reading, rhythm reading, white noise masking, and DAF conditions; C.D. was recorded under choral reading, rhythm speaking, white noise masking, and DAF conditions.

D.M. was very consistent in his stuttering pattern, and showed only minimal adaptation over the repeated readings of the passage. He was therefore asked to repeat single words from a list composed of the words on which he had consistently stuttered during the passage readings. In this way additional samples of fluent utterance were elicited.

C.D. did not have audible blocks while reading the experimental passage. He suggested that he would stutter if he tried to use certain words which were currently giving him difficulty. He therefore engaged in conversation with the experimenter, making frequent use of feared, "difficult" words. A majority of his blocks occurred on the words "syllable" and "syllables." Sentences in which blocks occurred were written down and C.D. was later asked to repeat these sentences; thus providing some opportunity for adaptation. In the choral reading condition the experimenter and C.D. read a list of these sentences. The recordings made under the other fluency evoking conditions consisted of samples of spontaneous conversation and repetitions of sentences in which the blocks occurred.

Data Treatment and Analysis

Description and Identification of Moments of Stuttering

Three experienced speech pathologists listened to audio tapes of the subjects' readings of the first paragraph of the "Quotations on Science Passage" (Appendix A). Following the directions, and using the forms shown in Appendix B, they identified and described the dysfluencies they detected in these readings. If two of the three listeners agreed on the occurrence of a given stuttering block, the block was counted in the frequency of dysfluencies computation reported in Chapter 3.

Tokens of stuttered and fluent utterances taken from the body of data not considered by the listeners (i.e., material other than that contained in the selected paragraph) were handled in a different manner. These were categorized by the experimenter as:

(1) clearly stuttered, because (a) there is an audible repetition of a part-word, (b) there is an audible prolongation of a phoneme, or (c) there is a long pause in a linguistically inappropriate place;

(2) audibly fluent (i.e., without audible evidence of blocking);

(3) ambiguous, neither clearly stuttered nor clearly fluent.

In comparisons of stuttered and fluent utterances only the clearly stuttered and audibly fluent categories were considered; the ambiguous tokens were excluded. When oscillographic tracings were made of the audio recordings of the utterances judged stuttered, the repetitions, prolongations, and pauses could be identified visually. This finding supported to some extent, the validity of the single listener judgments.

Collection and Processing of EMG Data

The data was collected and processed using the Haskins Laboratories EMG system (Fig. 7), described by Port (1971), Kewley-Port (1973 and 1974), and Bell-Berti (1973). The present study differs in one critical aspect from most of the previous research conducted using this system. The integrated EMG data presented in this study all represent single token utterances, rather than multiple token averages of a given utterance. All data were processed using the linear integration system described by Kewley-Port (1973) and an integration time of 35 msec (Kewley-Port, 1974).

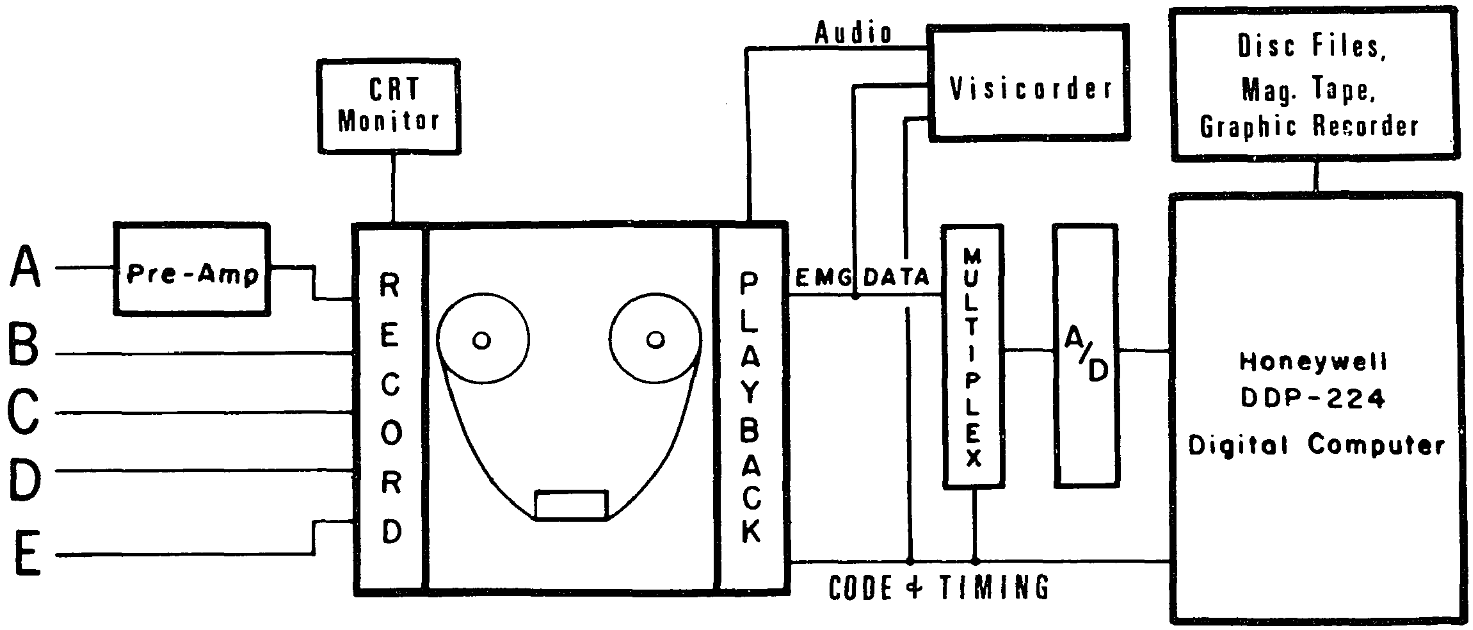
A slightly modified version of the E$MGSUMS program (Bell-Berti, 1973) was used to compute the average activity in each channel for segments which were two seconds in length.

The correlation program (E$MGCORL) described by Kewley-Port (1973) was used to calculate the correlation between the activity in the PCA and the activity in the INT for subject C.D.

Additional details relating to data processing are included with the presentation of the results in Chapter 3.

Figure 7

The Haskins Laboratories' EMG system as described by Port (1971) and by Bell-Berti (1973).



A - EMG (8 Channels)

B - Air Pressure (2)

C - Voice

D - Banter

E - Digital Code & Timing

NOTES

1. For subject C. D., paint-on, surface electrodes were used in recording activity from the orbicularis oris. SC20 electromedical silver skin contact paint (Micro-Circuits Co., INC.) was utilized. Comparison runs using the paint-on electrodes and hook wires showed no significant difference for the orbicularis oris. This conclusion was supported by Allen and Lubker, 1972. These electrodes were chosen because their use was less painful for the subject.

2. Cetacaine (trade name) is packaged in a 50 ml. aerosol bottle and contains the following: ethyl aminobenzoate, 14%; butylaminobenzoate, 2%; benzalkonium chloride, 0.5%; cetyldimethylethyl ammonium bromide, 0.005%. A one-second spray releases 0.1 ml. of solution, and usually three to four seconds of spray are needed to anesthetize the oral and pharyngeal mucosa (Gaskil and Gillies, 1966).

3. Shipp, Deatsch, Robertson (1968) found no discernible effect of topical anesthesia on normal laryngeal behavior.

4. Panjet-70 delivers approximately 0.1 ml. of the anesthetic solution to a circumscribed intradermal depth up to 6 mm penetration.

5. The muscle labeled thyroarytenoid (TA) in the present research is the same muscle as that referred to by Hirose as the thyrovocalis (VOC).

6. The categories of mild, moderate, and severe used to describe the subjects' dysfluency problems were based on the observations and judgments of the researcher, the clinicians who had been or were currently working with the subjects, and the physicians and speech pathologists who were present during the experiments.

7. The absence of widespread muscular involvement was fortuitous since gross movements of the head or neck might have created movement artifacts or even have dislodged the electrodes.

CHAPTER III

RESULTS

Overview of Results

The presentation of the results is divided into four topic areas. The first section presents data from the analysis of the audio recordings of subjects D.M., P.N. and G.G. (C.D., who did not stutter while reading the passage, is not included in this analysis.). The principal findings of this section--(1) stuttering frequency is reduced by fluency evoking conditions and (2) utterance rate increases as stuttering decreases--are not new, having been reported previously by a number of investigators. The information in this section is included primarily because of its value in evaluating other results of the study.

Section two, the description of types of dysfluencies, is also included because it aids in interpretation of other results which relate to the individual subjects.

Sections three and four present the results of the EMG experiments as they relate to levels of muscle activity and to coordination of muscle action. Presentation of EMG results are not parallel for all subjects because of (1) differences in the successful placements of electrodes (Table 2) and (2) differences in the way comparable fluent and stuttered utterances were secured (see Procedures for Securing the Desired Behavioral Samples, Chapter II).

In section three, level differences for passage readings are compared for D. M., P. N. and G. G.; while differences in peaks of activity are compared for C. D.

Section four reports results on only the two subjects (D. M. and C. C.) for whom PCA (abductor) recordings were secured. Examples typical of the stuttered and fluent utterances of these two subjects are used to illustrate EMG evidence of disrupted abductor-adductor reciprocity. A correlation study of abductor-adductor reciprocity was possible for only one subject, C. D., because:

1. Of the adductor group (TA, INT and LCA) only the INT is a "pure" adductor (see Chapter I) and shows unequivocal reciprocity with the PCA. The other adductors (TA and LCA) are always suppressed during PCA activation, but they are not always active when the PCA is suppressed. C. D. is the only subject for whom both PCA and INT recordings were secured.
2. Statistical treatment of the correlation data required
    (a) multiple stuttered and multiple fluent utterances of the same or very similar words, and
    (b) A word or words whose phonetic content included an initial syllable in which a shift from voicelessness to voicing occurred. C. D.'s 49 utterances of the words "syllable" and "syllables" comprised the only body of suitable data.

Utterance Rate and Frequency of Dysfluencies

Utterance Rate

Calculation

Oscillographic tracings of the audio recordings of each of the readings of the sixty-six syllable passages contained in Appendix A were prepared and measured for three subjects (D.M., P.N. and G.G). Utterance time measured from the onset of voicing for the first word, "the" to the cessation of frication for the last word, "ignorance," was divided by the number of sullables (66), to yield a ratio of syllables per second.

Findings

Utterance rates, in syllables per second, are presented in the first column of Table 3. The subjects' rates for the first readings range from 2.82 to 6.65 syllables per second; while their rates for the choral readings ranged from 5.64 to 8.57. The three rates for the rhythm readings were very similar (5.12, 5.65, 5.68), suggesting that the experimenter who set the rhythm was highly consistent across experiments.

Frequency of Dysfluencies

Calculation

Stuttering blocks were identified from the audio recordings

TABLE 3

Utterance rate (expressed as syllables per second) and frequency of dysfluencies (expressed as percent of syllables stuttered).

SUBJECT	CONDITION	SYLLABLES PER SECOND	% SYLLABLES STUTTERED
D.M.	Reading #1	2.82	23%
	Choral Reading	5.64	2%
	Rhythm Reading	5.65	0%
G.G.	Reading #1	4.54	20%
	Rhythm Reading	5.68	0%
	Choral Reading	8.57	2%
P.N.	Rhythm Reading	5.12	2%
	Reading #1	6.65	18%
	Reading #2	7.15	17%
	Choral Reading	7.36	2%
	White Noise	7.92	5%
	DAF Reading	8.93	3%

following procedures outlined in Appendix B, and discussed in Chapter 2. Each block was identified with one syllable, which was counted as a stuttered syllable. Only one block was counted for any syllable. Silent blocks were assigned to the syllable immediately following the pause. The number of stuttered syllables was then divided by the total number of syllables in the passage (66), yielding a percentage of stuttered syllables.

Findings

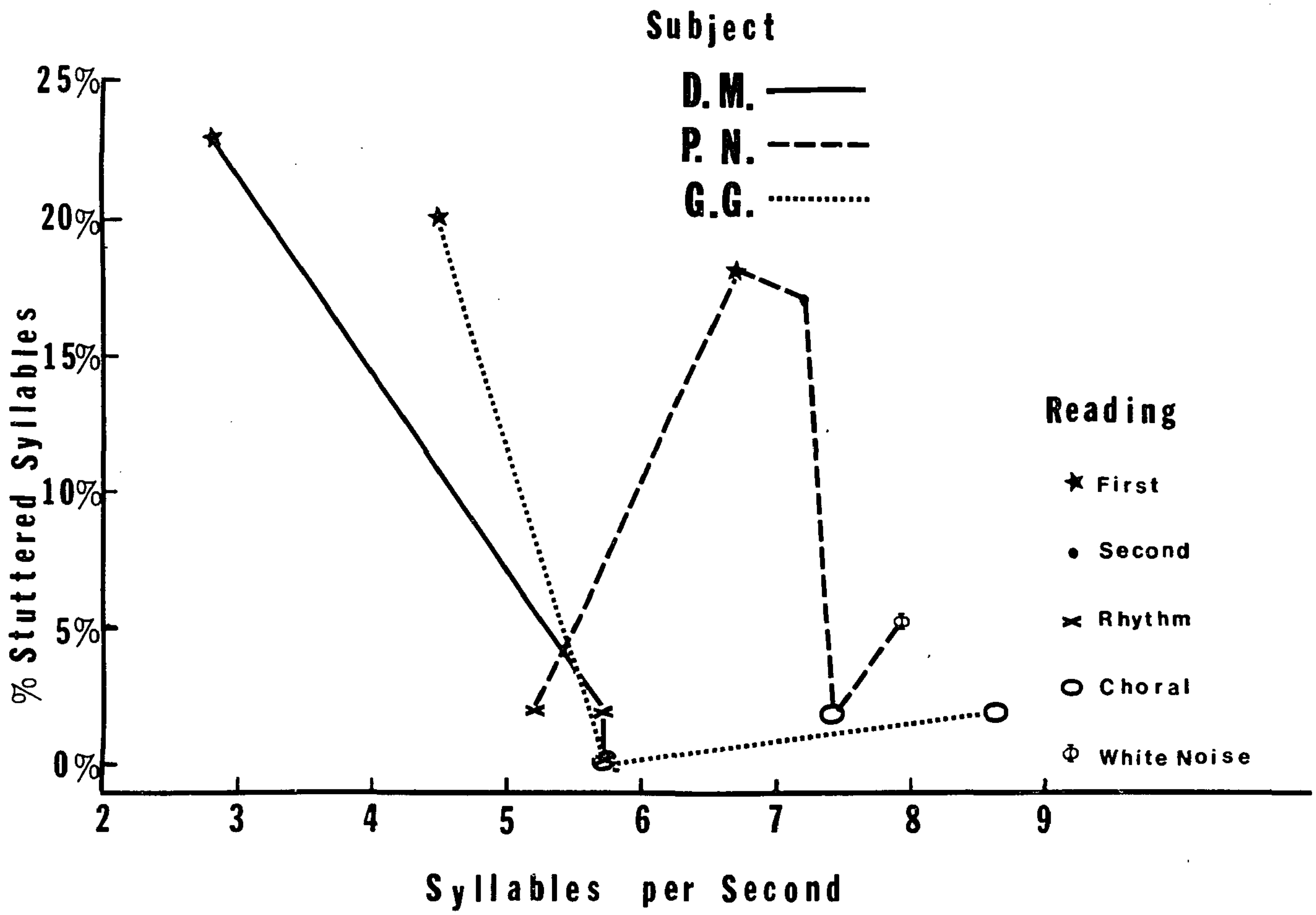
Frequency of dysfluencies, expressed as percent of syllables stuttered, are presented in the second column of Table 3. On their initial readings of the passage the subjects' frequency of dysfluencies ranged from 23% to 18%. Under the fluency evoking conditions, the dysfluencies were markedly reduced, falling below 5% in all cases.

Interaction Between Utterance Rate and Frequency of Dysfluencies

In Fig. 8 utterance rate (expressed as syllables per second) is plotted against frequency of dysfluencies (expressed as percentage of syllables stuttered). With one exception (P.N., rhythm reading), utterance rate and frequency of dysfluencies varied inversely. As the number of dysfluencies was reduced, utterance rate increased. The exception to this general rule occurred because P.N.'s first reading rate was faster than the rate imposed by the experimenter in the rhythm condition. For the other fluency evoking conditions, P.N.'s rate increased as the frequency of his dysfluencies decreased.

Figure 8

Interaction between utterance rate (expressed as syllables per second) and frequency of dysfluencies (expressed as per centage of syllables stuttered).


Subject
D. M.
P. N.
G.G.
25%
20%
15%
10%
5%
0%
% Stuttered Syllables
2
3
4
5
6
7
8
9
Syllables per Second
Reading
★ First
• Second
× Rhythm
O Choral
Φ White Noise

Description of Types of Dysfluencies

The descriptions of the stuttering patterns of each subject were based on (1) the systematic descriptions secured from the speech pathologists who listened to the audio recordings and identified the dysfluencies, (2) the observations of the researcher, (3) the observations of clinicians who had worked with, or were currently working with P.N. and G.G., and (4) the observations of the physicians and speech pathologists who were present during the experiments. These are summarized as follows:

D.M.

D.M. was considered a severe stutterer. His blocks included both repetitions and prolongations. Many of his blocks were quite long (2 to 4 seconds in duration). He frequently used "starters," such as /ʌ/, /a/, and /m/. The long blocks were often followed by rapid bursts of fluent speech.

P.N.

P.N. was considered a severe stutterer. His blocks included repetitions and hesitations (pauses), with a few prolongations. His stuttering blocks were frequently accompanied by a hoarse, breathy vocal quality.

G.G.

