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The Effect of Surface EMG Biofeedback on Muscular Tension and Stuttering Behavior

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THE EFFECTS OF EMG BIOFEEDBACK TRAINING ON MUSCULAR TENSION AND STUTTERING BEHAVIOR

By

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ABSTRACT

THE EFFECTS OF EMG BIOFEEDBACK
TRAINING ON MUSCULAR TENSION
AND STUTTERING BEHAVIOR

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This study uses a single subject research design to show the effects of biofeedback training on muscular tension and stuttering behavior. Such data can assist in developing a more efficient biofeedback therapy program for the treatment of stuttering. Four stutterers, were assigned randomly to two groups: S-F, treatment of specific site followed by frontalis training; or F-S, treatment of frontalis training first followed by specific site muscular training. A 12-session therapy program (six 20-minute sessions per site) was implemented to train the subjects to control muscular tension using visual and/or auditory feedback.

The data collected consisted of EMG readings for non-speech and speech tasks and the dysfluency percentage during pre-therapy, post-therapy, pretest and posttest within sessions for each muscle site tested. The results from this investigation indicated that a reduction of excessive muscle tension in the speech musculature is a basic factor in reducing stuttering behavior.

ACKNOWLEDGEMENTS

I wish to express my appreciation to Dr. Paul Cooke, my thesis director, and Dr. Richard Roppel and Dr. Leo Deal, the members of my thesis committee for their invaluable assistance and criticism in the preparation of this thesis.

I also wish to thank Dr. Michael Chial and Mr. George Gamble for their expertise in setting up the instrumentation for the experiment.

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INTRODUCTION: REVIEW OF LITERATURE

Numerous references in the research literature on stuttering are directed toward the hypothesis that stuttering is the result of excessive physiological tension. Many researchers are of the opinion that the stuttering block is accompanied by a spasm of the laryngeal muscle (Van Riper, 1971; Schwartz, 1975). Based on this hypothesis, Hanna, Wilfling, and McNeill (1975), in a single subject biofeedback treatment for stuttering, showed that laryngeal tension and stuttering were intimately related. Shrum (1967) measured surface electrical activity of facial, neck and chest muscles in stutterers. He observed a relationship between increases in EMG signal amplitude prior to speech and the subsequent stuttering. Bloodstein (1975) claims that for most stutterers the act of blocking is associated with some feeling of strain or tension. This is usually localized in the speech musculature, i.e., the muscles of articulation, phonation, or respiration, but in some cases this tension may also be manifest, elsewhere in the body, for example, the arms, legs, or shoulders. Stuttering therapy has for a long time focused on reducing this physiological tension by using relaxation techniques. In more recent times, biofeedback has been introduced as an effective means of reducing some of that physiological tension. Biofeedback is a method of learning to control body processes that ordinarily cannot be regulated volitionally. The concept of biofeedback is based upon three basic principles. First, an individual is able to assume control over a neurological or biological function if that function can be monitored or

amplified by electronic instruments and fed back to him. Second, every change in the physiological state is accompanied by an appropriate change in the mental emotional state and vice versa. The third principle is that a meditative state of deep relaxation is conducive to voluntary control of body processes because it allows the individual to become aware of internal imagery and sensations.

The amount of literature pertaining specifically to the use of electromyographic (EMG) biofeedback in the field of speech, particularly stuttering, is very limited. For this reason, articles dealing with other clinical problems related to tension reduction procedures using EMG biofeedback will also be reviewed. Studies have shown that EMG biofeedback may be used successfully for the treatment of several disorders including high blood pressure, digestive disturbances, irregular heart beat, migraine and tension headaches, and chronic anxiety.

Canter, Kondo and Knott (1975) did a comparative study using anxiety neurosis as the dependent variable and two different methods of training in deep muscle relaxation. These included biofeedback procedures and progressive muscle relaxation techniques. They concluded from their experiments that EMG feedback was generally superior in producing larger reductions in muscle activity, with concomitant relief in anxiety symptoms. Acosta et al.(1978) applied EMG biofeedback to the relaxation training of schizophrenic, neurotic and tension headache patients. Results from their study indicated that patients did exhibit a reduction of their frontalis muscle tension level across the ten successive biofeedback training sessions. They further noted that learning to reduce tension through EMG biofeedback was applicable to individuals with diverse backgrounds. In addition, Raskin et al. (1973) and Townsend and House (1975) also found

EMG biofeedback effective with the treatment of chronic anxiety patients.

The frontalis muscle has been frequently used as a popular site for electrode placement in various experiments. Canter et al. (1975) explained that the frontalis muscle was chosen as the target for feedback training in their experiment "since this muscle has been shown to reflect the general muscle tension levels in anxious patients". Alexander et al. (1975) compared the efficacy of auditory and visual feedback in EMG biofeedback-assisted relaxation training of the frontalis muscle. The authors found that biofeedback training with auditory feedback produced significant decreases in frontalis EMG, whereas training with visual feedback was ineffective. Haynes, Miseley and McGowan (1975) assessed the comparative effectiveness of frontalis EMG biofeedback and relaxation instructions in reducing frontalis EMG levels. Results from the one-session design demonstrated that the greatest decrement in frontalis EMG level was attained when subjects received biofeedback as opposed to other techniques. The authors indicated that previous research suggest that frontalis activity may be correlated with muscle tension levels in other areas of the body.

Many reserachers have shown that reduction of tension of specific speech muscle groups using biofeedback techniques is accompanied by decreases in stuttering frequency. The muscle groups chosen for electrode placement were specific for each individual case and included the orbicularis oris superior (lip), the anterior belly of the digastric (chin), laryngeal muscles, the frontalis muscle (forehead), masseter muscle (jaw), and other muscles of the face, head, and neck.

Guitar (1975) trained three adult male stutterers to reduce resting muscle activity by using analog EMG feedback from four sites over different

muscle groups. He then trained his subjects to reduce muscle activity prior to uttering selected sentences. A functional analysis of the relationship between the decrease in stuttering frequency on the initial phonemes and the reduction of electrical activity at each muscle site indicated that the subjects had different responses to the training. One subject's greatest decrease in stuttering frequency was associated with muscle activity training at the lip site; the second subject's greatest decrease was associated with training at a laryngeal site; and the third subject's decreases were related to both lip and laryngeal site training. Based on these results, the author designed a practical management program for a fourth stutterer. Feedback training to reduce EMG activity when paired with speech resulted in a reduction of stuttering in two monitored situations, conversations and telephone calls. Probes indicated that stuttering continued to be markedly reduced in all situations nine months after treatment.

Alexander (1975) designed an EMG feedback program to allow stuttering subjects the opportunity to perceive normally subliminal physiological events. The program trained subjects to utilize feedback of muscle potentials to control the amount and occurrence of tension in specific muscles. The muscles chosen were those judged to be most tense during the stuttering moment. Subsequently, the program was designed to teach the subjects to apply the learned muscle control to speech tasks to reduce tension prior to, during and immediately following the speech event. Finally, the program was designed to facilitate transfer of the newly acquired fluent status into the subject's nonclinical environment and to maintain fluency in that environment. Results indicated a reduction of the individual nonfluencies and a reduction of secondary characteristics not normally

related to speech.

Lanyon, Barrington and Newman (1976) conducted a preliminary study in which eight stutterers spent 10 to 18 one-hour sessions learning to relax their masseter muscles using EMG visual feedback. Major reductions in stuttering were demonstrated for all subjects in reading one-syllable words while feedback was present as compared to no feedback. Similar results were obtained with six subjects who progressed through the reading of two-syllable words, three-syllable phrases, and four-syllable sentences. Lanyon (1977) developed further methodology for the use of relaxation in the modification of stuttering, based on the general hypothesis that excessive tension in speech-relevant muscles was a key component in stuttering. He showed through replication and extension of the previous study, that the voluntary relaxation of the speech muscles could be learned with the aid of EMG visual feedback. Results indicated that subjects could successfully be taught to rely on internal cues to generalize fluent speech and relaxation behaviors to periods when feedback was absent.