G.G. was considered a moderate stutterer. He exhibited more frequent blocks during the experiment than he did in ordinary conversation. Although his blocks included repetitions and a few prolongations, pauses and hard attacks were the most frequent dysfluency types. In conversation, avoidance and circumlocutions were common, as were insertions of "uh" or "um."

C.D.

C.D. was considered a mild to moderate stutterer. He did not stutter while reading the experimental passage. His blocks were evoked through conversations in which he frequently used selected "feared" words. In initiating the conversation C. D. reported that he blocked most often on, "Words beginning with fricatives." Blocks occurred primarily on the words "syllable" and "syllables." The majority of these blocks involved the repetition or prolongation of the initial voiceless fricative.

Levels of Muscle Activity in Fluent and Stuttered Utterances

Differences Observed in "Raw" (Unrectified) EMG and Processed (Rectified) EMG

In tracings of the "raw" (unrectified) EMG signal, strength of muscle activity is represented both by the amplitude and frequency of the spikes. Figures 9-11 present examples of raw EMG recordings for D.M., P.N. and G.G. In each of these figures, the upper graph shows the activity recorded from three laryngeal muscles during the subject's first reading of the passage. The lower graph in each illustration shows the activity recorded from these same muscles under one of the fluency evoking conditions. The bottom line in each graph is an oscillographic tracing of the output of the subject's microphone. A phonetic transcription was placed below each graph. For each sample the subject was reading the same portion of the experimental passage. Visual inspection of the "raw" EMG indicates that the laryngeal muscles maintained higher levels of activity during the first (stuttered) reading than during the evoked (fluent) reading.

The differences observed in the "raw" tracings were, of course, apparent in the processed (rectified) EMG. Figure 12 shows recordings from four muscles for subject P.N. The graphs in the left portion of this illustration traced the course of the electromyographic activity for these muscles during a stuttered utterance of the word "causes";

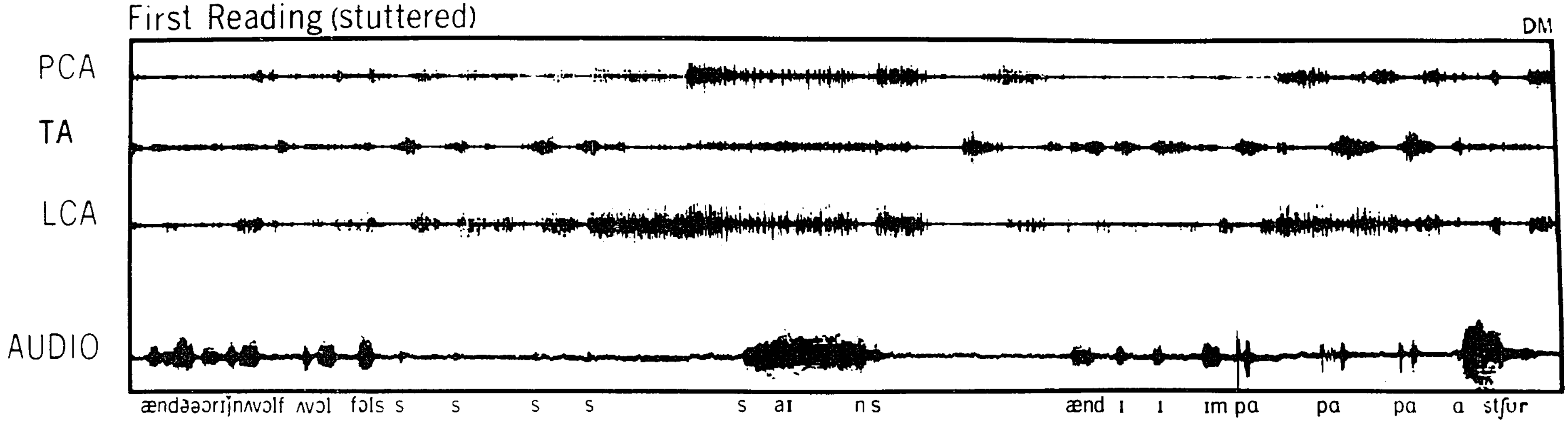
Figure 9

Raw EMG for subject D.M.

Figure 9

Raw EMG for subject D.M.

First Reading (stuttered)



Choral Reading (fluent)

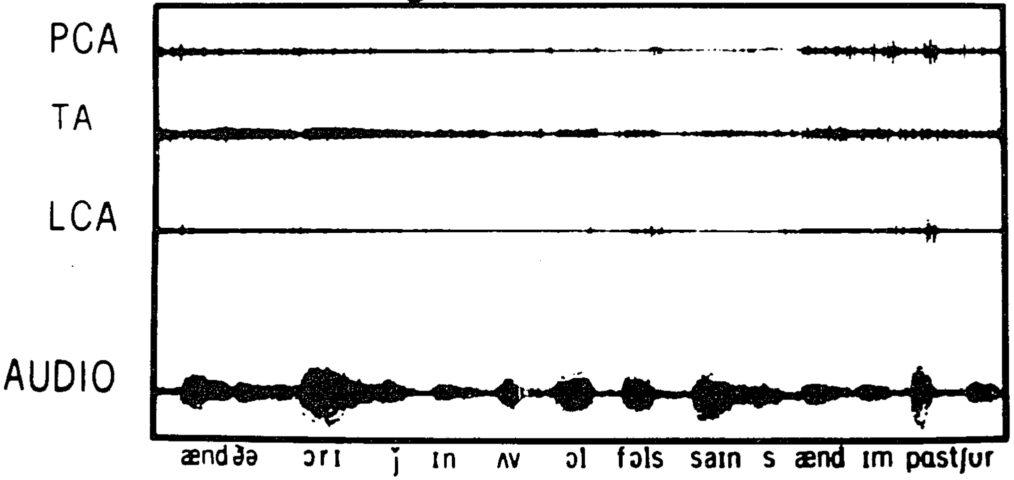
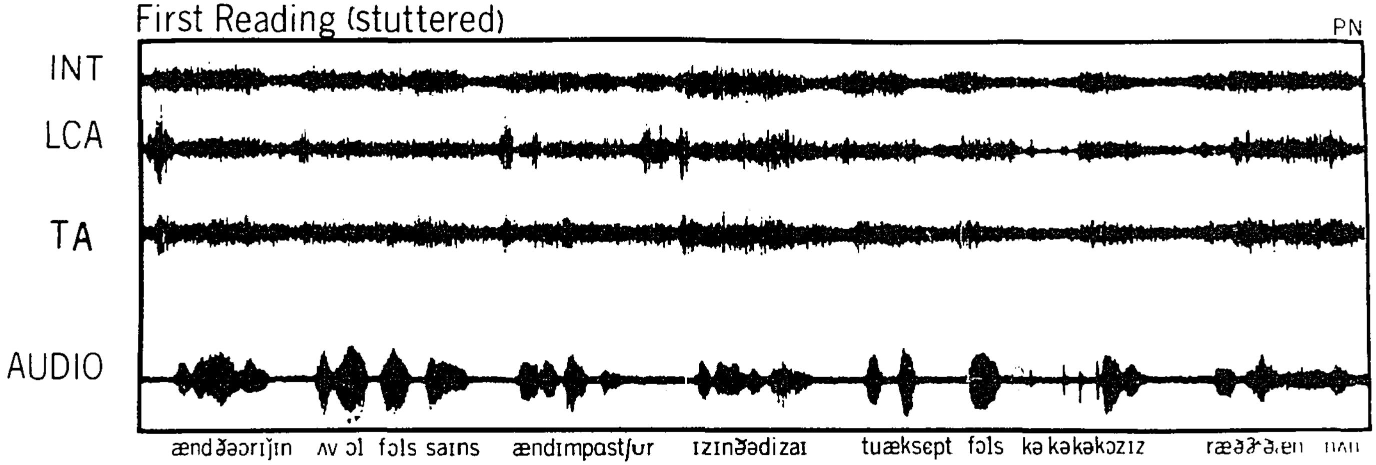


Figure 10

Raw EMG for subject P.N.

First Reading (stuttered)



White Noise Reading (fluent)

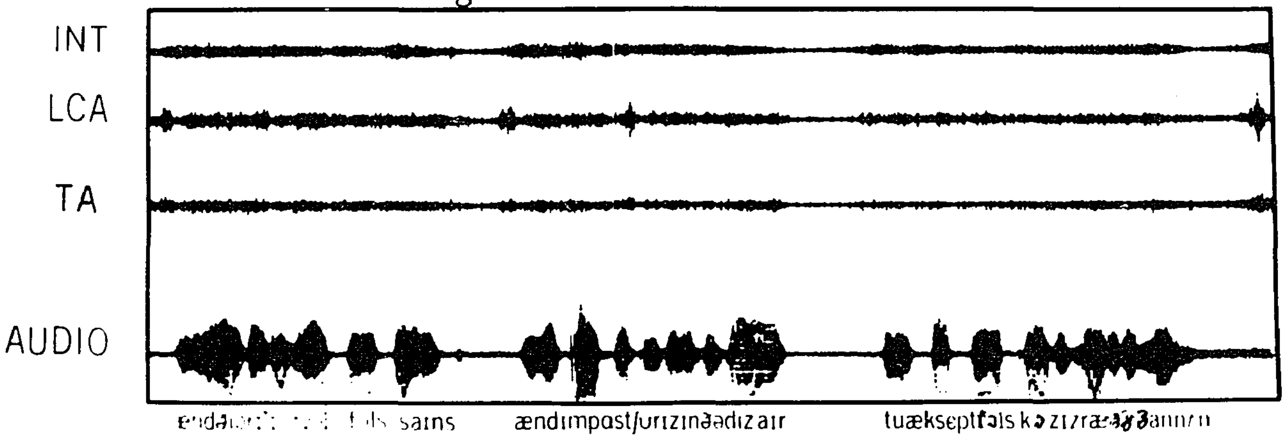
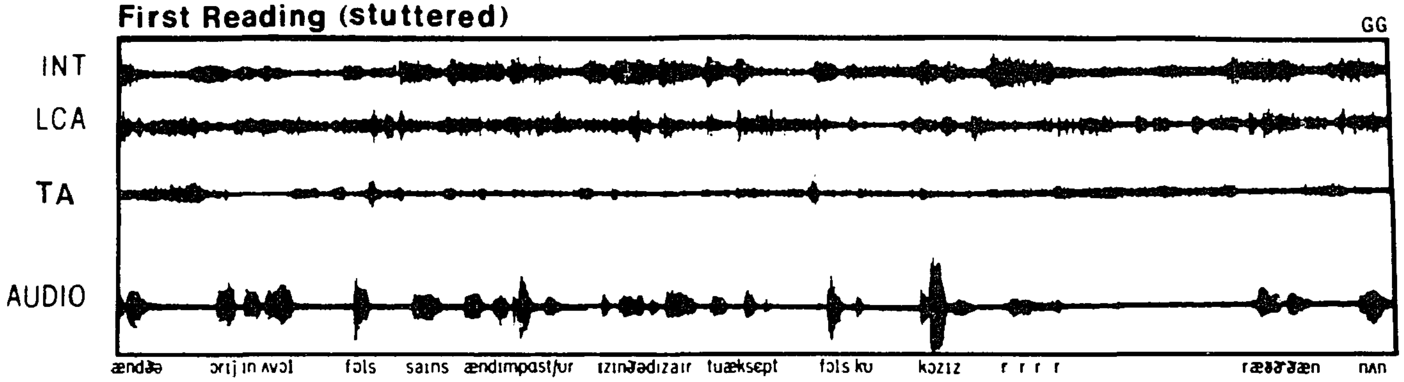


Figure 11

Raw EMG for subject G.G.

First Reading (stuttered)



Rhythm Reading (fluent)

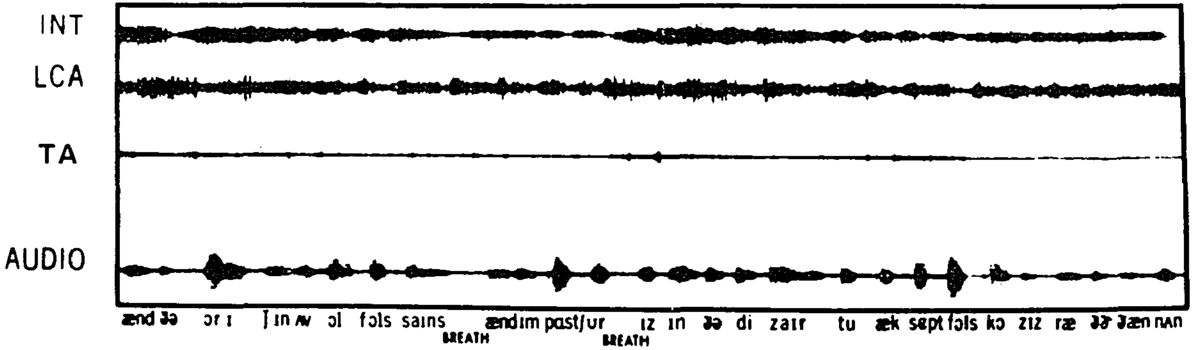
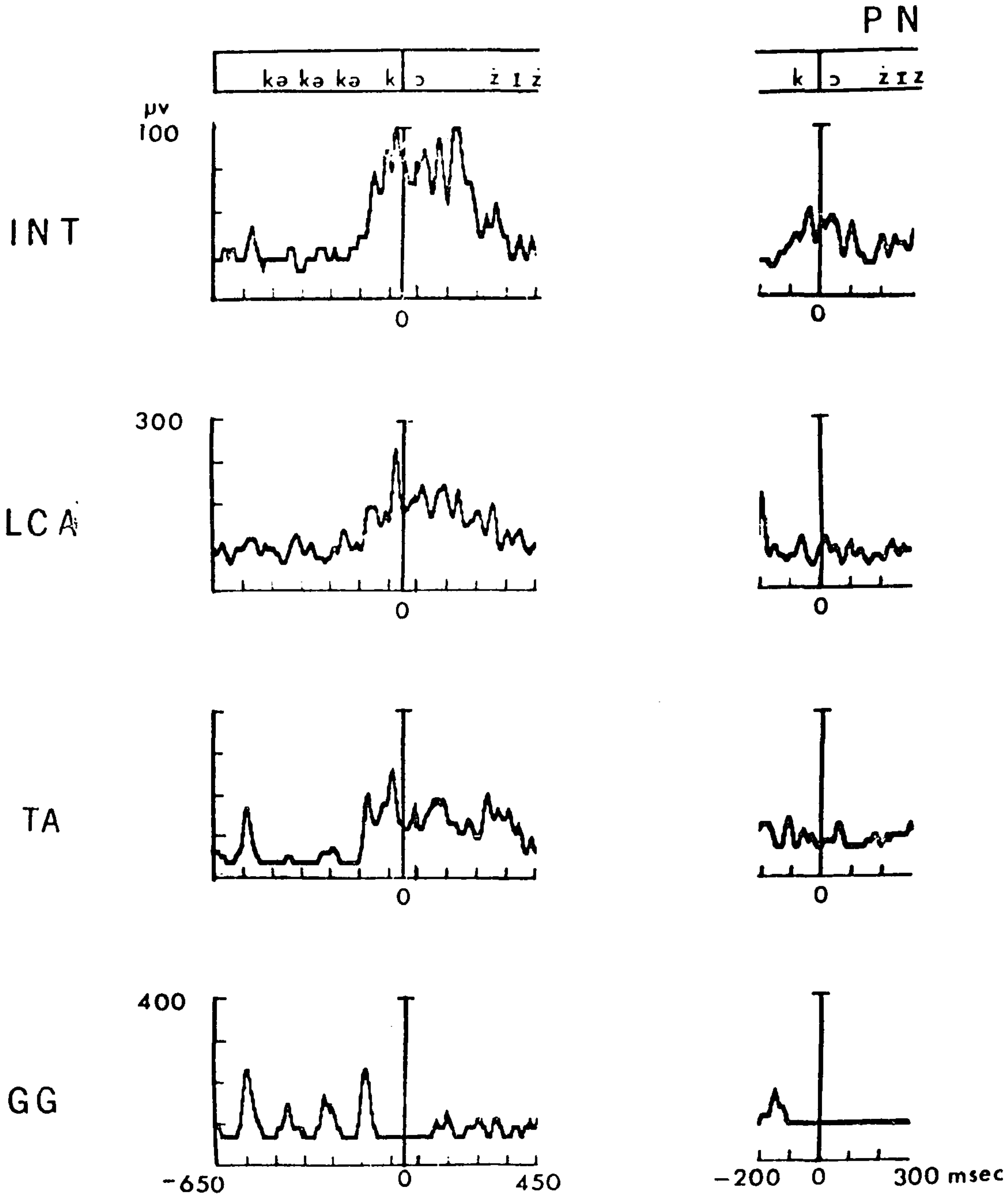


Figure 12

Fluent and stuttered utterances of the word "Causes" spoken by P.N.



P N
kə kə kə k ɔ ż ɪ ż
k ɔ ż ɪ z
µv
100
INT
0
0
300
LCA
0
0
TA
0
0
400
GG
-650
0
450
-200
0
300 msec

which occurred in the first (stuttered) reading. The fluent utterance is from his reading under white noise masking. Thus, the rectified EMG presented in Fig. 12 shows brief portions of the same information shown in the raw EMG of Fig. 10. Level settings (in microvolts) are given for each muscle.[1]

Figure 13 shows the activity of a single muscle, the LCA, for three utterances of the word "effect." The subject (D.M.) repeated the word three times, with progressive adaptation from a severe block to a mild block, to a fluent utterance. The reduction of activity in the LCA correlated with the reduction in detree of dysfluency.