The reports cited above depict a very limited number of cases in which reduction of tension of speech muscle sites using EMG biofeedback techniques has been accompanied by decreases in stuttering frequency. More research is needed to determine the variables that must be controlled if favorable clinical results are to be achieved.

Research Questions

1. What effect does EMG biofeedback training have on the tension level of the frontalis muscle of stutterers?
2. What effect does EMG biofeedback training have on the tension level of a specific muscle site of stutterers?

3. Is there a transfer effect of EMG frontalis biofeedback training from sessions with feedback to sessions without feedback?

4. Is there a transfer effect of EMG biofeedback specific site training from sessions with feedback to sessions without feedback?

5. What effect does EMG relaxation training of the frontalis muscle have on stuttering behavior?

6. What effect does EMG relaxation training of the specific site have on stuttering behavior?

Chapter II

METHOD

Subjects

Four male stutterers between the ages of 18 and 35, who had previously enrolled for stuttering therapy were selected following diagnostic evaluations conducted by the experimenter. Only subjects who exhibited high observable facial and/or laryngeal tension during the initial interview could volunteer to participate in the experiment. The subjects ranged in severity from mild to severe, based upon observations made by the experimenter during the diagnostic and therapy sessions. The subjects were assigned randomly to one of two groups: S-F, treatment consisting of specific site training followed by frontalis training; or F-S, treatment consisting of the frontalis training first followed by specific site muscular training.

Selection of Muscles

Two muscles were selected for biofeedback training during this experiment. Figures 1 and 2 provide schematic drawings of the muscles of the face, neck and larynx that were used during the diagnostic interview. These included the medial frontalis, temporalis, zygomatic, levator, orbicularis oris, masseter, mentalis, buccinator, geniohyoid and the cricothyroid. One of these muscles was chosen based upon the tension level that the experimenter observed during stuttered speech. This is referred to as the specific site placement and was selected on an individual basis.

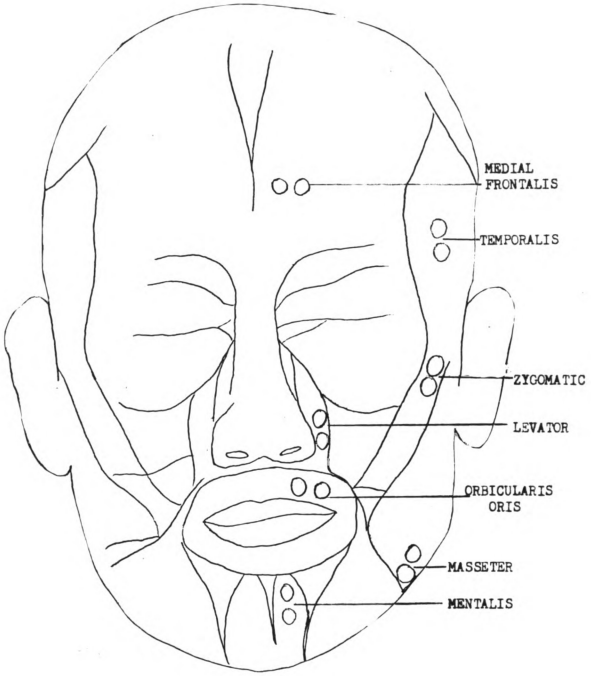

Figure 1. Schematic drawing illustrating muscles of the face observed during the diagnostic session.

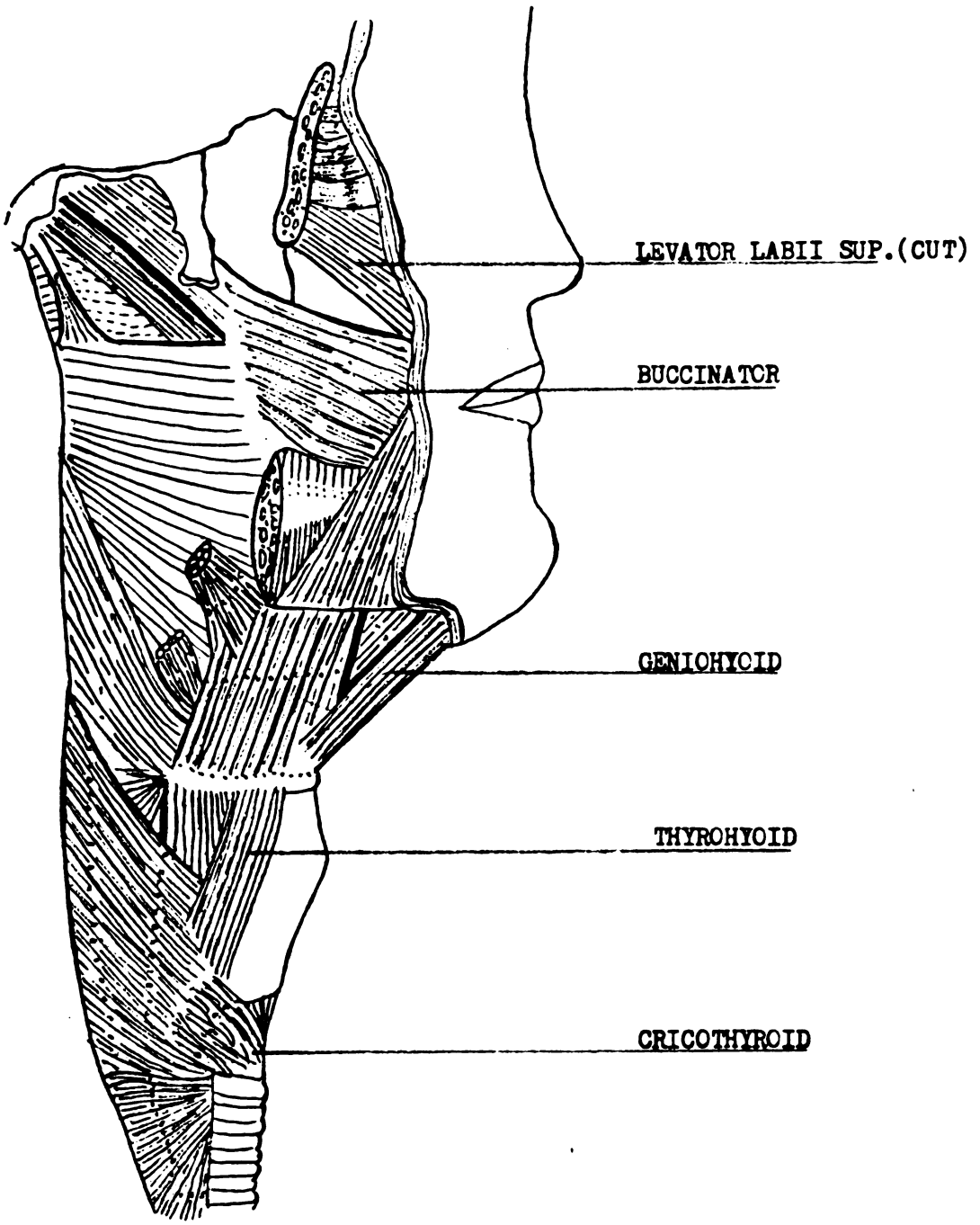

Figure 2. Schematic drawing illustrating muscles of the neck and larynx observed during the diagnostic session.

The second muscle, which was trained with all subjects, was the frontalis. The frontalis muscle was chosen as the non-specific site since research has suggested that tension reduction of this muscle is accompanied by general body relaxation (Canter et al. , 1975).

Electrode Placement

The subjects were seated in a comfortable chair with arm rests in a sound treated chamber. Bipolar EMG activity from each muscle region was recorded with a pair of miniature skin surface electrode placed in close proximity to each other. A neutral electrode was placed on the nearest bone to serve as a ground. The skin was prepared by first rubbing the area with a disposable skin cleanser swab formulated to remove insulating elements from the skin surface to minimize skin resistivity. The skin was allowed to dry while the electrodes were carefully filled with Beckman electrode paste and attached with adhesive collars over the muscle to be tested.

Instrumentation

Figure 3 presents a block diagram of the instrumentation used in sensing the raw EMG signal, amplifying and transforming the signal for visual and auditory feedback purposes, and converting the signal to a numerical equivalent for analytical purposes. The muscle being tested produced an electrical signal whose amplitude is proportional at each instant to its tension. This signal was picked up by EMG electrodes, preamplified (Hewlett Packard 1510B), and then transferred to an operational amplifier, which increased the signal level by a factor of 10. The amplified signal was then rectified and integrated using a contour

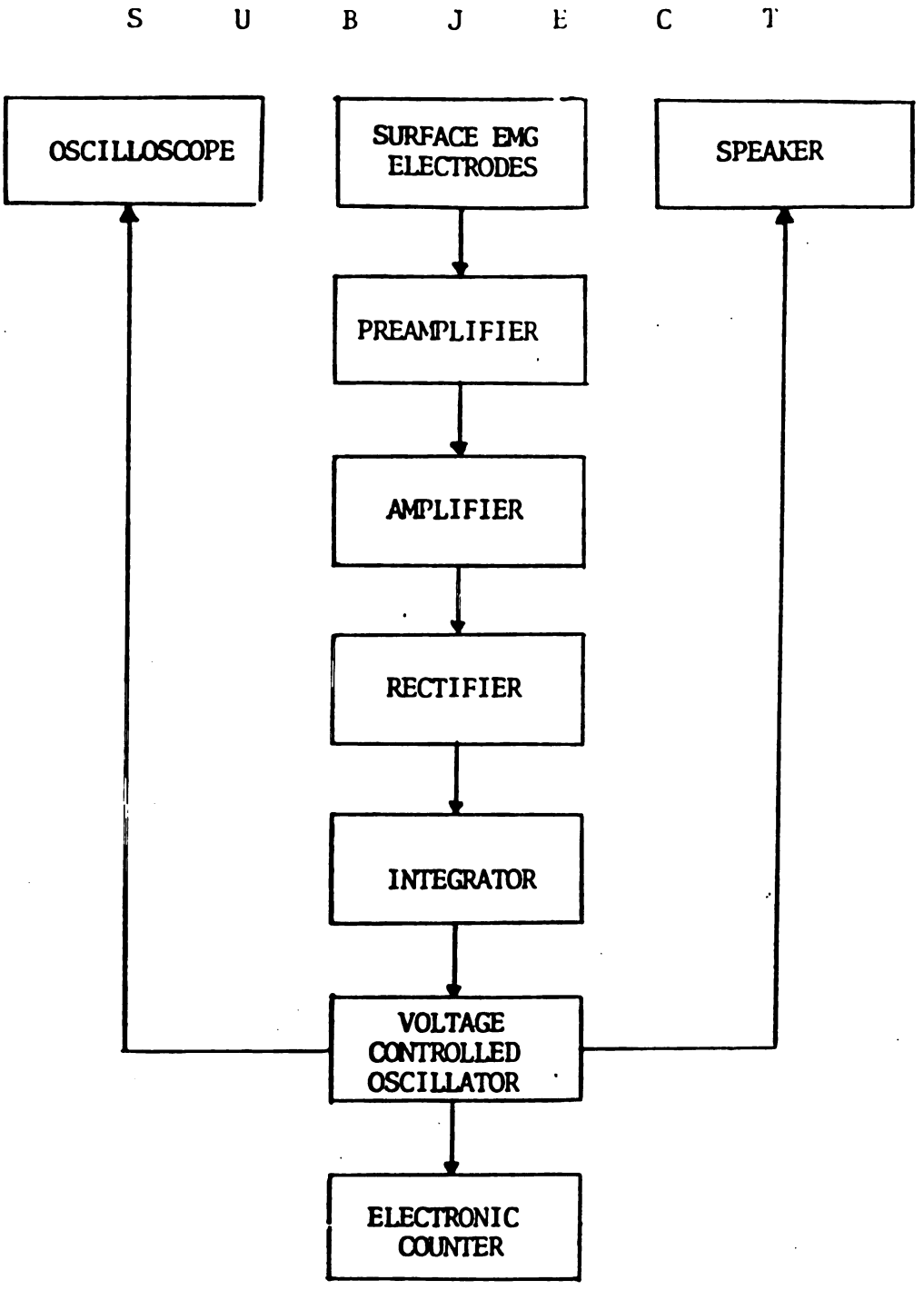

Figure 3. Block diagram of the instrumentation used during this experiment.

following integrator (Coulbourn S76-01). Rectification is the process by which the amplified altnerating current (AC) is converted to an unidirectional current (DC). The integration of the signal produces a relatively smooth curve whose amplitude at any point is proportional to the peak amplitude of the pulsating DC input. This smoothing function can be controlled by choice of an integrater time constant, which for this experiment was set at 500 msec. A voltage controlled oscillator (Coulbourn S24-05) accepts the smoothed DC signal and converts it to a frequency-modulated sinusoidal wave and a synchronous frequency-modulated square wave. The former was visually displayed by means of an oscilloscope and simultaneously transmitted through a loudspeaker as a pure tone by means of an audiomixer amplifier (Coulbourn S82-24). Both of these displays (auditory and visual) were used by the subject as biofeedback signals of his muscular activity. As the muscle became more relaxed (i.e., a decrease in electrical activity), the pure tone became lower in pitch and the oscilloscopic display indicated a smaller number of sinusoidal waves per unit of time. The square wave was converted to a numerical value by a digital counter (Coulbourn R11-01) which produced an output equal to the number of pulses counted within a given period. These values were used, in conjunction with a calibration factor derived by a method described in the "data acquisition" section, to determine the average microvolts (μv) emitted by the muscle over a period of time. The sampling interval used in this experiment was 60 seconds and was manually initiated by a switch module (Coulbourn S96-03).

Procedure

The four stutterers were assigned randomly to one of two groups: S-F, treatment consisting of specific site training followed by frontalis training; or F-S, treatment consisting of the frontalis training first followed by specific site muscular training. The experiment consisted of a pretest, six training sessions and a posttest for each of the two muscle sites investigated.

The pretest and posttest involved measuring muscular activity at the particular site during nonspeech and speech tasks without biofeedback training. Each pretest or posttest session was completed within a ten-minute interval. The nonspeech and baseline segment consisted of three consecutive one-minute intervals. The subjects were instructed to keep their heads still and to avoid unnecessary movements in order to minimize experimental artefacts. The speech segment composed of the subject speaking for three one-minute segments. Open-ended general topic questions were asked by the experimenter to stimulate the speech activity.

The six training sessions for each of the muscular sites was approximately 45 minutes in length. The first and last ten minutes of each session involved a pretest and posttest segment as described above. The middle twenty minutes of the training session focused on EMG biofeedback therapy. During the training sessions the subject was instructed on the association between the variations in the auditory and visual signals and muscular tension. The subject's task was to try to maintain a low frequency tone from the loudspeaker and/or elongate the sinusoidal wave on the oscilloscope. During the therapy sessions, each subject controlled the type of biofeedback received, (i.e., the subject had the option to look away from the visual display or to vary the amplitude of the

auditory signal). Throughout the therapy session the experimenter used open-ended questions to elicit the maximum amount of continuous speech. Four one-minute samples of EMG readings while speaking were taken at intervals of approximately five minutes.