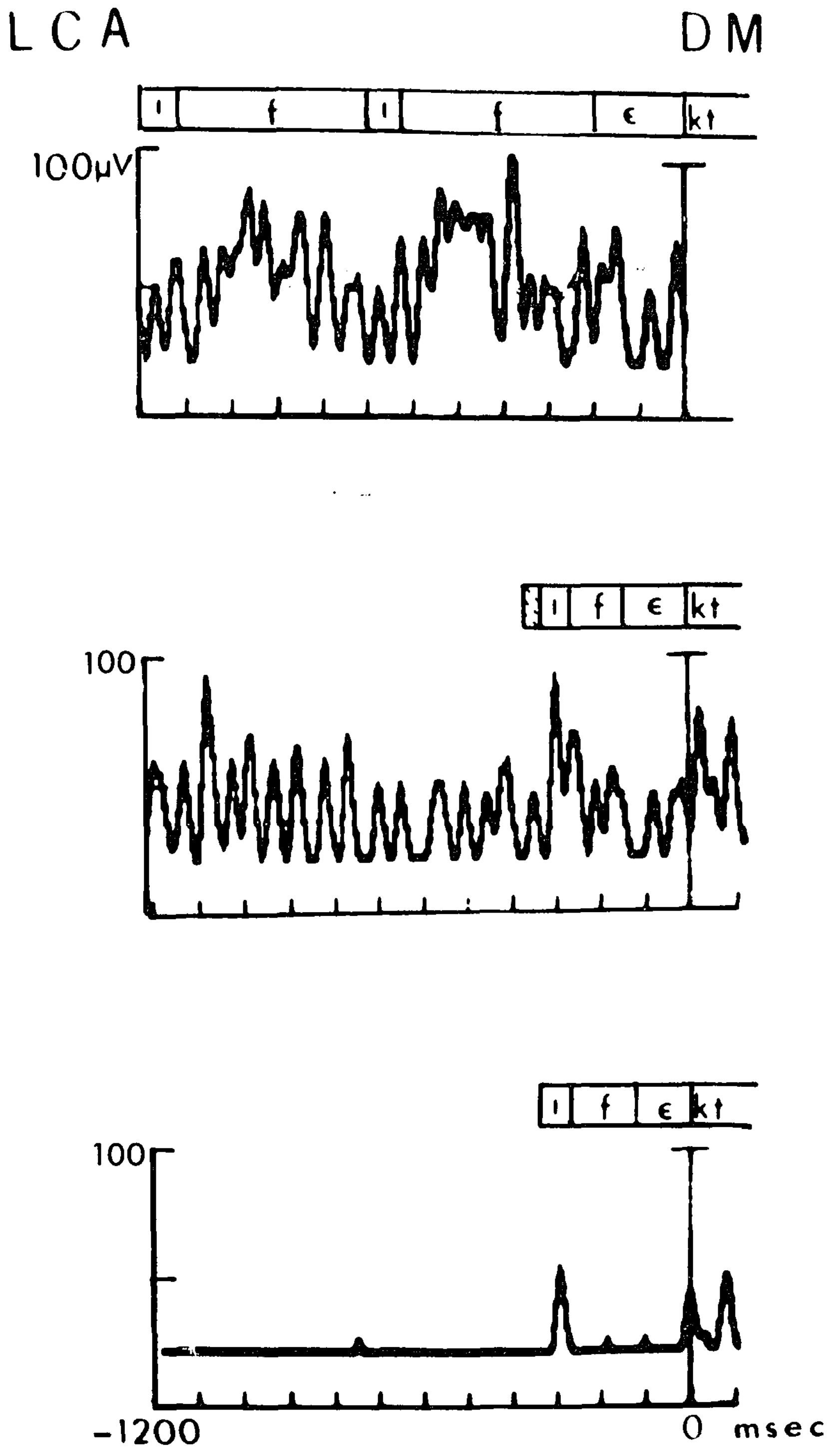
Differences in Average Levels Calculated in Two-Second Segments

In order to quantify the differences in levels of muscle activity observed in the raw and processed EMG, the selected speech samples (consisting in each case of readings of the first paragraph of the passage reproduced in Appendix A) were divided into segments of two seconds duration. The average level of activity in microvolts was calculated for each muscle for each two-second segment. The mean values for the two-second segments constituting one speech sample were then averaged together, yielding a single mean value for each muscle for each speech sample.

For each speech sample, utterance content was held relatively constant, but the total length of the sample (number of two-second

Figure 13

Recordings from the LCA for three utterances of the word "effect" spoken by subject D.M. The first utterance was strongly stuttered, the second was mildly stuttered, and the third was fluent.


LCA
DM
l
f
l
f
ɛ
kt
100μV
l
f
ɛ
kt
100
l
f
ɛ
kt
100
-1200
0 msec

reading was compared with each of the readings under the fluency evoking conditions. Results are presented for each muscle for each subject.

Results for D.M.

Results for subject D.M. are presented in Table 4. In the upper portion of the Table, means and comparisons of means are given for four upper tract articulator muscles (orbicularis oris, OO); genioglossus, GG; inferior longitudinal, IL; and superior longitudinal, SL). In the lower portion, results are given for three intrinsic laryngeal muscles (thyroarytenoid, TA; posterior cricoarytenoid, PCA; and lateral cricoarytenoid, LCA). For each muscle for each speech sample (first reading, rhythm reading, and choral reading) the mean levels and standard deviations are given in microvolts. Then each fluency evoking condition is compared with the first reading, and the resulting _t_ scores are presented.

Of the eight comparisons made for the upper tract articulator muscles, only one (GG, choral reading) shows a significant difference (.01 level of confidence). Of the six comparisons made for the laryngeal muscles, all six show significant differences (two at the .01 level of confidence and four at the .001 level of confidence). For all of the significant differences, the mean levels were higher for the first (stuttered) reading and lower for the other (evoked fluent) conditions.

Figure 13 illustrates these differences by converting the

TABLE 4

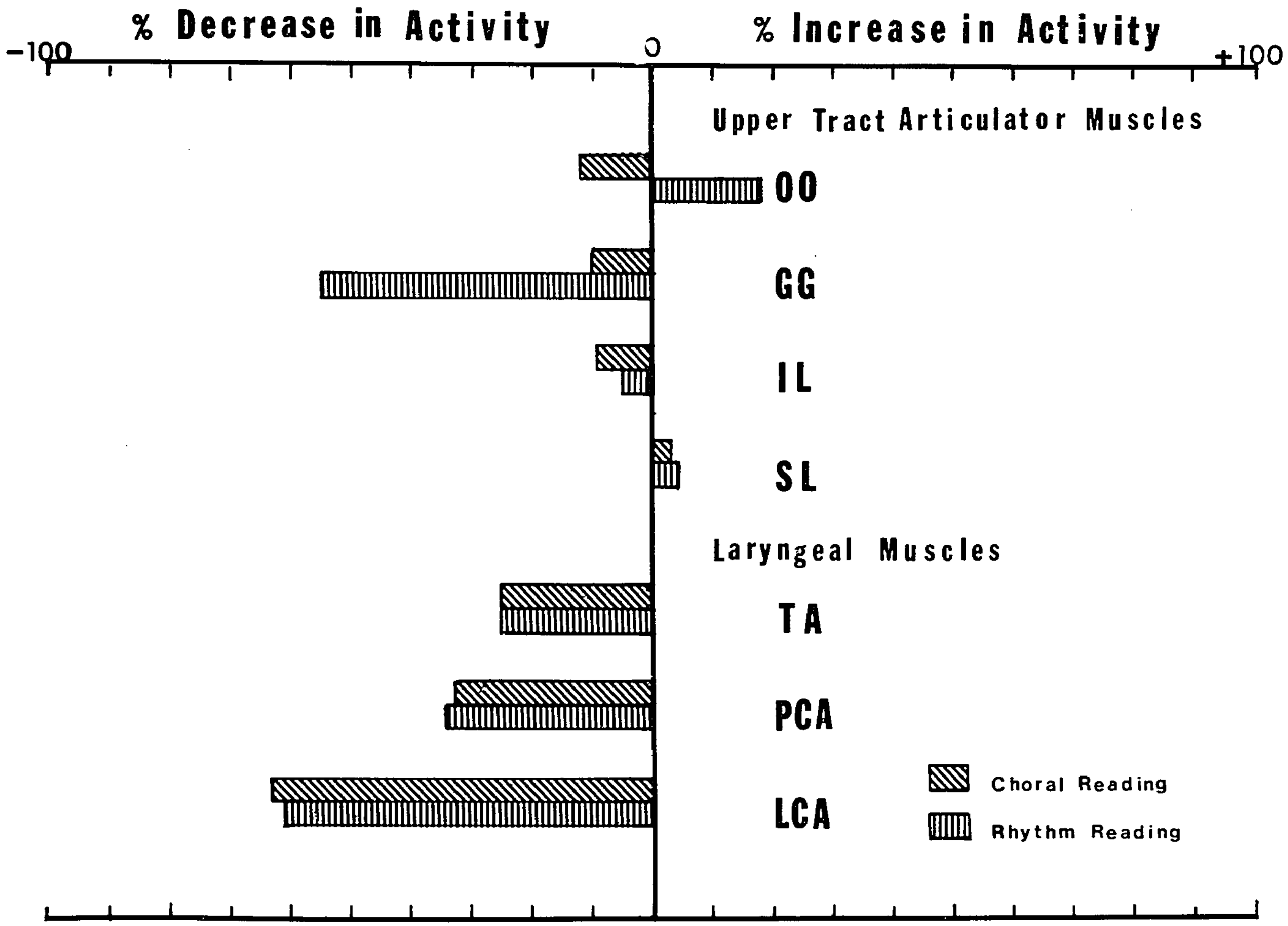
Results for D.M.
Differences in average levels calculated in two-second segments

MUSCLE		R#1	RHYTHM	CHORAL
OO	(mean)	22.82	20.04	27.00
	(SD)	10.81	2.22	2.24
	t =		1.28	2.29
GG	(mean)	9.37	8.45	4.25
	(SD)	6.58	6.62	5.40
	t =		0.51	2.96*
IL	(mean)	47.78	43.93	45.15
	(SD)	23.16	10.71	10.48
	t =		0.77	0.51
SL	(mean)	13.96	14.33	14.54
	(SD)	4.25	4.37	8.48
	t =		0.31	0.31
TA	(mean)	29.93	22.41	22.50
	(SD)	8.68	6.79	7.14
	t =		3.48*	3.25*
PCA	(mean)	38.33	25.63	25.13
	(SD)	9.51	6.76	10.66
	t =		5.55**	4.596**
LCA	(mean)	86.59	32.30	33.75
	(SD)	23.57	11.22	9.03
	t =		10.61**	10.12**

Levels of Confidence: * = .01 and ** = .001

Figure 14

Average levels for two-second segments for the fluent readings are compared with the first (stuttered) reading for subject D.M.



0 indicates level of activity for the First (stuttered) Reading

TABLE 5

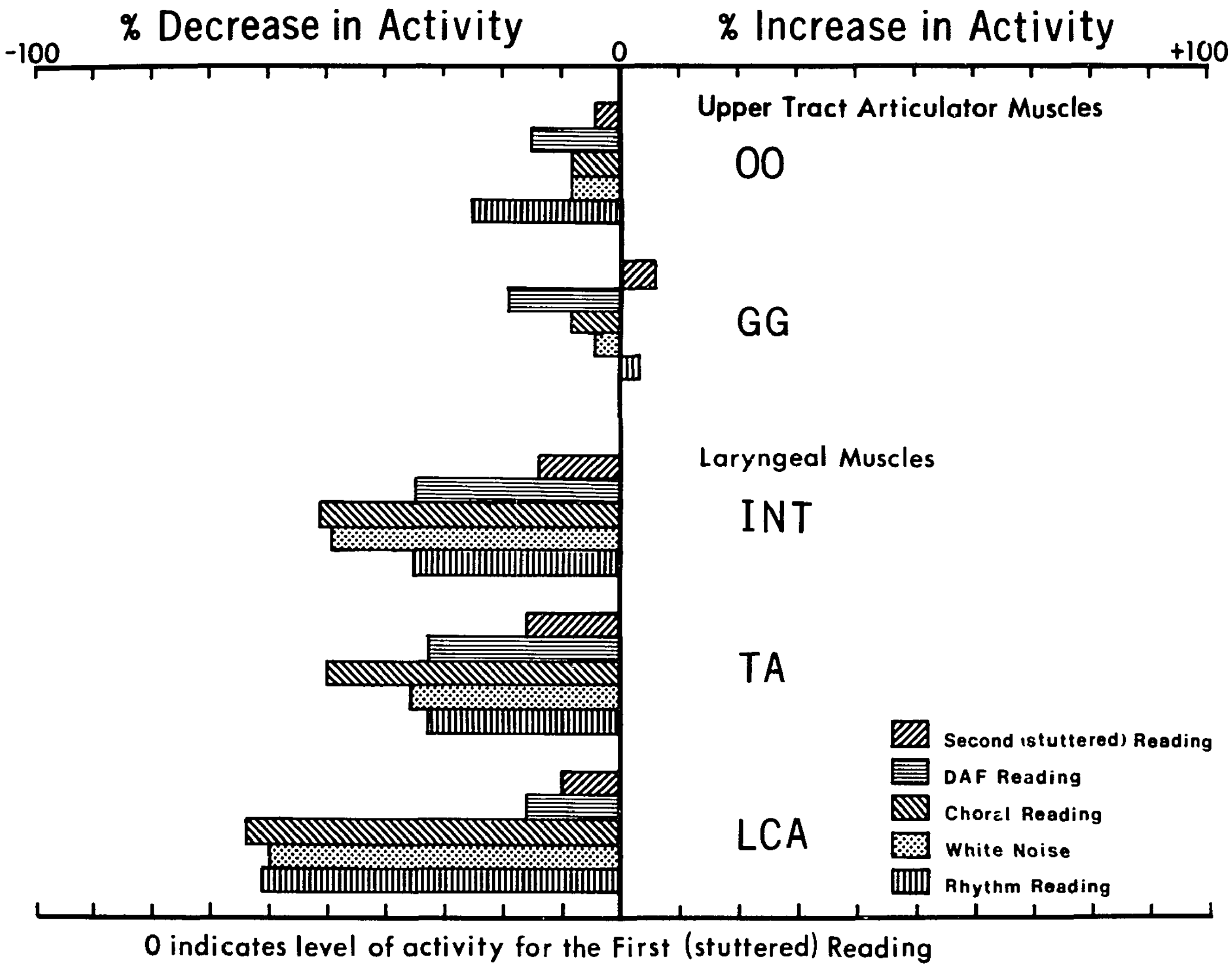
Results for P.N.
Differences in average levels calculated in two-second segments

MUSCLE		R#1	R#2	DAF	CHORAL	WHITE NOISE	RHYTHM
OO	(mean)	20.36	19.50	17.31	18.63	18.80	15.18
	(SD)	4.02	7.25	5.59	2.11	4.16	4.42
	t =		.76	2.64	1.52	1.35	5.42**
GG	(mean)	110.91	117.78	90.00	102.50	106.67	114.09
	(SD)	22.34	35.83	12.25	6.61	13.98	23.77
	t =		.72	3.30**	1.42	.64	.45
IA	(mean)	62.91	54.11	40.63	30.69	31.93	40.59
	(SD)	11.18	14.57	7.28	2.99	2.67	11.16
	t =		2.11	6.79**	10.94**	10.24**	6.48**
TA	(mean)	207.14	173.78	158.50	125.00	133.07	159.55
	(SD)	57.49	37.24	47.66	13.29	30.98	35.79
	t =		2.07	2.69*	5.46**	4.43**	3.22*
LCA	(mean)	147.23	132.72	124.19	52.50	58.73	57.91
	(SD)	18.46	19.25	15.79	3.71	6.44	12.87
	t =		2.36	3.92**	19.69**	17.35**	18.19**

Levels of Confidence: * = 01 and ** = 001

Figure 15

Average levels for two-second segments for the fluent readings are compared with the first (stuttered) reading for subject P.N.


% Decrease in Activity
% Increase in Activity
-100
0
+100
Upper Tract Articulator Muscles
OO
GG
Laryngeal Muscles
INT
TA
LCA
Second (stuttered) Reading
DAF Reading
Choral Reading
White Noise
Rhythm Reading
0 indicates level of activity for the First (stuttered) Reading

microvolt values to percentages, using the mean level of the first reading as a reference.

Results for P.N.

Table 5 presents similar results for subject P.N. for five muscles, two upper tract articulators (OO and GG) and three intrinsic laryngeals (interarytenoids, INT; TA and LCA). Six speech samples are utilized--first reading, second reading, reading under delayed auditory feedback (DAF), reading under white noise masking, choral reading and rhythm reading. Levels for the second reading were generally slightly lower than for the first reading, but these differences were not significant. Since the reduction in per cent of syllables stuttered from P.N.'s first to second reading was only 1% (18% to 17%) this finding is not surprising.

Of the eight other comparisons made for the upper tract articulators two were significant (.001 level of confidence). Of the twelve other comparisons made for the laryngeal muscles, all twelve showed significant differences (two at the .01 level of confidence and ten at the .001 level of confidence).

Figure 15 graphically illustrates these differences. As in Fig. 14, the means of the other speech samples are presented as per centages of the mean of the first reading.

For all of the significant differences, the mean level was higher for the first (stuttered) reading and lower for the other (evoked fluency) conditions. In this respect D.M. and P.N. were alike.

Results for G.G.

Results for subject G.G. are presented in Table 6 and illustrated in Fig. 16. The upper portions of the table and the figure both show findings for the two upper tract articulators (OO and SL); while the lower portions show findings on four intrinsic laryngeals (INT, TA, and LCA; and cricothyroid, CT) and one extrinsic laryngeal strap muscle (sternohyoid, SH).

For each muscle, the first (stuttered) reading is compared with the fluency evoking conditions of rhythm reading, choral reading, and whispering.