Data Acquisition

The EMG microvolt readings were computed using the following calibration procedure. Known signals of 0, 20, 40, and 60 microvolts were introduced into the input of the system to produce an equivalent electronic counter reading for a 60 second period of time. The counts collected were divided by 60 to obtain the counts-per-second average and these values were plotted as a function of the input signal. A straight line approximation ($y = mx + b$) was applied to these data. The readings recorded throughout the experiment were substituted into the equation to convert electronic counter readings into amplitude values of the EMG signals.

The percentage of dysfluency presented for each subject was calculated using Wingate's (1964) definition of stuttering as a guide to determine the occurrence of a stuttering block. The term stuttering focused on the disruption in the fluency of verbal expression by repetitions and prolongations. Samples of speech collected during each pretest and posttest session were transcribed, and only the first two minutes of continuous speech for each session was used in the analysis. The percentage of dysfluency was obtained by dividing the number of dysfluencies counted by the total number of words uttered during the two-minute sample of speech and multiplying the result by 100.

Chapter III

RESULTS

The data collected on each subject during this experiment are described individually. This information is displayed in two figures, one for each muscle site investigated. Each figure presents the percentage of dysfluency and EMG data for a pretesting and a posttesting period across the six therapy sessions. Two different y-axis scales were required to represent the data in this fashion. The left hand scale is for the EMG values measured in microvolts, which are symbolized by an "x" for speech tasks and by an "o" for non-speech (resting) conditions. The right hand scale represents the percentage of dysfluency and are symbolized by a "Δ". Each pretest and posttest session incorporated EMG recordings during non-speech and speech tasks without the presense of biofeedback. Lines are drawn between the data obtained during pre- and post- testing conditions indicating that biofeedback training had occurred during this interval. In addition, pre-therapy and post-therapy readings were obtained. A note of caution must be interjected regarding the interpretation of these figures. Muscle regions differ with respect to the absolute magnitude of activity that can be presented by the underlying muscles. Thus, the total range of activity recorded from the surface electrodes will vary from region to region as well as from individual to individual.

Subject F-S1

Figure 4 illustrates the data from subject F-S1 when the frontalis was the training muscular site. From pre-therapy to post-therapy testing his EMG activity while resting reduced from 12 μv to 6 μv. In addition, this subject consistently reduced his EMG activity for speech during the therapy session, with the exception of session 2 where the frontalis activity increased slightly from 36 to 38 μv. However, any carryover from the biofeedback training for speech purposes was not apparent, since the pre-therapy value of 15 μv. increased by 12 to 27 μv. during the post-therapy measurement. Thus, the reduction of dysfluency from 28% at pre-therapy to 7% during post-therapy testing cannot be accounted for by the reduction in frontalis activity. This is supported by the .37 correlation that existed between EMG activity and the dysfluency percentage.

Figure 5 displays data from F-S1 when the orbicularis oris muscle was being trained with biofeedback. Comparing pre- and post- therapy conditions, the resting muscular activity demonstrated a slight increase from 13 to 16 μv., while the activity during speech reduced 6%, from 90 to 85 μv.

This relatively modest change is indicative of the inconsistent pre- and posttest values obtained at each therapy session. The subject demonstrated slight reductions in EMG activity during sessions 2 and 4 and a sharp decrease in orbicularis oris activity (112 to 79 μv) during session 3. However, modest increases in the electrical activity were reported during the other three training sessions. The correlation between the muscular activity and the dysfluency percentage was .21 across the six therapy sessions. Such a correlation was a result of inconsistent associations from session to session, as the values for the first three

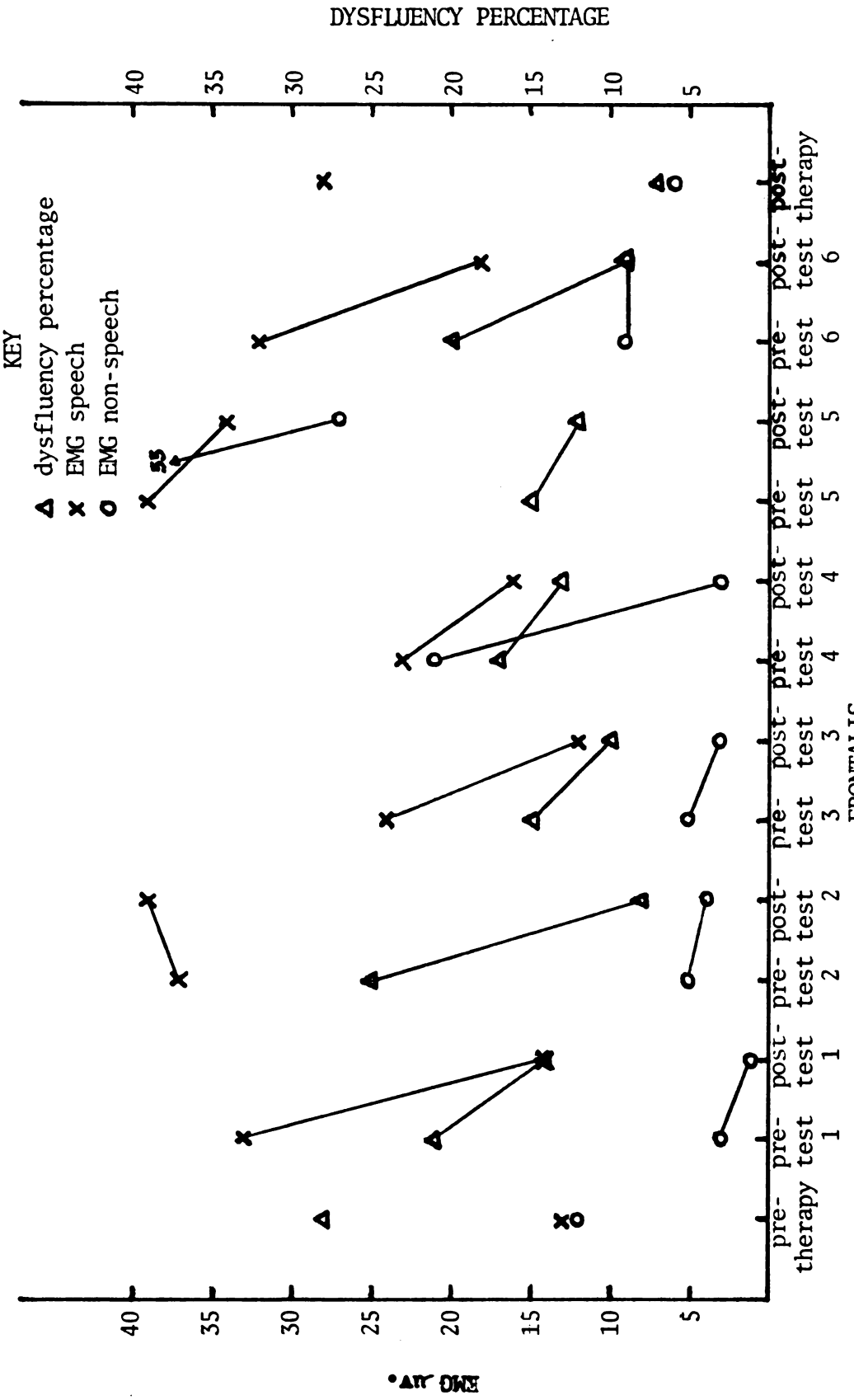

Figure 4. Subject F-S1's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for frontalis muscle training without biofeedback. The lines represent the biofeedback training interval.