Both of the upper tract articulator muscles show significant differences, but the differences reflect opposite effects. For the labial muscle, the mean levels were significantly lower for the fluency evoking conditions and higher for the first (stuttered) reading. For the lingual muscle, the opposite was true, with the fluency evoking conditions showing significantly higher mean levels of activity.

The laryngeal muscles also show significant differences in both directions. The SH shows a significantly lower level for only one condition, rhythm. The LCA and INT both show significantly higher levels for the choral and rhythm conditions. Conversely, the LCA shows a significantly lower level for whispering. Unlike the other three intrinsic laryngeals, the TA shows significantly lower levels for both choral and rhythm readings.

TABLE 6

Results for G.G.
Differences in average levels calculated in two-second segments

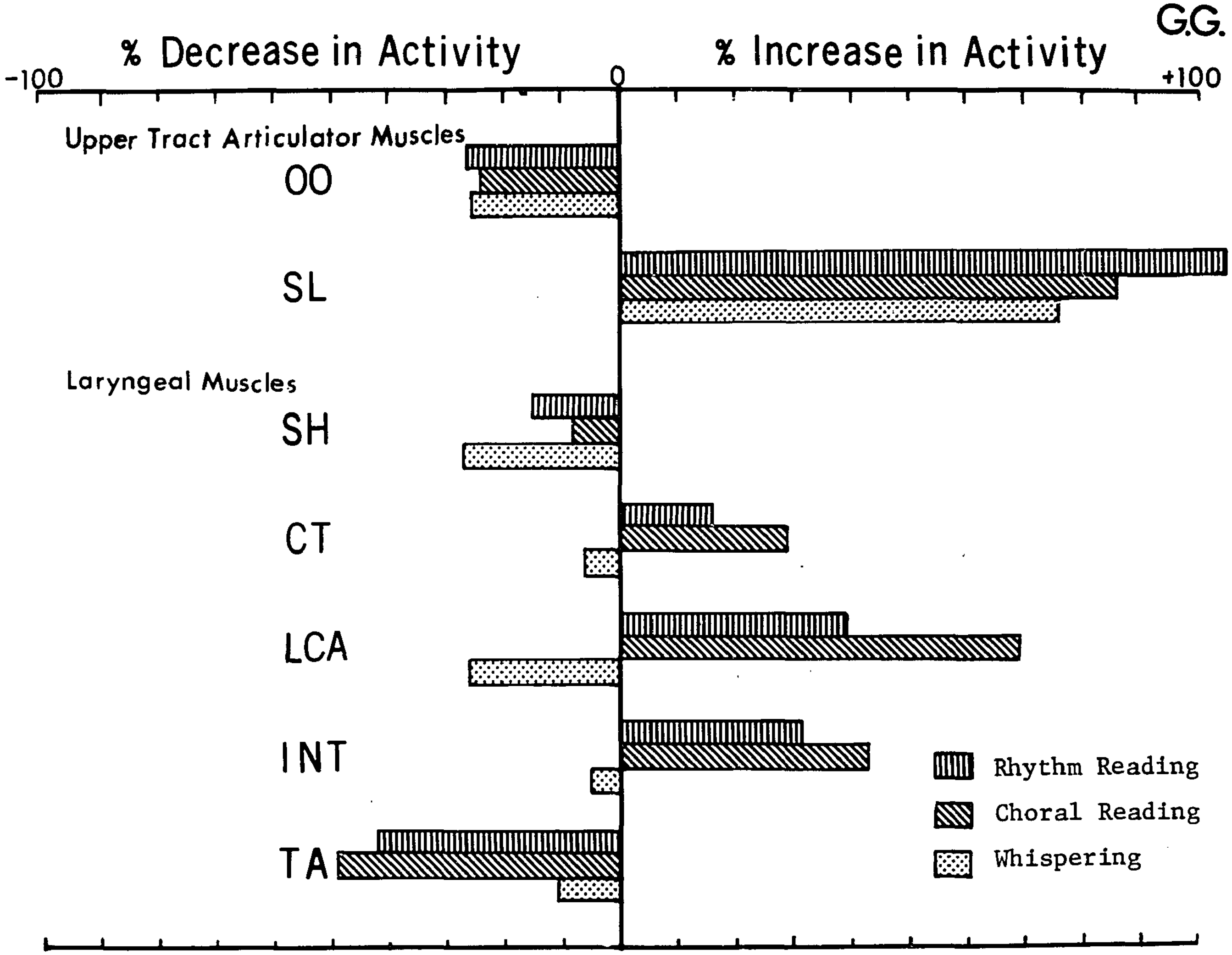
MUSCLE		R#1	CHORAL	RHYTHM	WHISPER
00	(mean)	16.77	12.47	12.77	12.50
	(SD)	1.48	.50	.94	.50
	t =		11.48**	9.14**	10.64**
SL	(mean)	16.24	33.21	30.18	28.56
	(SD)	6.22	8.99	7.40	11.84
	t =		6.33**	5.77**	3.66**
SH	(mean)	23.06	19.63	21.12	16.75
	(SD)	3.39	3.99	3.14	2.41
	t =		2.68	1.68	5.94**
CT	(mean)	14.71	17.05	18.94	13.75
	(SD)	1.67	7.08	1.77	1.56
	t =		1.30	6.97**	1.64
LCA	(mean)	21.65	30.00	36.65	15.94
	(SD)	3.88	5.98	6.31	6.91
	t =		4.77**	8.11**	2.86*
IA	(mean)	76.24	99.58	108.71	72.44
	(SD)	24.68	15.82	16.50	25.71
	t =		3.32*	4.38**	0.43
TA	(mean)	19.53	11.32	10.00	17.31
	(SD)	10.57	1.66	1.33	1.69
	t =		3.25*	3.58**	0.81

Levels of Confidence: * = .01 and ** = .001

Underline indicates those differences where levels were higher for the fluent conditions.

Figure 16

Average levels for two-second segments for the fluent readings are compared with the first (stuttered) readings for subject G.G.



0 indicates level of activity for the first (stuttered) Reading

Combined Results

Table 7 summarizes a portion of the results relating to decreases in average levels calculated in two-second segments by showing for each subject and for each muscle the number of comparisons (NC) made between the first reading and the fluent readings, and the number of significant decreases (NSD). Decreases in activity with differences reaching the .01 and .001 levels of confidence were considered significant. The percentage of significant decreases (NSD/NC),was computed for each subject and for each muscle. The percentage of significant decreases (NSD/CD) was then computed for the laryngeal muscles group and for the upper tract articulator muscle group. The laryngeal muscles as a group showed significant decreases for 67% of the comparisons, while the upper tract articulator group showed significant decreases for 29% of the comparisons.

Figure 17 also allows comparison of the upper tract articulator muscles with the laryngeal muscles for each subject. The dashed line labeled 100% shows the reference, based on each subject's mean levels for the first (stuttered) reading. The horizontally striated bar for each subject is the average of all the upper tract articulator muscles for all of the fluency-evoking conditions. The two bars on the right, labeled "Total" are the averages for the upper tract articulator muscles and the laryngeal muscles for all subjects for all of the fluency-evoking conditions. In each case, the average levels are lower for the laryngeal muscles and higher for the upper tract articulator muscles.

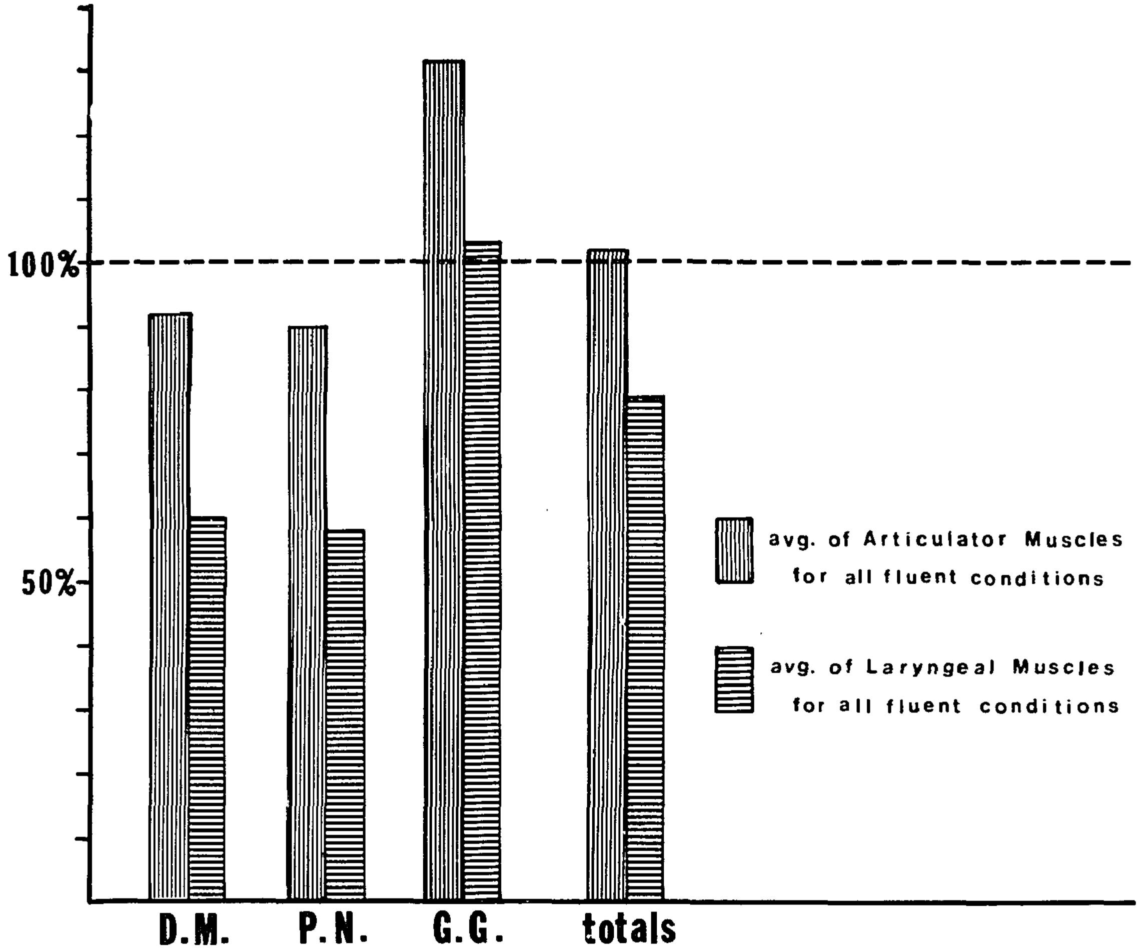
TABLE 7

Number of Significant Differences (NSD) divided by the Number of Comparisons (NC) for each muscle for each subject, with combined totals for laryngeal muscles and upper tract articulators for each muscle, and for each subject.

MUSCLE	SUBJECTS			TOTALS
	D.M.	P.N.	G.G.	
	NSD/NC = %	NSD/NC = %	NSD/NC = %	NSD/NC = %
PCA	2/2 = 100%			2/2 = 100%
INT		4/4 = 100%	0/3 = 0%	4/7 = 57%
LCA	2/2 = 100%	4/4 = 100%	1/3 = 33%	7/9 = 78%
TA	2/2 = 100%	4/4 = 100%	2/3 = 67%	8/9 = 89%
CT			0/3 = 0%	0/3 = 0%
SH			1/3 = 33%	1/3 = 33%
LARYNGEAL TOTALS	6/6 = 100%	16/16 = 100%	4/15 = 27%	22/33 = 67%
IL	0/2 = 0%			0/2 = 0%
SL	0/2 = 0%		0/2 = 0%	0/4 = 0%
GG	1/2 - 50%	1/4 = 25%		2/6 = 33%
OO	0/2 = 0%	1/4 = 25%	3/3 = 100%	4/9 = 44%
UPPER TRACT TOTALS	1/8 = 13%	2/8 = 25%	3/5 = 60%	6/21 = 29%

Figure 17

Comparison of average levels for two-second segments for upper tract articulator muscles and laryngeal muscles



100% indicates levels for the First (stuttered) Reading

Differences in Average Levels

Per Syllable

The findings of higher levels of muscle activity for the fluency evoking conditions for subject G.G., at first appears contradictory. However, decreases in frequency of dysfluencies are accompanied by increases in utterance rate (see Fig. 8). Increases in utterance rate may be accompanied by increases in average level of muscle activity for two reasons. First, an increase in syllables per second would results in an increase in the number of speech gestures per second, and hence an increase in the average level of muscle activity per two-second segment. Second, an increase in rate would result in a higher velocity of articulator movement, which should require a higher level of muscle activity (Bigland and Lippold, 1954a).[2] For these two reasons, if other factors were held equal, increases in utterance rate should result in increases in average level of muscle activity per two-second segment. However, for the subjects in this study (see Fig. 8) increases in rate correlated with decreases in frequency of stuttering blocks. Clearly, two opposite, and potentially cancelling, effects were simultaneously operative.

For two of the three subjects (D.M. and P.N.) the effects of the reduction in stuttering appear to dominate; while for the third subject (G.G.) effects of increased rate were stronger for some muscles.

In order to neutralize the effects of the increase in utterance rate, the syllables in each two-second segment were counted, and the average level of muscle activity in each segment was divided by the number of syllables uttered in that segment. The resulting means were then used to calculate an average level per syllable for each muscle, for each speech sample. Results for each muscle, for each subject, for each speech sample are presented.

Results for D.M.

Table 8 shows the average level (in microvolts) of muscle activity per syllable for the first (stuttered) reading and for the rhythm reading condition. The choral reading condition was omitted because it was not possible to separate the voice of the experimenter from the voice of the subject for the syllable count.

For the subject D.M. differences were significant at the .001 level of confidence for the OO, the PCA and the LCA. These comparisons are graphically illustrated in Fig. 18. The levels for the rhythm readings are presented as percentages of the levels of the first (stuttered) reading.

Results for P.N.

Results for P.N. are presented in Table 9 and Fig. 19. For this subject four fluency-evoking conditions are compared with the first (stuttered) reading. None of the eight comparisons of the upper tract articulator muscles show significant differences; however, eight of the twelve comparisons of laryngeal muscles do show significant differences.

Results for G.G.

Two fluency-evoking conditions (rhythm reading and choral reading) are compared with the first (stuttered) reading. The

TABLE 8

Results for D.M.
Differences in average levels per syllable

MUSCLE		R 1	RHYTHM
OO	(mean)	11.80	4.53
	(SD)	7.54	0.90
	t =		3.63**
GG	(mean)	6.00	1.65
	(SD)	7.30	0.31
	t =		2.25
IL	(mean)	30.46	10.09
	(SD)	30.25	2.78
	t =		2.53
SL	(mean)	8.02	3.07
	(SD)	5.75	0.94
	t =		3.22*
TA	(mean)	17.93	5.87
	(SD)	13.59	2.84
	t =		3.30
PCA	(mean)	23.46	5.67
	(SD)	17.70	1.68
	t =		3.77**
LCA	(mean)	52.27	6.78
	(SD)	41.74	2.11
	t =		4.11**

Levels of Confidence: * = .01 and ** = .001

TABLE 9

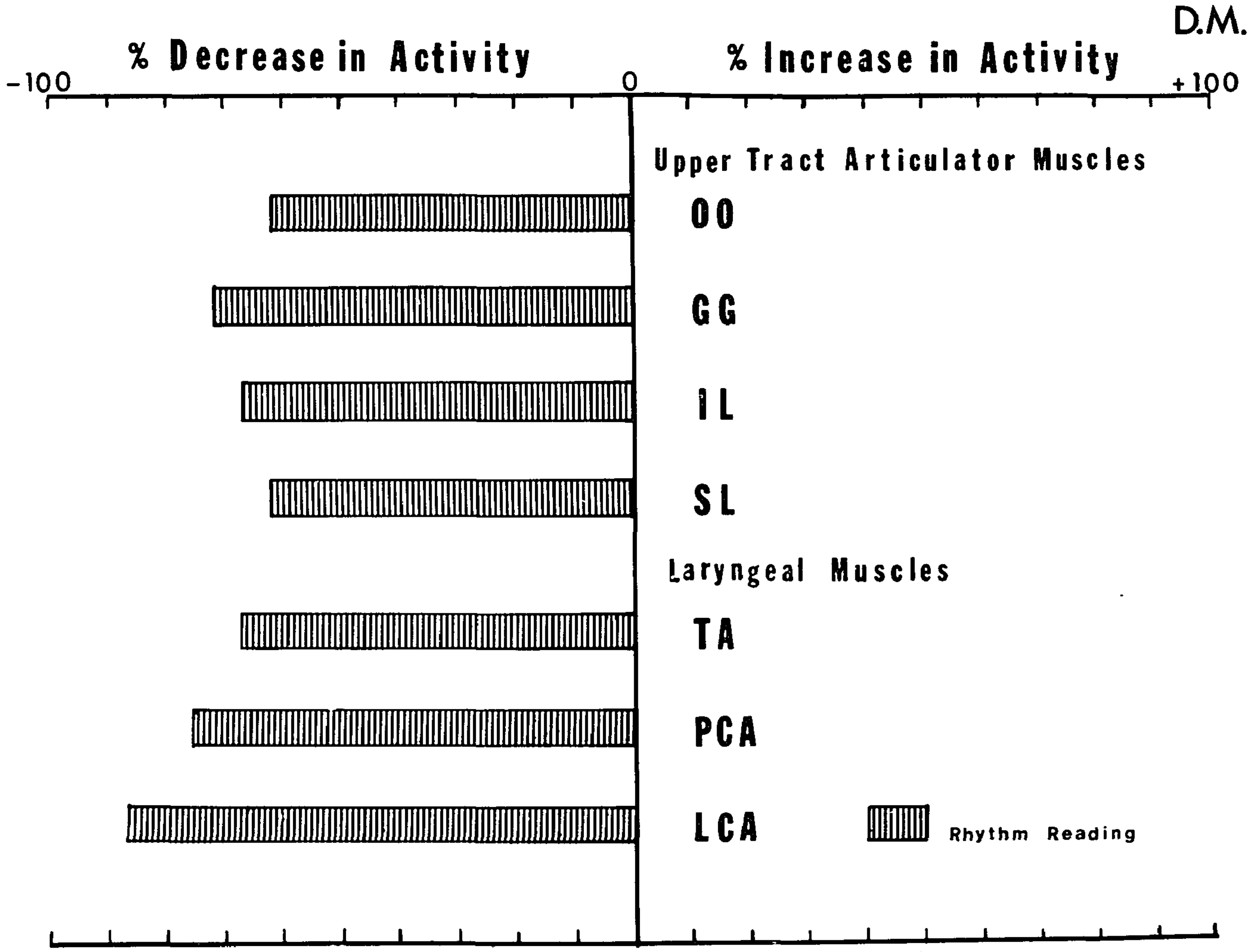
Results for P.N.
Differences in average levels per syllable