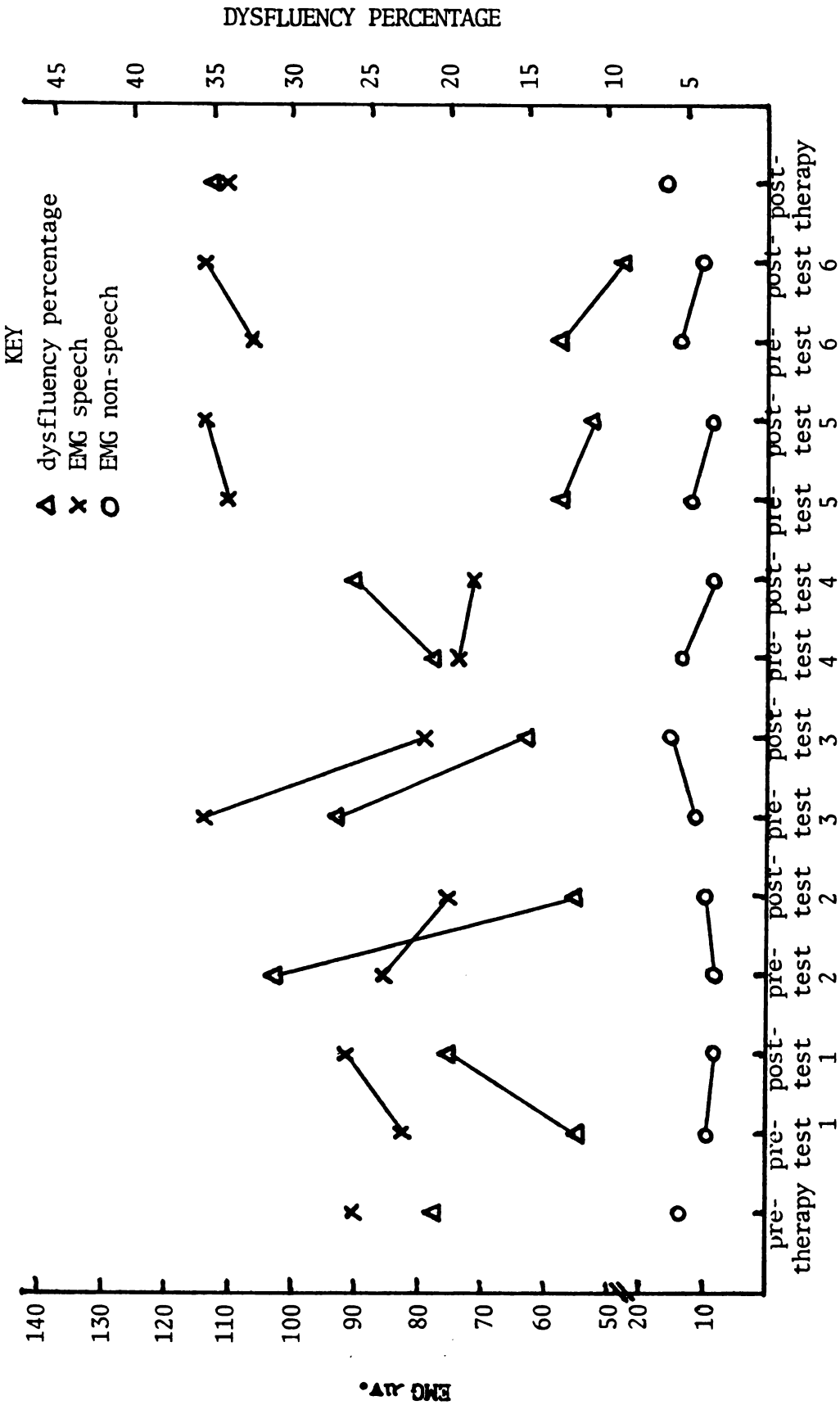

Figure 5. Subject F-S1's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for orbicularis oris muscle training without biofeedback. The lines represent the biofeedback training interval.

therapy intervals demonstrated a direct association between the two variables, whereas the values for the last three sessions were inversely related.

Subject F-S2

Figure 6 represents data of subject F-S2 when the frontalis was being trained. For five of the six therapy sessions the subject demonstrated a consistent pattern. In those sessions the EMG baseline data was always higher than the EMG speech data during pretest but were reversed during the posttest evaluation. Session 5 was the exception with the EMG baseline being lower than the EMG activity during both pretest and posttest conditions. Thus, while the biofeedback training did result in lower frontalis activity for the baserate condition by the end of the session, it increased the EMG activity during speech. In fact, the electrical activity during post-therapy for speech, after six therapy sessions had been completed, was 5 μv higher than before biofeedback training was initiated (8 μv. compared to 3 μv.). A moderate correlation of .52 occurred between the frontalis activity for speech and the percentage of dysfluencies. The amount of dysfluency decreased across the six biofeedback sessions from 17% to 12%.

Figure 7 displays data from subject F-S2 during orbicularis oris training. The non-speech data were consistent throughout the therapy sessions, with a slight increase in muscular activity at the termination of therapy when compared to the initiation of the session. The largest increment of this nature occurred during session 4 as the pretest EMG non-speech recording was 4 μv. compared to 14 μv. during the posttest interval. The most notable change that occurred throughout the orbicularis

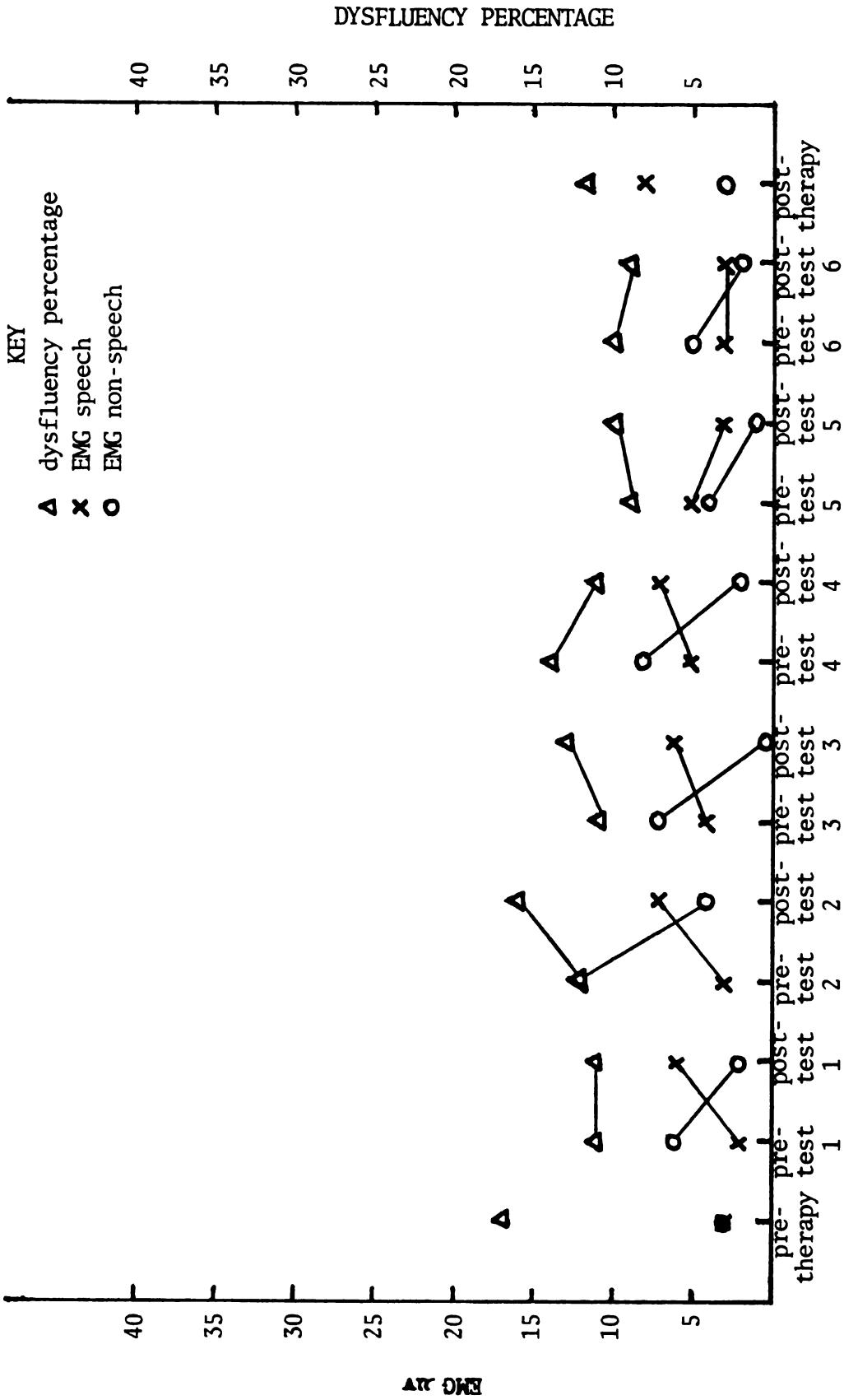

Figure 6. Subject F-S2's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for frontalis muscle training without biofeedback. The lines represent the biofeedback training interval.