MUSCLE		R 1	RHYTHM	CHORAL	NOISE	DAF
00	(mean)	4.67	3.83	3.40	3.30	3.95
	(SD)	2.86	1.75	0.61	0.83	2.40
	t =		0.95	1.46	1.48	0.67
GG	(mean)	26.27	28.17	18.77	19.68	19.56
	(SD)	9.44	9.37	3.51	5.84	9.80
	t =		0.53	2.50	1.95	1.71
IA	(mean)	14.71	10.79	5.49	5.73	8.39
	(SD)	5.37	4.96	1.37	1.16	2.70
	t =		2.01	5.56**	5.22**	3.55*
LCA	(mean)	33.48	16.18	9.71	11.00	28.02
	(SD)	10.94	7.30	1.53	1.91	12.92
	t =		4.98**	7.17**	6.48**	1.12
TA	(mean)	48.13	44.59	23.14	23.97	36.58
	(SD)	19.04	21.56	5.49	7.64	18.51
	t =		0.46	4.22**	3.81**	1.50

Levels of Confidence : * = .01 and ** = .001

Figure 18

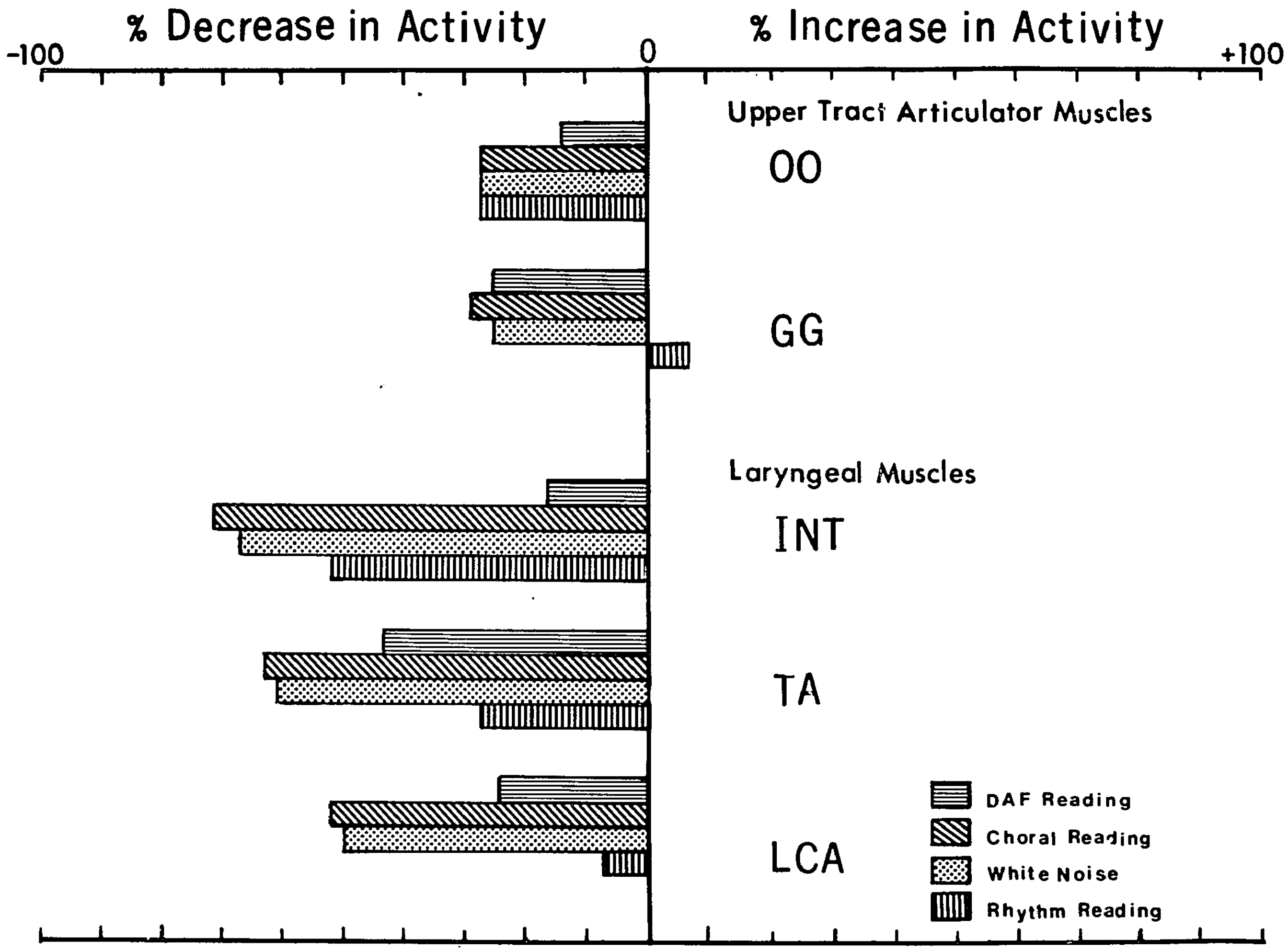
Average levels per syllable for the fluent readings are compared with the first (stuttered) reading for subject D.M.



0 indicates level of activity for the First (stuttered) Reading

Figure 19

Average levels per syllable for the fluent readings are compared with the first (stuttered) reading for subject P.N.



0 indicates level of activity for the First (stuttered) Reading

whispered condition is omitted because of the problems involved in counting the syllables in each two-second segment from the oscillographic tracing of unvoiced speech.

The results for G.G. presented in Table 10 and Fig. 20, show lower levels of muscle activity per syllable for the fluency-evoking conditions and higher levels for the first (stuttered) reading. These findings may be contrasted with those shown in Table 6 and Fig. 16. Two muscles, the OO and the SH, show differences which reach the .01 level of confidence.

Combined Results

Table 11 summarizes the results relating to decreases in activity in the average levels per syllable. Again, the number of significant decreases (NSD) are divided by the number of comparisons (NC) to yield a percentage of significant decreases for each muscle and for each subject. Subtotals are computed for the laryngeal muscles and for the upper tract articulator muscles. Fifty-two percent of the laryngeal muscle comparisons showed significant (.01 or .001 level of confidence) decreases, while 18 percent of the upper tract articulator muscle comparisons showed significant decreases.

In Fig. 21, the broken line labeled 100% indicates the reference level of the first (stuttered) reading. The horizontally striated bars are the average of all the upper tract articulator muscles for all of the fluency-evoking conditions. The vertically striated bars are the average of all the laryngeal muscles for all

TABLE 10

Results for G.G.
Differences in average levels per syllable

MUSCLE		R 1	CHORAL	RHYTHM
OO	(mean)	6.69	1.94	2.70
	(SD)	5.08	0.28	0.35
	t =		2.85*	2.84*
SL	(mean)	7.01	4.07	5.89
	(SD)	8.25	0.84	1.29
	t =		1.09	0.49
SH	(mean)	8.74	2.68	4.13
	(SD)	5.85	0.62	0.53
	t =		3.15*	2.84*
CT	(mean)	5.50	2.39	3.84
	(SD)	3.51	0.51	0.32
	t =		2.74	1.61
LCA	(mean)	7.86	4.11	7.07
	(SD)	4.75	1.06	0.92
	t =		2.38	0.59
IA	(mean)	28.80	14.99	22.11
	(SD)	21.82	2.91	3.33
	t =		1.92	1.10
TA	(mean)	9.80	1.79	2.14
	(SD)	13.59	0.35	0.39
	t =		1.80	2.04

Levels of Confidence: * = .01

TABLE 11

Number of Significant Differences (NSD) divided by the Number of Comparisons (NC) for each muscle for each subject, with combined totals for laryngeal muscles and upper tract articulators for each muscle, and for each subject.

MUSCLE	SUBJECTS			TOTALS
	D.M. NSD/NC = %	P.N. NSD/NC = %	G.G. NSD/NC = %	NSD/NC = %
PCA	1/1 = 100%			1/1 = 100%
INT		3/4 = 75%	0/2 = 0%	3/6 = 50%
LCA	1/1 = 100%	3/4 = 75%	0/2 = 0%	4/7 = 57%
TA	1/1 = 100%	2/4 = 50%	0/2 = 0%	3/7 = 43%
CT			0/2 = 0%	0/2 = 0%
SH			2/2 = 100%	2/2 = 100%
LARYNGEAL TOTALS	3/3 = 100%	8/12= 67%	2/10= 20%	13/25= 52%
IL	0/1 = 0%			0/1 = 0%
SL	1/1 = 100%	0/4 = 0%	0/2 = 0%	1/7 = 14%
GG	0/1 = 0%	0/4 = 0%	0/2 = 0%	0/7 = 0%
OO	1/1 = 100%	0/4 = 0%	2/2 = 100%	3/7 = 43%
UPPER TRACT TOTALS	2/4 = 50%	0/4 = 0%	2/6 = 33%	4/22= 18%

Figure 20

Average levels per syllable for the fluent readings are compared with the first (stuttered) reading for subject G.G.

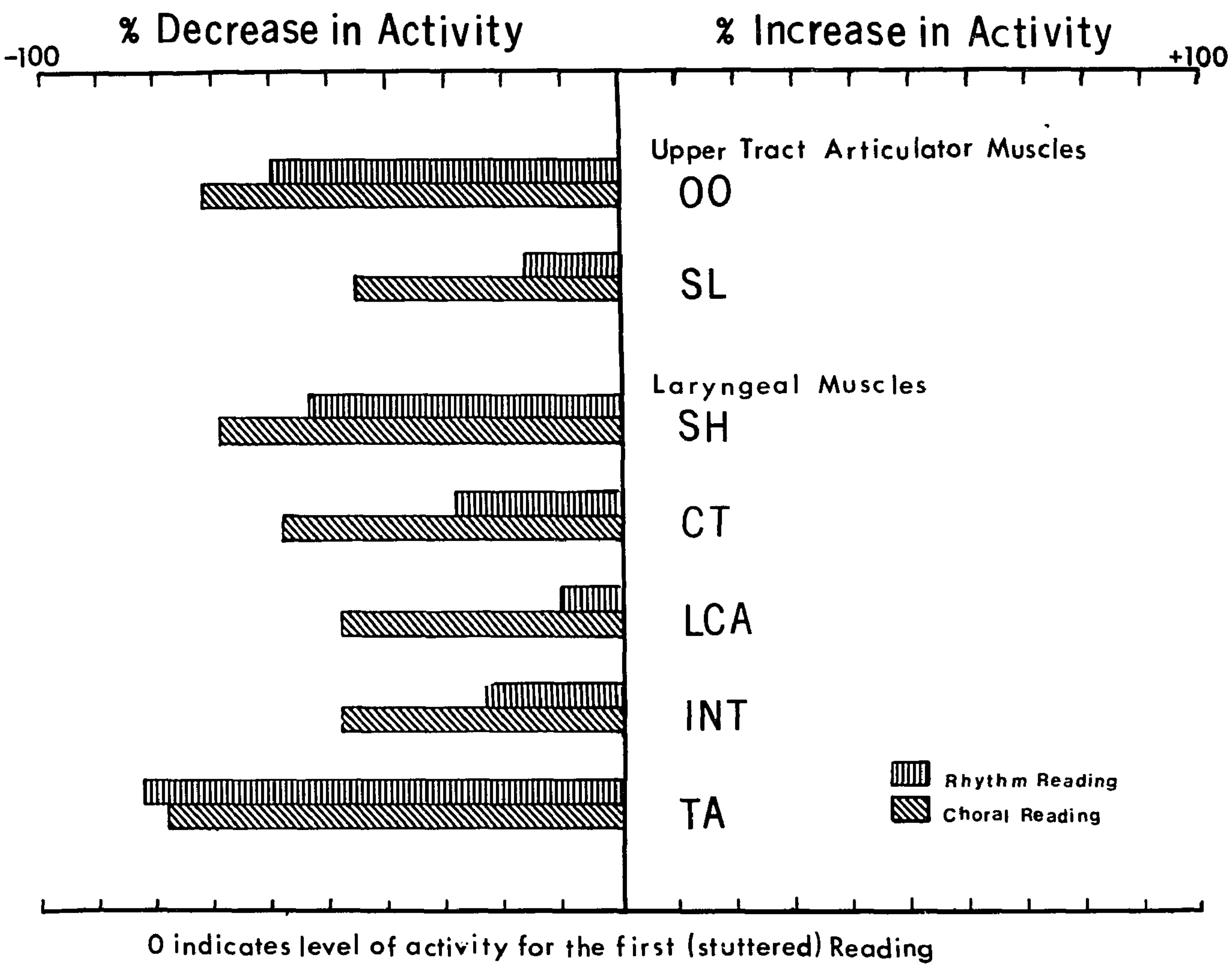
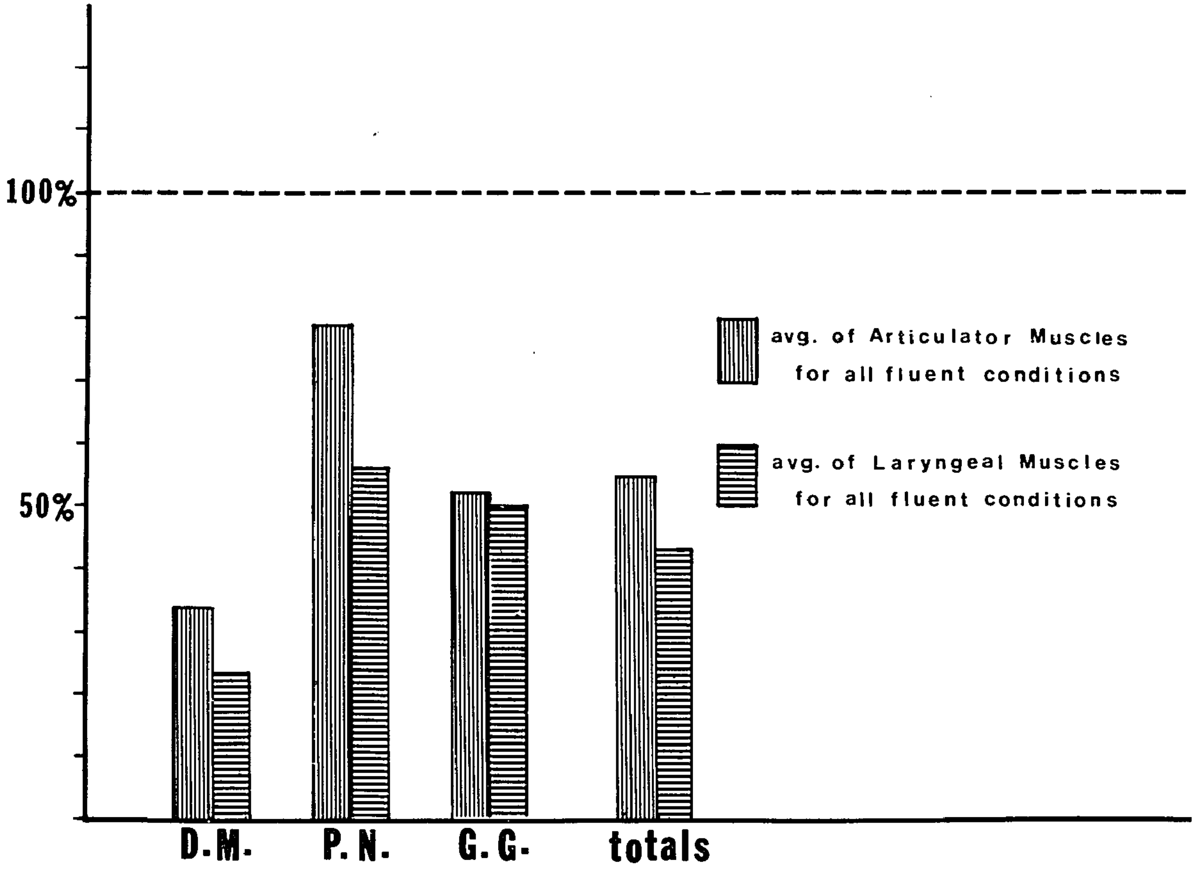

% Decrease in Activity
% Increase in Activity
-100
+100
Upper Tract Articulator Muscles
OO
SL
Laryngeal Muscles
SH
CT
LCA
INT
TA
Rhythm Reading
Choral Reading
0 indicates level of activity for the first (stuttered) Reading

Figure 21

Comparison of average levels per syllable for upper tract articulator muscles and laryngeal muscles.



100% indicates levels for the First (stuttered) Reading the fluency evoking conditions. When Fig. 21 is contrasted with Fig. 17, the differences between the average levels by two-second segments and the average levels by two-second segments and the average levels per syllable become apparent.

The use of the average-level-per-syllable computation results in greater differences between the first (stuttered) reading and the fluency-evoking conditions. Both upper tract muscles and laryngeal muscles show larger per centages of decreased activity. However, the use of the average level per syllable computation effects the upper tract articulator muscles more than it effects the laryngeal muscles. This is particularly true for subject G.G.

Differences in Averaged Peak Values for Stuttered and Fluent Utterances

Since subject C. D. did not stutter while reading the experimental passage, data collected could not be analized in the same way. Instead, C. D.'s 49 utterances of the words "syllables"and "syllables" were utilized to learn whether the peak levels of muscle activity were different for fluent and stuttered utterances. In each utterance, the time period between the initial muscle activity for the production of the voiceless fricative (indicated by activity in the SL for raising the tongue tip and activity in the PCA for opening the glottis), and the point in the acoustic tract which indicated the onset of voicing for the vowel [I] was identified. Within this time period, the highest peak of muscle activity was identified for each muscle. The level of this peak of activity was then computed in microvolts for each muscle for each utterance.

The experimenter, after listening to audio recordings of the words, identified 23 utterances as stuttered and 26 as fluent. The peak levels of muscle activity for the utterances judged stuttered were averaged for each muscle, yielding mean peak values for the stuttered utterances. The peak levels for the utterances judged fluent were similarly averaged, and the means for the stuttered and fluent utterances were compared.

Results (in microvolts) are presented in Table 12. Significant differences between fluent and stuttered utterances were found for all five muscles. Four of the five reached the .001 level of confidence.

Contrasts between the peak levels for the fluent and stuttered utterances are graphically illustrated in Fig. 22. In each case, the average peak levels for the stuttered utterances serve as the reference and the average peak value for the fluent utterances is expressed as a percentage.

TABLE 12

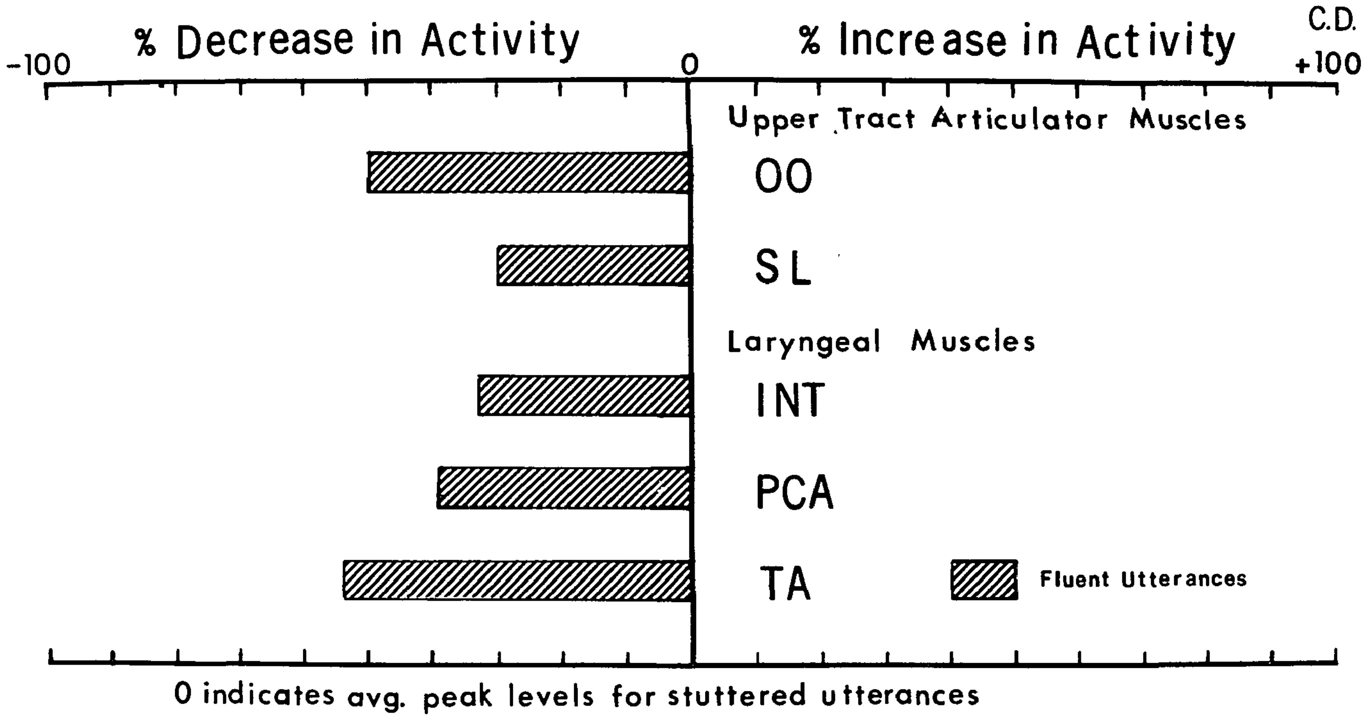
Results for C.D.
Differences in averaged peak values for stuttered and fluent utterances

MUSCLE		STUTTERED	FLUENT
OO	(mean)	244.44	121.89
	(SD)	142.78	57.19
	t =		3.94
SL	(mean)	806.52	562.08
	(SD)	322.31	268.14
	t =		2.84*
IA	(mean)	328.39	218.81
	(SD)	69.67	60.80
	t =		5.76**
PCA	(mean)	288.96	175.65
	(SD)	70.67	73.44
	t =		5.37**
TA	(mean)	101.48	46.27
	(SD)	55.83	14.83
	t =		4.75**

Levels of Confidence: * = .01 and ** = .001

Figure 22

Average of peak values of stuttered and fluent utterances of the words "syllable" and "syllables" spoken by subject C.D.


C.D.
-100
% Decrease in Activity
0
% Increase in Activity
+100
Upper Tract Articulator Muscles
OO
SL
Laryngeal Muscles
INT
PCA
TA
▨ Fluent Utterances
0 indicates avg. peak levels for stuttered utterances

Disruption of Coordination in Stuttered Utterance

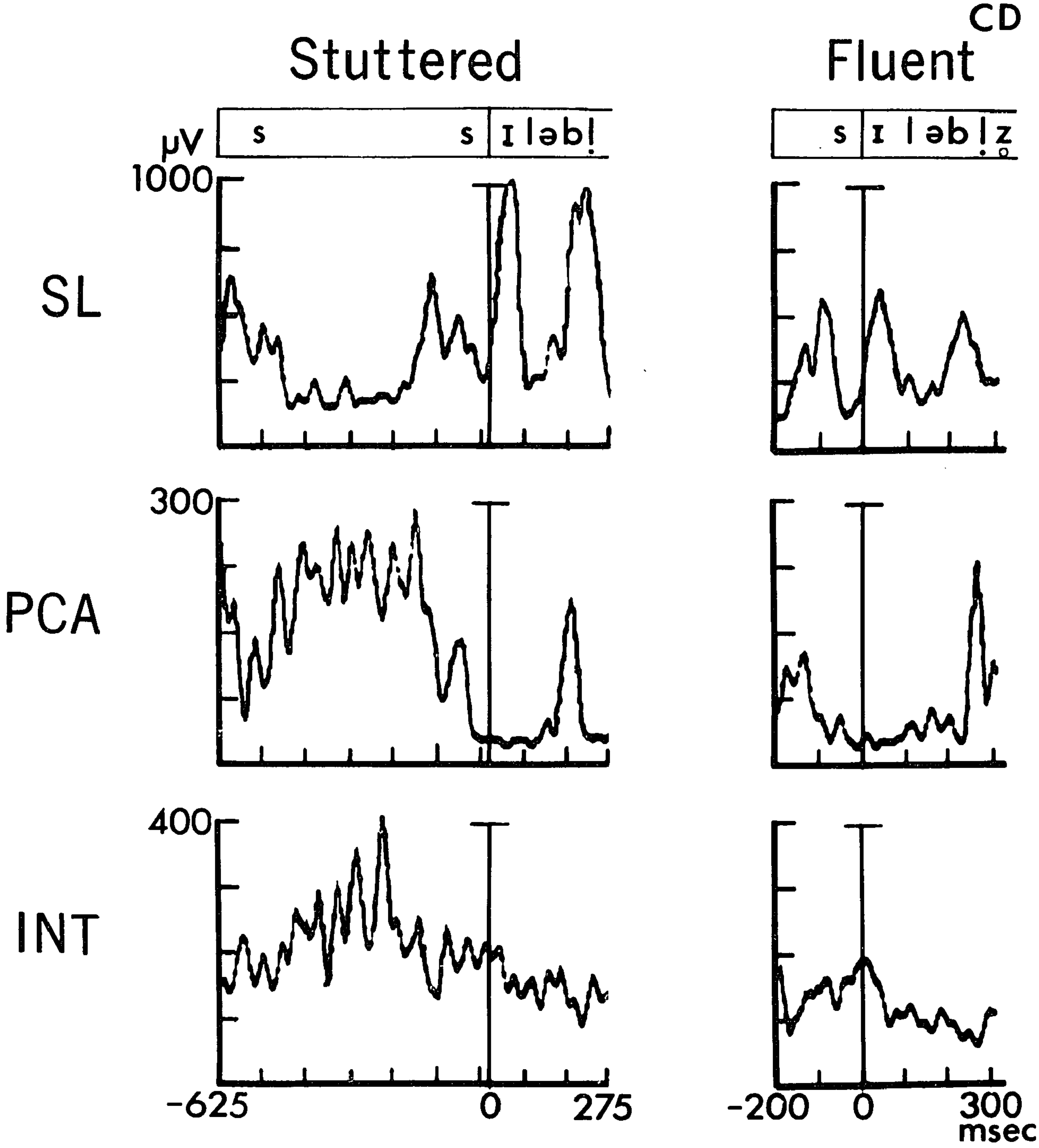
The study of disruption of coordination in stuttered speech is restricted to some extent by our imprecise knowledge of many aspects of coordination in normal speech. On one point, however, studies of normal laryngeal articulation have resulted in relatively clear and consistent findings. These studies indicate that the abductor and adductor forces in the larynx normally act with reciprocity. When the glottal abductor (the PCA) is strongly active, the adductors (INT, TA and LCA) are suppressed, and conversely, when the adductors are strongly active, the abductor is suppressed. Since recordings from the abductor were secured for two of the four subjects, it was possible to investigate the reciprocal activity of the antagonist muscles.

Examples of Disrupted Reciprocity

Figure 23 shows recordings from three muscles for subject C.D., for contrasting stuttered and fluent utterances of the word "syllable." The line-up point for both utterances is the onset of voicing for the vowel. In the bottom graph, the peaks of activity in the SL relate to tongue tip raising. During the stuttered prolongation of the initial voiceless fricative the PCA (glottal abductor) and the INT (glottal adductor) are both active. During the fluent utterance the

Figure 23

Fluent and stuttered utterances of the word "syllable" spoken by C.D.


CD
Stuttered
Fluent
µV
s
s
ɪ l ə b l̩
s
ɪ l ə b l̩ z̥
1000
300
400
SL
PCA
INT
-625
0
275
-200
0
300
msec

antagonist forces act with reciprocity.

Figure 24 shows recordings from three muscles for subject D.M. The graph on the left is from a stuttered utterance of the word "less," while the graph on the right is from a fluent utterance of the same word.

The boxes at the top of the graph contain phonetic symbols and represent the relative length of each segment as measured in oscillographic tracings. The line-up or 0 point on each graph represents the end of voicing for the vowel. In the bottom graph, the peaks of activity for the SL relate to tongue tip raising for the [l] and the [s]. During the prolongation of the [l] sound, the PCA (glottal abductor) and the TA (a glottal adductor) are both active. During the fluent utterance these two muscles show reciprocal activity.

Figure 25 shows three utterances of the word "ancient," with progressive adaptation from a strong block to a mild block to a fluent utterance. During the prolongation of the [e] in the strong block, the PCA (glottal abductor) and the TA and the LCA (glottal adductors) are all active. During the fluent utterance the antagonist muscles act with reciprocity. What is happening in the mildly stuttered utterance is not completely clear, but overlapping co-contraction of antagonist forces may be involved.

Correlations of Abductor (PCA) and Adductor (INT) Activity in Fluent and Stuttered Utterance

The first syllable of the word "syllable" has phonetic content

Figure 24

Fluent and stuttered utterances of the word "less" spoken by D.M.

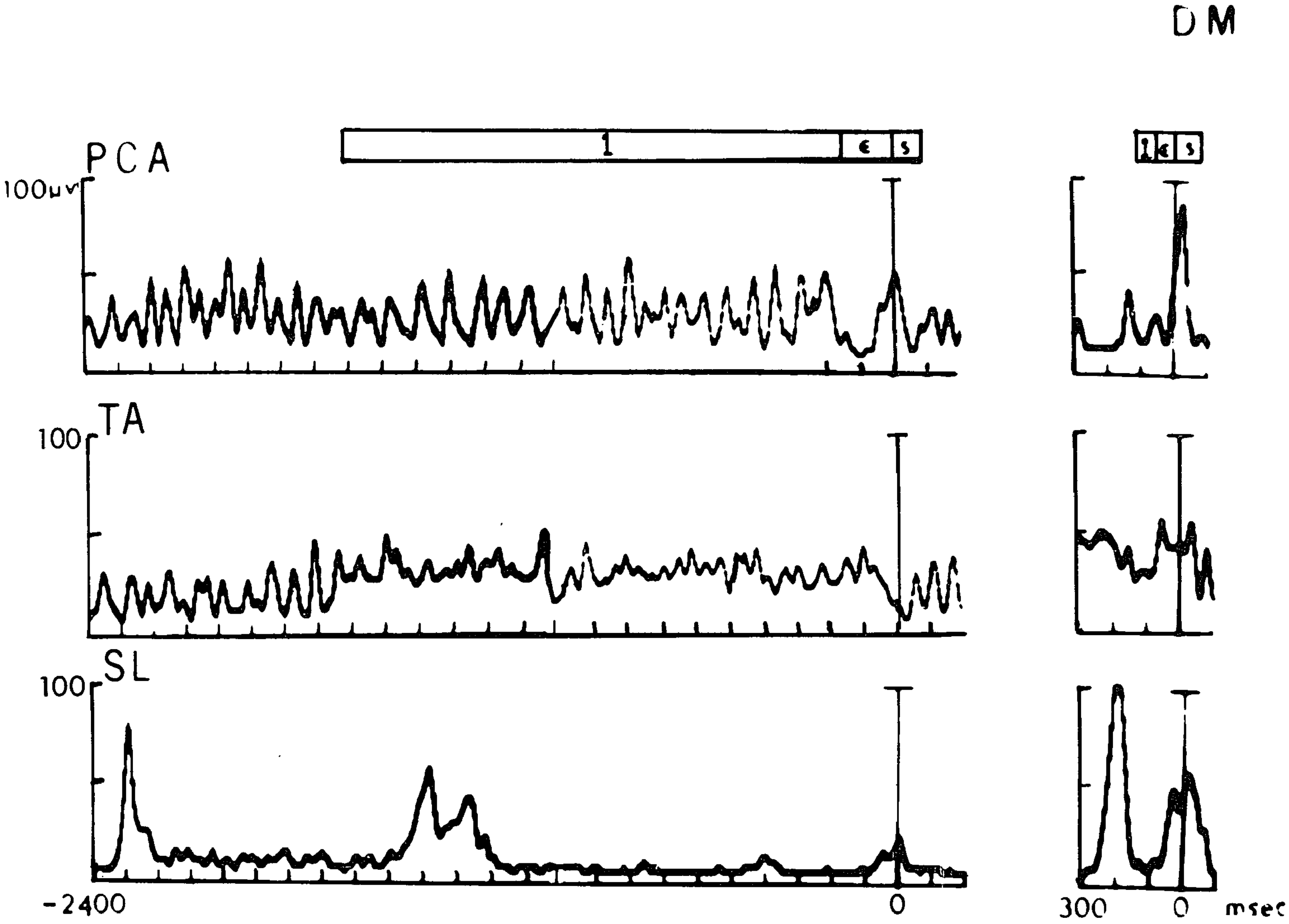
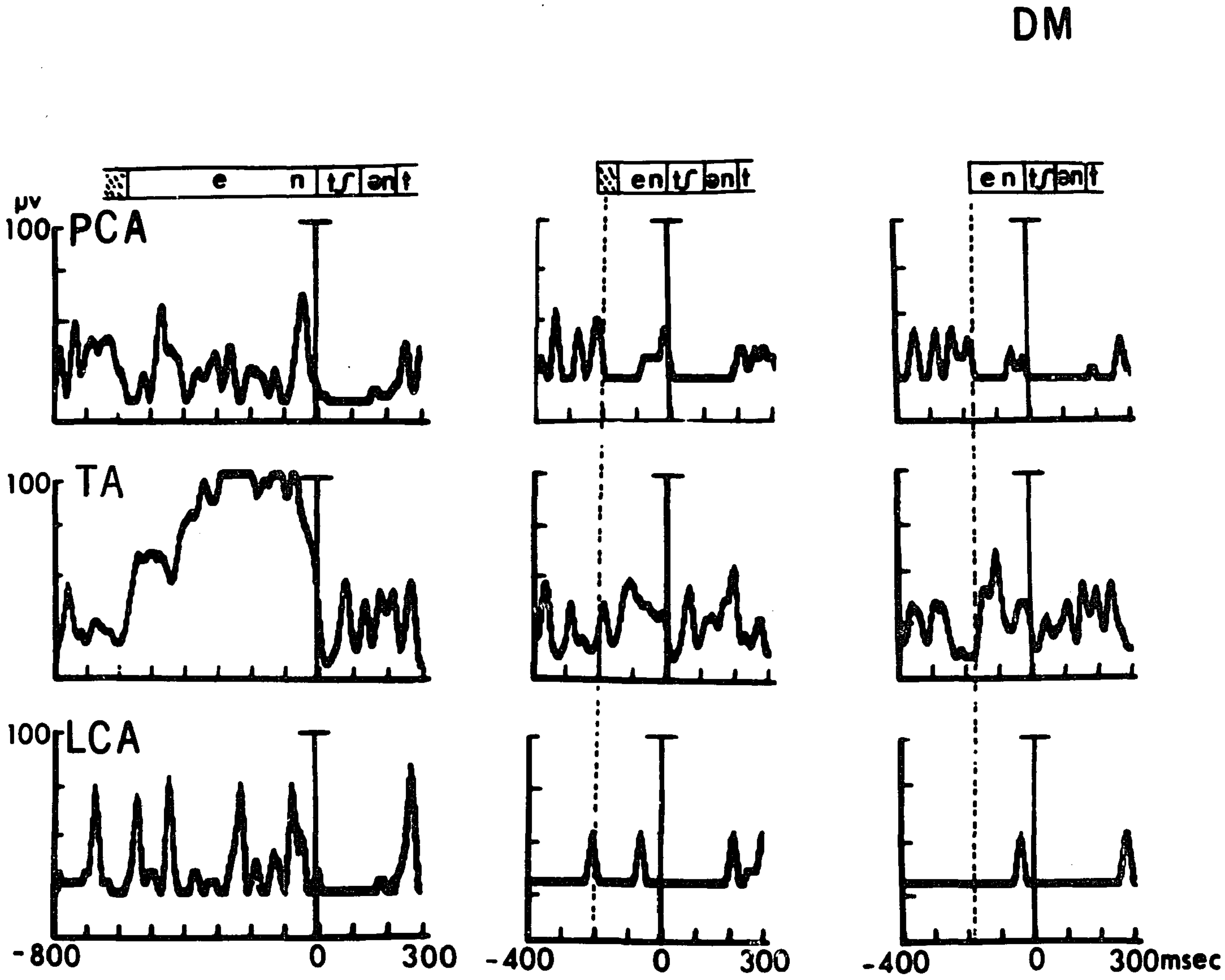

DM
1
ɛ
s
PCA
100µv
TA
100
SL
100
-2400
0
300
0
msec

Figure 25

Fluent and stuttered utterances of the word "ancient" spoken by D.M. The first utterance was strongly stuttered, the second mildly stuttered, and the third was fluent.