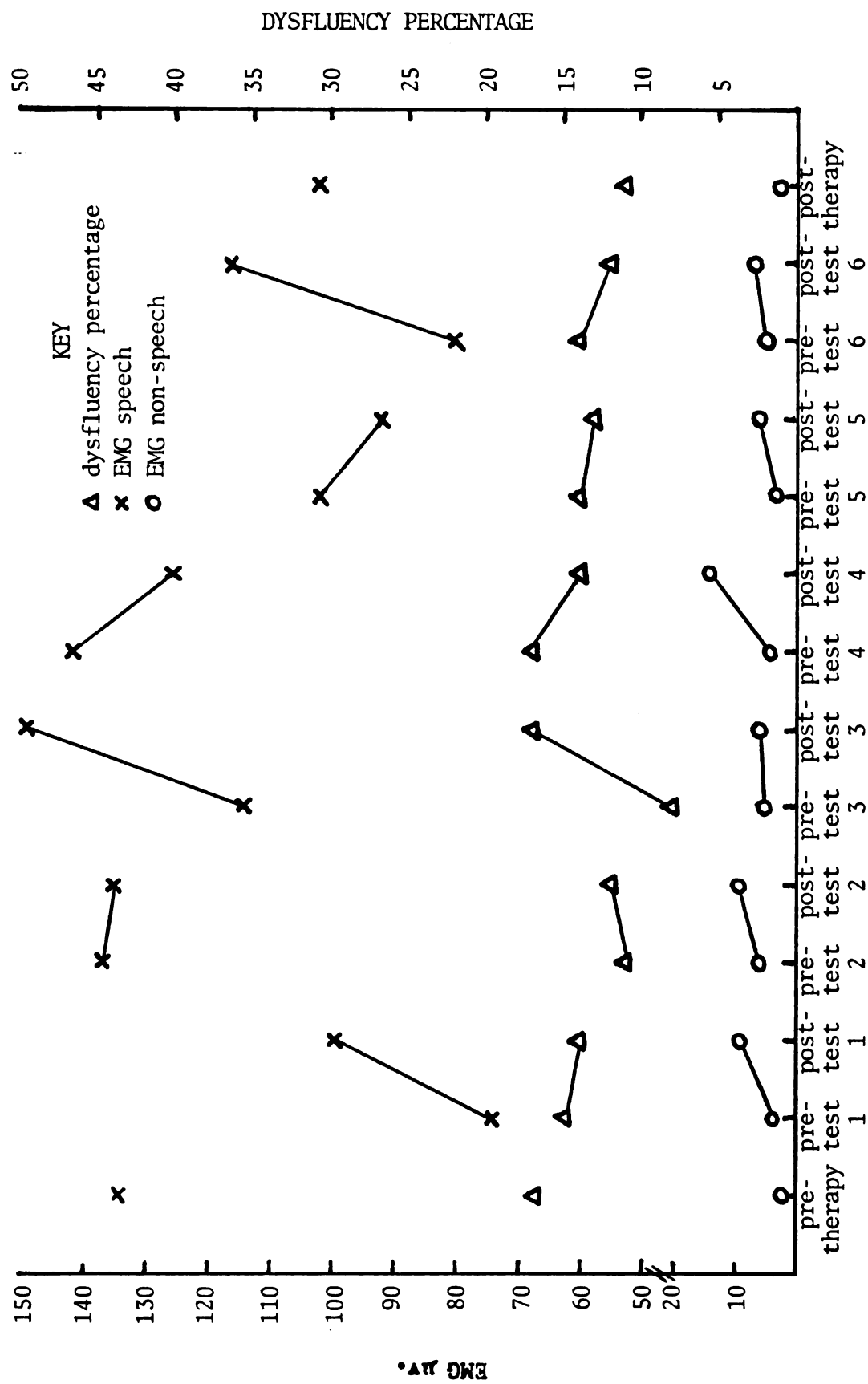

Figure 7. Subject F-S2's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for orbicularlis oris muscle training without biofeedback. The lines represent the biofeedback training interval.

oris training was the sharp reduction of the EMG activity for speech. Although the EMG activity during pre- and post- testing evaluations were highly variable, the pre-therapy session was 134 μv while the post-therapy reading was 102 μv, a reduction of 24%. This was consistent with the decrease in stuttering behavior, from 17% before therapy to 11% after the biofeedback sessions. Across the entire therapy program, a correlation of .45 existed between the orbicularis oris activity and the percentage of dysfluency.

Subject S-F1

Subject S-F1 was the only individual for whom a muscle other than the orbicularis oris was used for the specific site biofeedback training. The geniohyoid was the muscle selected and the data are displayed in Figure 8. For both, the tension levels were higher for the geniohyoid during posttesting compared to pretesting. For the baseline measures, this occurred in five of six sessions (the exception being session 3); and for speech activities an increase occurred in four therapy sessions. The effect of this during the post-therapy session was consistent. The EMG activity for baseline had increased from 7 μv before the onset of therapy to a level of 20 μv after biofeedback training had terminated. The electrical activity during speech, however, was markedly lower, starting at 112 μv and dropping down to 70 μv at posttesting evaluation. The stuttering behavior similarly decreased in frequency as demonstrated by the 38% to 36% reduction from pre- to post-therapy testing.

Figure 9 represents data from S-F1 during frontalis biofeedback training. While the therapy consistently reduced the EMG activity by 2 μv during baseline or resting conditions, the muscular levels were higher at