DM
e n tʃ ən t
en tʃ ən t
e n tʃ ən t
μv
100
PCA
100
TA
100
LCA
-800
0
300
-400
0
300
-400
0
300msec

suitable for a correlation study of PCA-INT activity. During the first segment of the syllable, the PCA is active and the INT is suppressed for the production of the voiceless fricative. The INT is then active while the PCA is suppressed for the production of the vowel. This pattern is shown in the fluent utterance of Fig. 23. If this normal activity of these antagonist muscles were correlated over time, a negative correlation should result. And, indeed, the plotting of such a correlation for the fluent utterance in Fig.23 yields an *r* of -.83. Conversely, the plotting of the correlation between the INT and the PCA for the stuttered utterance in Fig. 23 yields an *r* of +.80.

The program (E$MGCORL, Kewley-Port, 1973) used for these calculations plotted and correlated points at 5 millisecond intervals. Correlations were plotted for the time period between the first activity of the SL and PCA for the [s] and the onset of voicing for the vowel [I]. Coefficients of correlation were calculated for 49 utterances of the words "syllable" and "syllables," As previously discussed, the experimenter had judged 23 of these utterances to be stuttered and 26 to be fluent.

Of the 23 utterances judged stuttered, 20 yielded positive correlations and 3 yielded negative correlations; while of the 26 utterances judged stuttered, 19 yielded negative correlations and 7 yielded positive correlations. These findings are presented in Tables 13 and 14 and are graphically illustrated in Fig. 26.

In Fig. 26, the 23 stuttered utterances are shown on the top half of the graph; while the 26 fluent utterances are shown on the lower half. All positive correlations are shown to the right of center,

TABLE 13

Results for C.D.
Correlations of abductor (PCA) and adductor (IA) activity in utterances judged stuttered. Words studied were "syllable" and "syllables"

Utterance Number	Speaking Condition	Stuttering Pattern	Correlation
1.	Conversation	/ss/	+ .8039
2.	Conversation	/-ss/	+ .8038
3.	Conversation	s-s/	+ .7154
4.	DAF	/ss/	+ .7030
5.	Conversation	/-s/	+ .6804
6.	Conversation	/sasasas/	+ .6638
7.	Conversation	/-s/	+ .6263
8.	Conversation	/-s/	+ .5814
9.	Conversation	/ss/	+ .5143
10.	Conversation	/ss/	+ .4968
11.	Conversation	/-s/	+ .4810
12.	Conversation	/ss/	+ .4229
13.	Conversation	/s-s/	+ .4192
14.	Conversation	/ss/	+ .3922
15.	Conversation	/-s/	+ .3775
16.	DAF	/-s/	+ .3736
17.	Noise	/ss/	+ .3373
18.	Conversation	/ss/	+ .3123
19.	Noise	/ss/	+ .2507
20.	Noise	/ss/	+ .0147
21.	Conversation	/ssas/	- .0348
22.	Conversation	/ssss/	- .0625
23.	Conversation	/ssss/	- .3835

Prolongation or repetition of voiceless fricative -- /ss/
Longer prolongation or repetition of voiceless fricative -- /ssss/
Pause /-s/
Repetition of [s] followed by a brief vowel -- /sasa/

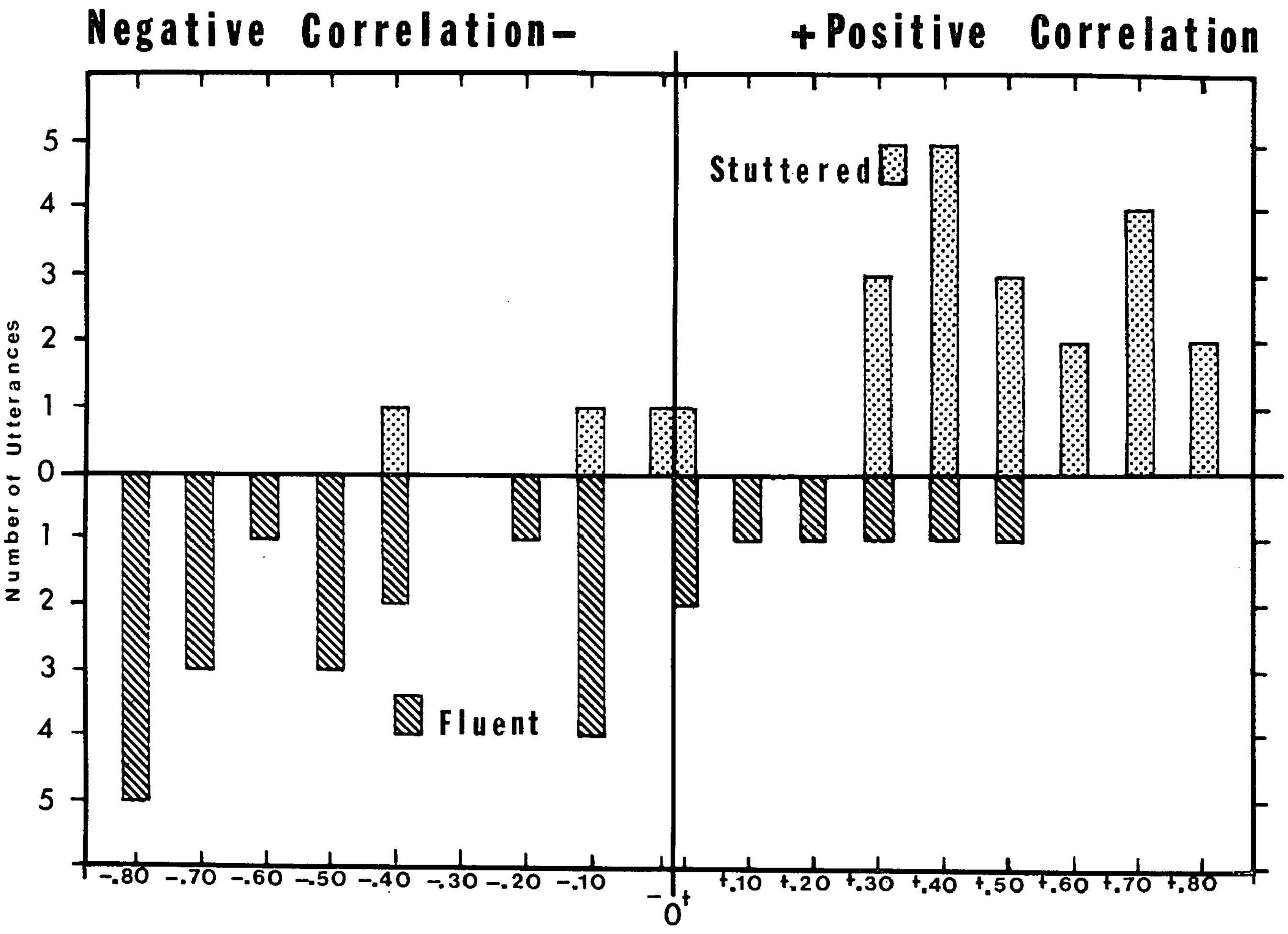
TABLE 14

Results for C.D.
Correlations of abductor (PCA) and adductor (IA) activity in utterances judged fluent. Words studied were "syllable" and "syllables"

Utterance Number	Speaking Condition	Correlation
1.	Noise	- .8268
2.	Noise	- .8139
3.	Noise	- .7938
4.	Noise	- .7774
5.	Rhythm	- .7488
6.	DAF	- .7360
7.	Rhythm	- .7319
8.	Noise	- .6815
9.	Conversation	- .5667
10.	DAF	- .5443
11.	Noise	- .5372
12.	Choral	- .4644
13.	Conversation	- .3892
14.	Choral	- .3643
15.	Conversation	- .2070
16.	Choral	- .1132
17.	Rhythm	- .1129
18.	Rhythm	- .1103
19.	Choral	- .0719
20.	Conversation	+ .0184
21.	Rhythm	+ .0264
22.	DAF	+ .0633
23.	Conversation	+ .2423
24.	Rhythm	+ .2904
25.	Conversation	+ .3972
26.	Conversation	+ .4860

Figure 26

Abductor-adductor correlations for fluent and stuttered utterances of C.D.


Negative Correlation–
+Positive Correlation
Number of Utterances
5
4
3
2
1
0
1
2
3
4
5
Stuttered ▨
▨ Fluent
–.80
–.70
–.60
–.50
–.40
–.30
–.20
–.10
–0+
+.10
+.20
+.30
+.40
+.50
+.60
+.70
+.80

NOTES

1. Level differences between fluent and stuttered utterances were so great that it was difficult to display them on graphs using the same maximum microvolt values. Activity patterns in the fluent utterances were minimized by the high scale value settings; while the stuttered utterance displays were "off the graph" if low scale values were used. The compromise values used are not totally satisfactory for either display. The apparent peak clipping in the stuttered utterances are the result of display level settings not processing errors.

2. Hirose and Gay (1973) studied the effects of speaking rate on labial consonant production and reported that, "the major effect of an increase in speaking rate on labial consonant production activity is a generalized increase in the activity levels of the muscles, this in turn indicates an over all increase in articulatory effort for these consonants during faster speech.

CHAPTER IV

DISCUSSION

and negative correlations to the left. The probability of these patterns (based on a sign test) is less than .01 and .05 respectively.

There remains a question of why some of the utterances judged fluent did not show the "normal" negative correlation. One simple explanation is that the experimenter was unable to recognize all of the stuttering blocks from the audio tape, and thus erroneously judged some stuttered utterances as fluent.

The question of why three of the utterance judged stuttered showed negative correlations is more complex: Column three of Table 13 shows the stuttering pattern associated with each of the 23 utterances judged stuttered. Four different types of blocks were recorded. The most commonly occurring pattern was a simple prolongation or repetition of the voiceless fricative. Transcribed as [ss], this pattern occurred in 10 of the stuttered utterances. A simple pause, followed by the utterance of the word, transcribed as [-s], occurred in six stuttered utterances. A combination of prolongation, repetition and pause occurred in three cases. The remaining four blocks were the longest in duration. Two of these long blocks consisted of repetitions of the voiceless fricative, and were transcribed as [ssss]. The other two were repetitions of [s] followed by a brief vowel, and were transcribed as [sasa]. The three stuttered utterances which failed to show negative correlations were all long blocks of the last two types. The procedures followed in designating the time period over which the correlations were calculated may not have been optimal for these very long blocks. Indeed, only the final portions of the total moments of stuttering were correlated for these blocks. It may be that the initial portion of each block is the critical point for the disruption of agonist-antagonist reciprocity. Future work should include correlation studies in which this possibility is investigated.

General Considerations

Although some movements of anatomical structures occur because of non-muscular forces (such as gravity, air pressures, or tissue elasticity), most changes in structure position occur in response to muscle contraction and relaxation. Through electromyographic investigation of the laryngeal muscle activity accompanying stuttering, this research sought first to describe the activity, and second to understand which of the behaviors collectively termed "stuttering" resulted from the activity.

The review of the literature suggested two types of relevant muscle activity which might be investigated through electromyography: (1) tension levels and (2) coordination. Clearly abnormal tension levels might be found in (1) laryngeal muscles and/or (2) upper tract articulator muscles.[1] Coordination, on the other hand, might be disrupted on three levels: (1) intra-laryngeal, (2) intra-articulator, or (3) inter-laryngeal-articulator.

The present study investigated laryngeal tension effects and intra-laryngeal coordination effects. Limited data were collected on selected upper tract articulator muscles and tension effects in these muscles are briefly discussed. Intra-articulator and inter-laryngeal-articulator coordination are not dealt with in the present study.

Findings relating to the four experimental subjects are discussed first, and then the implications of such findings are considered in relation to stuttering.

Conclusions Relating to the Experimental Subjects

The results presented in the preceding chapter clearly demonstrate the existence of a laryngeal component in the stuttering patterns of each of the four experimental subjects. Analysis of the EMG data provided evidence on two aspects of laryngeal muscle activity accompanying stuttering--(1) tension levels and (2) disrupted coordination.

Findings Related to Tension

Laryngeal Tensions

For all four subjects the levels of laryngeal muscle activity accompanying stuttered speech were higher than those accompanying comparable fluent utterances. Most of the information relating to tension levels was derived from comparisons of stuttered readings and fluent readings, in which the fluency resulted from use of one of the fluency-evoking conditions (rhythm reading, choral reading, DAF and white noise). In all cases the fluency-evoking conditions were accompanied by reduction in levels of laryngeal muscle activity and reduction in frequency of stuttering.

Although the data did not permit comparable study of fluent passages resulting from adaptation words on which adaptation occurred were examined (Figs. 13, 23, 24, 25, and also show that the fluent (by adaptation) utterances were accompanied by lower levels of laryngeal muscle activity.

The tension levels averaged across time segments (two seconds' duration) might be higher for stuttered speech because of any one of three alternative reasons: (1) individual moments of stuttering are accompanied by sudden bursts of laryngeal muscle activity which reach high levels, but levels are normal between such bursts; (2) the stutterer when speaking (in his usual manner) maintains generally higher base levels of laryngeal muscle activity; or (3) the stutterer when speaking in his usual manner maintains generally high base levels of muscle activity and these generally high levels rise even higher during individual moments of stuttering. It is not possible to make any definitive differentiation between these three hypotheses from the data analysis used in this study. However, some enlightened observations are possible. Examination of the processed EMG data comparing the stuttered and fluent utterances (Figs. 12, 13,and 23, 25) clearly support the contention that individual moments of stuttering are accompanied by sudden bursts of high levels of laryngeal muscle activity. Further, the comparison of average peak levels for fluent and stuttered utterances for subject C.D. (Table 12 and Fig. 22, could be interpreted as demonstrating the higher levels for individual moments of stuttering.

On the other hand, examination of the raw EMG (Figs. 9, 11, for passages of stuttered and fluent speech suggests that the higher levels are present throughout the first (stuttered) reading. Either there is a generally higher level of tension which is maintained throughout, or there are many bursts of higher levels of activity occurring more frequently than identifiable moments of stuttering occur. On the basis of the existing analysis of data, the third hypothesis, suggesting a combination of generally high levels and sudden bursts of higher levels, appears most likely.

<u>Effects of excessive laryngeal muscle tension</u>. The levels of muscle activity recorded in the stuttered speech samples are deemed to be excessive for two reasons. First, they may be considered excessive because they were significantly higher than those levels which were "necessary" to produce the desired articulator movements and vocal tracts shape changes for the segmental content of the experimental passage. Since the low levels recorded in the fluent conditions were adequate to produce clearly intelligible, adequately loud[2] speech, these levels may be considered representative of the "necessary" activity for articulation and phonation.

Second, the levels of the adductor muscles are considered excessive because they can be demonstrated to equal or exceed the levels necessary for tight glottal closure. For each of the subjects, levels of activity in the adductor muscles during moments of stuttering frequently equaled the levels recorded for that subject during reflexive protective laryngeal closure. Comparison of Fig. 3 with Figs. 13, 24 and 25, illustrates this finding. If a given level of adductive muscle activity is sufficient for the tight glottal closure of swallowing or coughing, it is almost certainly excessive for normal phonation.[3] In the light of these observations, it is important to consider the effects of excessive phonatory and articulatory muscle tensions.

At the level of the larynx, phonation may be prevented in at least three ways: (1) the vocal folds may be too tightly closed (excessive adduction); (2) the vocal folds may be too far apart (excessive abduction); and (3) the vocal folds may be properly approximated, but so rigid as to prevent normal interaction with subglottal and supraglottal air pressure (excessive tension). Excessive adduction, excessive abduction, and excessive vocal fold tensions are produced by high levels of laryngeal muscle activity. It follows then that excessively high levels of laryngeal muscle activity will result in prevention or interruption of normal phonation.

Normal phonation involves a delicate balance of laryngeal muscle forces. Further, segmental speech articulation requires rapid and continuous adjustments in the fine balance of these muscle forces. Any factor (including excessive laryngeal muscle tensions) which might interfere with the delicate balance of laryngeal muscle forces, or with their rapid adjustments during ongoing speech, has the potential for disrupting fluent utterance.

Causes of excessive laryngeal muscle tension. Although the physiological precipitants of laryngospasm have been studied electromyographically (Whiddicomb, 1974)[4], with one peripheral exception (Shipp, 1973), EMG has not been used to study psychologically or emotionally induced tension effects in laryngeal muscles. The anecdotal evidence of laryngeal and/or respiratory response to emotionally charged states is so well known as to make any discussion redundant. Fear, joy, anger, any intense emotion, may lead to "speechlessness," "loss of voice," being "struck dumb," etc. The phylogenic, physiological reasons for laryngeal response to intense emotion are found in the "fight-flight" reflex and are discussed by Negus (1949). The possible relationship of this reflex to stuttering is discussed by Van Riper (1971).

Upper Tract Articulator Muscle Tensions

For each of the subjects, the muscle showing the greatest level reduction (from stuttered to fluent utterance) was always a laryngeal adductor muscle (TA for C.D. and G.G.; LCA for D.M. and INT and LCA for P.N.). There were, however, marked individual differences as to which muscles showed significant level differences and which muscles showed minimal differences. Although there was a trend for the laryngeal muscles as a group to show greater level reduction effects than the upper tract articulator muscles as a group, conclusive statements on this point are not possible at present.

Effects of upper tract articulator tensions.

It is not clear if, or how, high levels of muscle activity in the upper tract articulator muscles would exert a disruptive effect on speech comparable to that of excessive laryngeal tension. It is possible to speak (albeit with some loss of intelligibility) while engaging in tasks which generate high levels (higher than normal speech levels) of activity in jaw, tongue, and lip muscles (i.e., biting, chewing, puckering). It is certainly true, however, that excessive tension in these muscles may disrupt their coordination.

Findings Related to Coordination

The findings of co-contraction of abductor and adductor muscles (illustrated in Fig. 23-24) are of special significance for five reasons: (1) based on the existing studies of normals, the phenomenon does not appear to occur in normals during fluent utterance;[5] (2) the phenomenon violates the principles of normal coordination; (3) the activity pattern is clearly counter-productive in relation to achieving an appropriate glottal speech gesture; (4) in the stutterers, the occurrence of the phenomenon correlates closely with perceived stuttering blocks; and (5) the phenomenon would be expected to disrupt normal phonation.

The results of studies on normals, which show highly consistent reciprocity between abductor-adductor muscles have already been reviewed, as have studies on other muscle systems, indicating that normal coordination involves reciprocal inhibition of antagonist muscles. The counter-productive effects of simultaneously attempting to abduct and adduct the vocal folds are self-evident. The relationship between disruption of reciprocity and perceived blocks was demonstrated for subject C.D. (Tables 13 and 14; Fig. 26). A similar test was not possible for D.M., but early in the analysis of data on D.M., the experimenter, working to transcribe phonetically the audio recordings onto the processed EMG, discovered that stuttering blocks could be identified on the EMG printouts by looking for the occurrence of co-contraction.

For both D.M. and C.D. there were times when co-contraction occurred at points where stuttering blocks were not identified by listeners. (Note the six examples of words judged to be fluent for C.D., but found to show positive INT-PCA correlations, Table 14 and Fig. 26). There are instances for both C.D. and P.N., where co-contraction appears to occur during a silent period just prior to an utterance. When co-contraction occurs during sound production, audible disruptions generally occur. Observations relating to the perceptual and acoustic correlates of abductor-adductor co-contraction therefore seemed appropriate.

All of the words C.D. reported as "difficult," and all of his words identified as stuttered, were words whose phonetic content required an initial shift from voicelessness to phonation. The co-contraction of abductor-adductor (PCA-INT) muscles occurred either during (1) silence or (2) during prolongation or repetition of the initial voiceless consonant. The termination of the co-contraction was almost invariably followed (50 to 150 msec) by the onset of the vocalic element. Interpretation of the EMG evidence therefore suggests that the effect of the co-contraction was to prevent, or at least momentarily delay or inhibit, the normal transition from voicelessness into the periodic vocal fold vibration of vowel phonation. The inference is that the vocal folds will not vibrate normally if muscular forces are simultaneously acting to pull them together and to pull them apart. It follows that C.D. was not blocking on the production of the consonantal element which he produced repeatedly. The problem was the transition into phonation.

Normal, fluent utterance of a CV syllable requires a specific change of laryngeal muscle tension pattern (this is true even if the consonant is voiced), and a specific change in glottal state (glottal constriction is different for consonants and vowels), within a limited time frame. Disruption of coordination (in this case the abductor-adductor reciprocity) can prevent the accomplishment of the gesture within the required time frame, thus preventing normal fluent utterance. At this physiological level, cause and effect are relatively straightforward.

In the light of these observations, the work of Adams and his colleagues (Adams and Reis, 1971 and 1974; and (Adams, et al., 1974), should be re-evaluated. They have demonstrated that the residual blocks (blocks which remain after adaptation has occurred) tend to occur on words requiring an initial shift from voicelessness into phonation. They concluded that initiation of phonation may indeed constitute a primary source of difficulty for stutterers. It is possible that in C.D. we are observing the physiological mechanism which underlies many stutterers' difficulties in initiation of phonation.

With D.M. it was possible to observe the effects of co-contraction of abductor-adductor (PCA, LCA and TA) muscles during prolonged voiced segments, as for example the prolonged [l] of "less" illustrated in Fig. 24. Although C.D. was able to initiate phonation in spite of abductor contraction, the sound produced differs from normal phonation. Spectrograms and oscillographic tracings of his vowel prolongations (for example, the prolonged [e] of "ancient in Fig. 25), show breaks in phonation, irregular periods, and abrupt shifts in amplitude. Similar abnormalities have been reported in acoustic studies of stutterers (Agnello, 1971; Van Riper, 1971; Stromstra, 1965: and Webster, 1975). Vocal fry frequently occurred in D.M.'s prolongations a finding reported for other stutterers by Adams (1974) and Van Riper (1971). Listeners described D.M.'s prolongations as sounding "tense" or "strained."

Contour, Brewer and McCall (1974), after fiberoptic investigations of glottal configurations during stuttering, suggested that they had observed instances in which "laryngeal adductors and abductors are simultaneously and/or alternately contracting in a manner that results in a fluency disruptive laryngeal tug-of-war...". The present findings tend to support and verify this description.

Causes of Disrupted Reciprocity.

As discussed in Chapter I, co-contraction of antagonists has been found to occur: (1) under stress, (2) in rapid movements, (3) in unskilled subjects, (4) in infants and young children, and (5) in neurological impairment. It is notably absent during rhythmic activities. The similarity in this list of conditions for disrupted reciprocity and the conditions under which fluency is most likely to be disrupted in striking. The finding by Kozmyan (1965) that "the dissociation of reciprocal inhibition" is diminished with "rhythmic repetitive movement" suggests a neurophysiological mechanism which might account for the ameliorative effects of rhythm on stuttering.

Relationships between tension and coordination

Coordination in general is related to levels of muscle activity. Those concerned with the teaching and performance of skilled motor tasks (singers, dancers, athletes, musicians, typists) have traditionally felt that excessive tensions were counter-productive, and have sought to improve performance through reduction of tensions. As discussed in Chapter I, EMG studies (Basmajian, 1974) have demonstrated that high levels of muscle tensions may lead to disruption of the fine coordination of agonist-antagonist forces. It is likely then that one effect of the excessive muscle tension of stuttered speech may be the disruption of coordination. This may be true in one or both of the muscle groups (laryngeals and upper tract articulators) studied.

For the laryngeal muscles an alternative relationship is also plausible. It might be argued that high levels of muscle activity are generated as the system attempts to overcome the disruptive effects of co-contraction of the antagonists. The two hypotheses are not mutually exclusive and may indeed act to reinforce one another.

Summary

For these four subjects, stuttering was found to be accompanied by abnormal laryngeal muscle activity including (1) excessively high levels of muscle activity and (2) disruption of normal abductor-adductor reciprocity. These demonstrated patterns of abnormal laryngeal muscle activity appear to be incompatible with normal phonation or with fluent production of sequences of phonetic segments which require rapid coordinated changes in glottal configuration. The abnormal activity levels in laryngeal muscles and/or the co-contraction of abductor-adductor forces are sufficient[6] to produce disruption of normal fluent speech, and may be considered proximal causes of some of the observable behaviors collectively labeled stuttering.

Conclusions Relating to the Nature of Stuttering

The EMG experiments have established the existence of a laryngeal component sufficient to produce interruption of normal phonation, breaks in phonation, temporary inhibition of phonation, or temporary inability to initiate phonation.

If we accept the models of stuttering recently offered by Bloodstein (1974) and Van Riper (1971), it follows that a laryngeal component may be sufficient to account for the critical or core behaviors of stuttering. Bloodstein describes the "deeper level of the stutterer's behavior as consisting of tensions and fragmentations;" while Van Riper "finds the essence of the disorder in this fracturing and disruption of the motor sequence of the word," or in the "broken word." Bloodstein's tension component is fully supported by the present research. Further, the obvious outcome of interrupted phonation or inability to initiate phonation would be "fragmentation," "fracturing" or "breaking" of words.

Whether such abnormal laryngeal muscle activity does indeed produce the "fragmentations" and "fractured words" of most, or all stutterers, is of course another matter. Generalization from EMG findings on only four subjects is in many respects questionable. The

EMG experiments do however take on additional significance when viewed in relation to the other physiological studies of laryngeal functioning in stuttering (Chevrie-Muller, 1963; Fujita, 1966; Ushijima, Kamiyama, Hirose and Niimi, 1965; Contour, Brewer and McCall, 1974).

The EMG findings are compatible with, and indeed supplement the findings of these studies. The picture which emerges from these experiments, independently conducted, utilizing a variety of instrumentation with stutterers speaking three languages, is highly coherent. They plainly suggest that laryngeal involvement in stuttering is not an isolated or idosyncratic phenomenon.

In most cases, the present study has verified the hypotheses generated by those researchers who utilized indirect approaches for studying phonation in stuttering. Adams and Reis (1971 and 1974), Riemenschneider (1974), Agnello (1974) and Adams and Hayden (1974), all predicted the initiation-of-phonation problem, found for subject C.D. From oscillograms Webster (1975) predicted "deviant tensions" in laryngeal muscles and "excessive or unstable" vocal fold tensing. If these investigators are correct in their interpretations, then they are observing indirectly in their subjects the same types of abnormal laryngeal muscle activity studied directly in the present research.

Perhaps the best evidence that the laryngeal component is a factor in the stuttering patterns of large numbers of stutterers is found in the studies of fluency evoking conditions. The "almost universal" reduction in dysfluencies under these conditions is well documented. The EMG experiments support Wingate (1969 and 1970) in his hypothesis that the fluency evoking conditions effect changes in the manner of vocalization. When experimental subjects spoke under the fluency evoking conditions, laryngeal muscle tensions were significantly reduced and fewer instances of abductor-adductor co-contraction occurred. Since these are the effects of the fluency evoking conditions on these four subjects, it is at least probable that similar laryngeal effects occur in other stutterers speaking under the fluency-evoking conditions. The effects of the fluency-evoking conditions on normal speakers is certainly worth further investigation as a means of understanding their ameliorative effects on stuttering.

At a different level these results raise the question of "why" the fluency evoking conditions lead to reduction in levels of laryngeal muscle activity. There is some preliminary evidence which suggests that the fluency-evoking conditions also produce changes in laryngeal muscle activity in normal speakers (Dorman, Freeman, and Borden, 1975).

This investigator believes that the recent findings of longer vowel durations in fluency evoking conditions (Brayton, 1975) is related to the present research findings on laryngeal muscle activity patterns, but the precise manner in which these two interact is not apparent.

Summary

Results of these experiments indicate that stuttering is accompanied by abnormal laryngeal muscle activity which includes (1) excessive tension and (2) disruption of coordination. As a proximal cause of stuttering, these activity patterns are sufficient to produce behaviors which have been described as the essence of the disorder--tension and fragmentation (Bloodstein, 1974) and fracturing or broken words (Van Riper, 1971). They complement and supplement the existing physiological studies of laryngeal behavior in stuttering, and support many of the hypotheses generated by investigations of phonation in stuttering.

They do not preclude the existence of other proximal causes of stuttering behaviors, including upper tract articulator tension or coordination disruptions. They are compatible with a number of the well-known theories of stuttering, but may require some reorganization of priorities in both theory and therapy.

NOTES

1. Disruption could of course also occur within the respiratory or in the coordination of respiration with phonation and articulation.

2. In two of the fluency evoking conditions (DAF and white noise masking) the subjects spoke more loudly than they did in the first (stuttered) reading. Under the fluency evoking conditions intonation and stress patterns were perceptually different, however, systematic analyses of these variables were not conducted. These subjects, like those studied by Brayton (1975), showed increased vowel durations in the fluency evoking conditions.

3. Systematic study of exact level differences between speech and tight, reflexive glottal closure has not been conducted on normally fluent subjects.

4. Widdicomb also reported that individuals with asthma were predisposed to laryngospasm.

5. A current series of experiments (Dorman, Freeman, and Borden, 1975) were undertaken in order to investigate effects of fluency-evoking conditions, including DAF, on laryngeal activity in normals. Preliminary results indicate that the laryngeal muscle activity accompanying the dysfluencies of normals under DAF differs markedly from the laryngeal muscle activity accompanying stuttering.

6. The conclusion that the laryngeal component is a sufficient cause does not imply that it is an exclusive or even a necessary cause.

APPENDIX A

EXPERIMENTAL PASSAGE

Quotations on Science

The origin of science is in the desire to know causes; and the origin of all false science and imposture is in the desire to accept false causes rather than none; or which is the same thing, in the unwillingness to acknowledge our own ignorance.

Science, like life, feeds on its own decay. New facts burst old rules; then newly divined conceptions bind old and new together into a reconciling law.

The aim of science is to seek the simplest explanation of complex facts. We are apt to fall into the error of thinking that the facts are simple because simplicity is the goal of our quest. The guiding motto in the life of every natural philosopher should be, "Seek simplicity and distrust it."

APPENDIX B

INSTRUCTIONS

Step 1: Listen to the tape, and mark (in pencil) the nonfluencies using the following system. Listen to each segment until you are satisfied you have identified all the blocks.

Repetitions of phrases (two or more words) -- Place brackets [___] around the words repeated. If they are repeated more than once, place an additional set of brackets for each repetition.

Insertions of extra words or sounds - If recognizable words words, or transcribable sounds (not part of the text) are added, write these in between the appropriate syllables. If nonphonetic sounds (clicks, smacks, etc.) are heard, place small x symbols in the appropriate place.

Omissions -- Circle syllables or words omitted. If the omission is felt to be as a stuttering block, also underline the syllable.

Repetitions of words or part words -- Underline the word or syllable repeated.

Prolongations -- Underline the syllable in which the prolongation occurs.

Hard attack - Underline the syllable.

Pause -- Underline the syllable following the pause.

Other -- If another type of nonfluency or stutter occurs involving more than a single syllable, place parentheses (____) around the syllables involved and write a note describing the nonfluency. If another type of nonfluency or stutter occurs involving a single syllable, underline that syllable.

Step 2: In the column labeled syllable number, write the number of each underlined syllable. Nonfluencies involving two or more words are not underlined, and will not be listed here. Make a count to be sure that every underlined syllable is listed in the column.

Step 3: In the column labeled level of confidence you will indicate how certain you are that this was actually a block. Use the following scale:

1. Possible block (but somewhat questionable)

2. Almost certainly a block
3. Clearly and undoubtedly a block

You may listen to the tape as many times as you feel necessary. It is essential that every underlined syllable be listed in the first column and rated in the second column.

Step 4: In the blanks laveled description of the block, use the following categories:

Repetition
Prolongation
Hard attack
Pause
Other

After the categories of "repetition, prolongation, and hard attack" indicate the phoneme involved. After the category "other," write your description. If more than one category applies to a single syllable, use multiple descriptions. For example: "repetition [k]; prolongation [e]" or "pause; hard attach [f]." For each syllable there must be at least one description.

Step 5: If the x symbol was used for an inserted sound, write a note describing what was heard.

Subject: ______________________ Evaluator: ______________________

Reading: ______________________ Date: ______________________

1	2	3	4	5	6	7	8	9	10	11	12	13	14
The	or	i	gin	of	sci	ence	is	in	the	de	sire	to	know

15	16	17	18	19	20	21	22	23	24	25	26	27
cau	ses,	and	the	or	in	gin	of	all	false	sci	ence	and

28	29	30	31	32	33	34	35	36	37	38	39	40	41
im	pos	ture	is	in	the	de	sire	to	ac	cept	false	cau	ses

42	43	44	45	46	47	48	49	50	51	52	53
ra	ther	than	none.	Or	which	is	the	same	thing	in	the

54	55	56	57	58	59	60	61	62	63	64	65	66
un	will	ing	ness	to	ack	now	ledge	our	own	ig	nor	ance.

syllable number	level of confidence	description of the block

Note: Additional blocks or descriptive notes may be indicated on the back of this form.

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