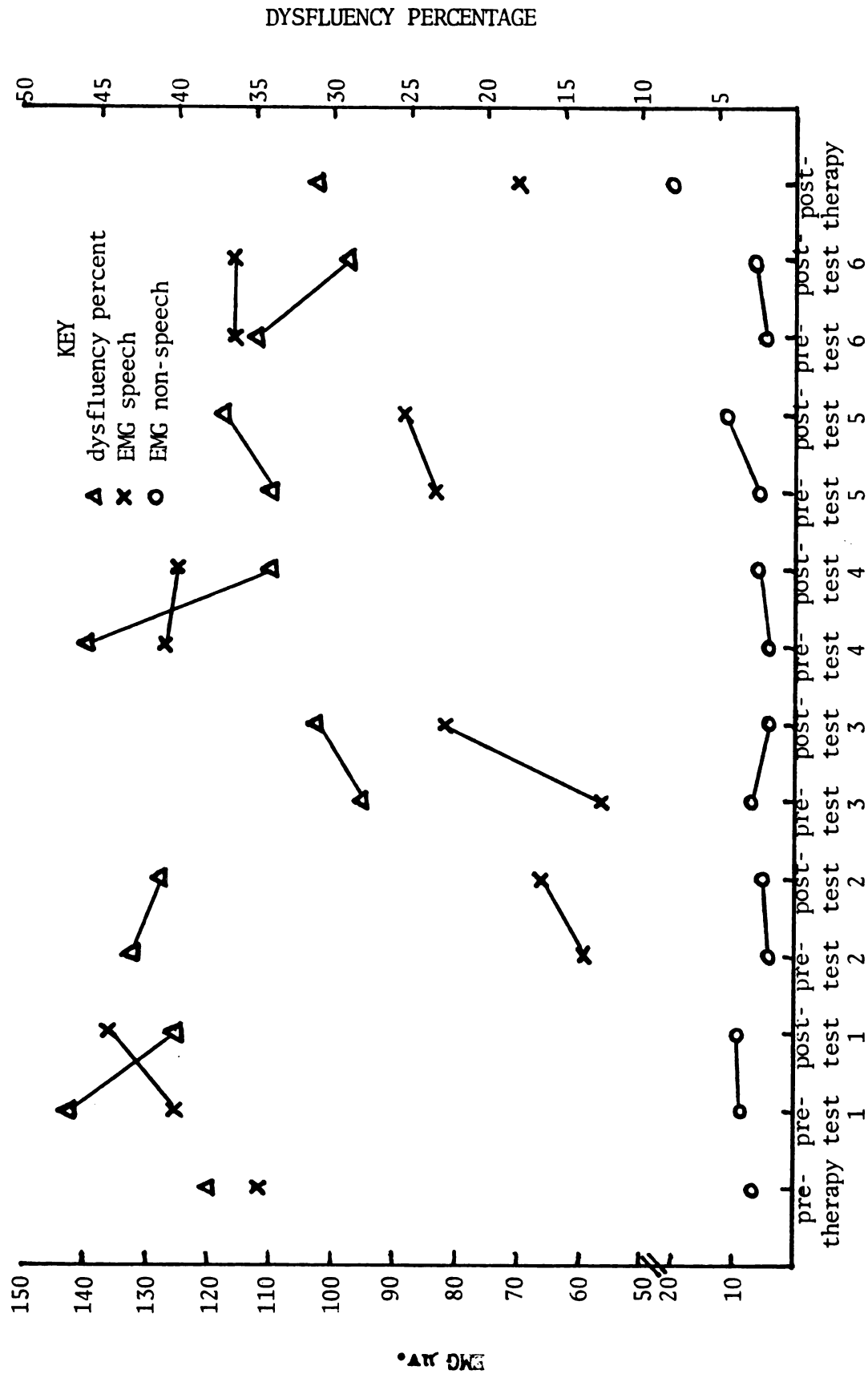

Figure 8. Subject S-F1's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for geniohyoid muscle training without biofeedback. The lines represent the biofeedback training interval.

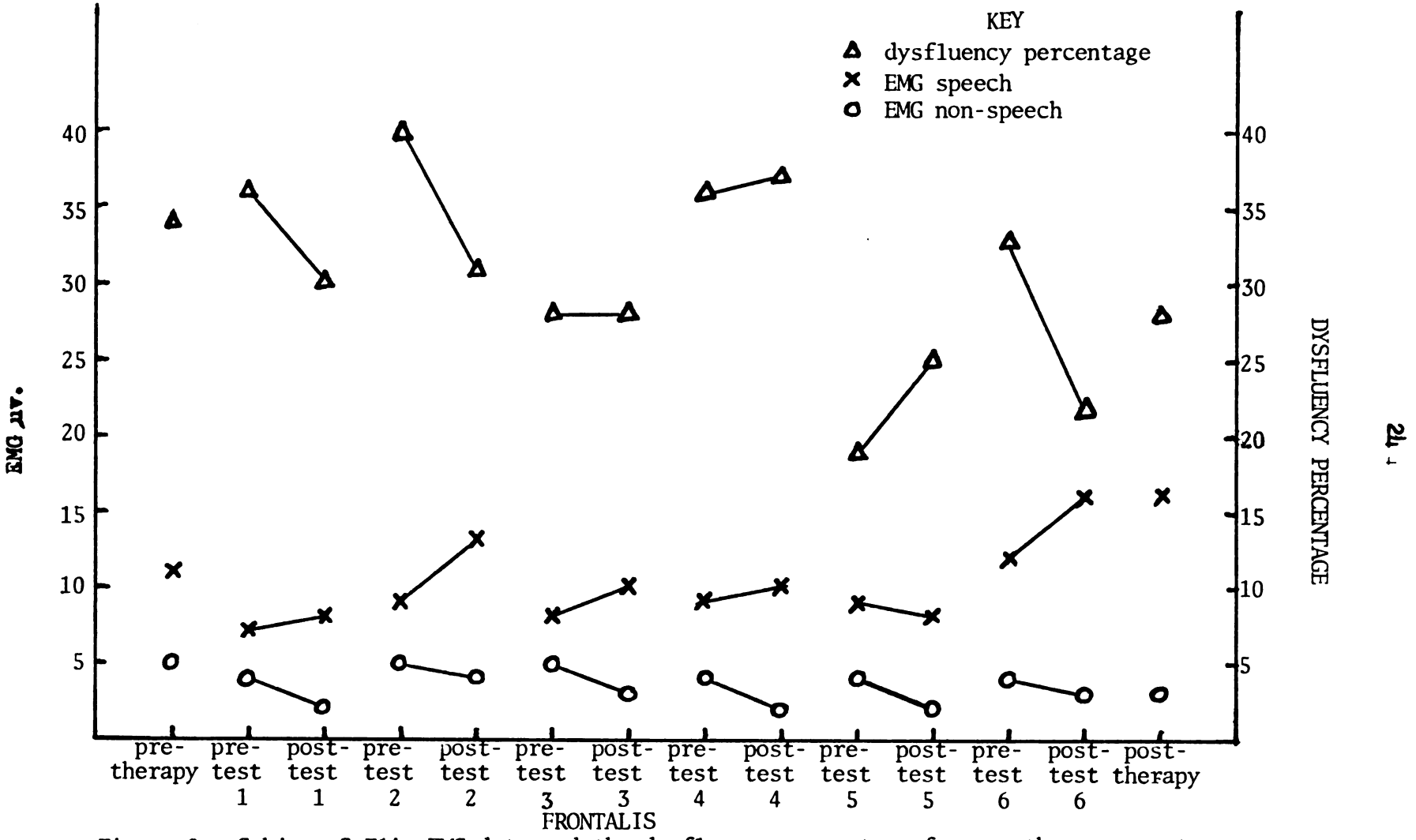

Figure 9. Subject S-F1's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for frontalis muscle training without biofeedback. The lines represent the biofeedback training interval.

the end of a therapy session compared to the beginning. This increase of muscular activity was reflected in the pretherapy and post-therapy speech data. Before biofeedback training was initiated, the frontalis activity was 11 μv. compared to the level of 16 μv after the six therapy sessions. However, the percentage of dysfluency behavior decreased over this same period, from 33% to 28%. Therefore, it is not unexpected that the correlation between EMG speech levels and the percentage of dysfluency was .27.

Subject S-F2

Figure 10 displays data of subject S-F2 during orbicularis oris biofeedback training. The figure displays that the therapy sessions resulted in drastic reductions of muscular activity while speaking. Decreases of 40 μv within a particular session was commonplace. In addition, comparing the EMG activity for speech at all the pretest conditions illustrates a systematic decrease of the muscular activity across time. This is reflected in the comparison of the pre-therapy to post-therapy values which indicates a decrease during speech from 143 μv to 102 μv. The other two measures, EMG activity during baseline recordings and the dysfluency percentages were relatively stable throughout the course of the biofeedback training.

The data from S-F2 during frontalis muscle training is focused in Figure 11. His EMG levels for speech and non-speech were consistently the lowest of all the subjects. Slight decreases in the three measured variables were observed across the sessions. The baseline muscular activity was reduced after every biofeedback session compared to the pretest condition. Such a training effect is evident by the decrease in the baseline EMG

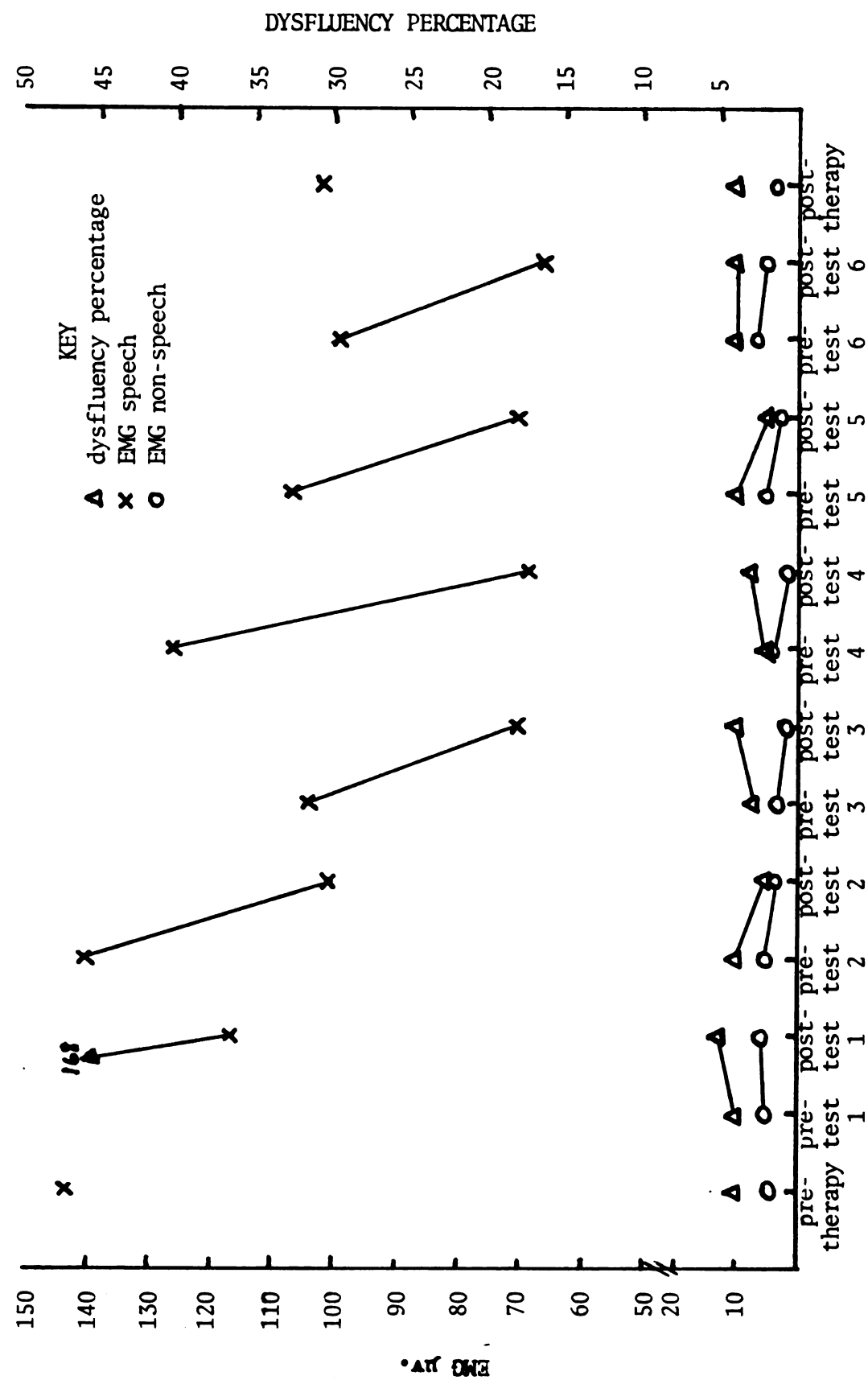

Figure 10. Subject S-F2's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for orbicularis oris muscle training without biofeedback. The lines represent the biofeedback training interval.

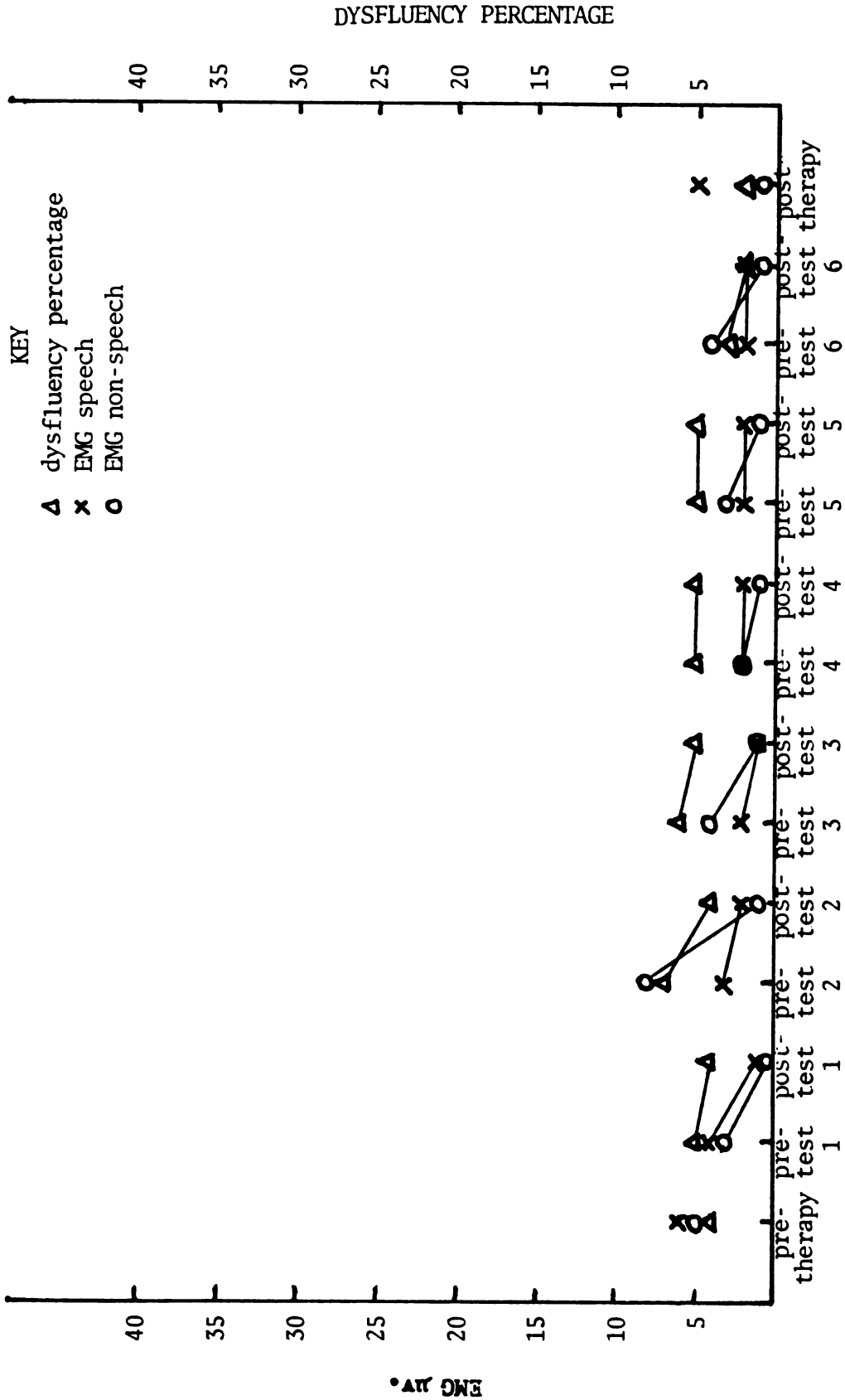

Figure 11. Subject S-F2's EMG data and the dysfluency percentage for pre-therapy, post-therapy, pretest and posttest within sessions for frontalis muscle training without biofeedback. The lines represent the biofeedback training interval.

reduction from 5 μv to 1 μv at pre-therapy and post-therapy intervals. The EMG for speech was reduced to 3 μv by session 4 and remained there throughout the remaining therapy sessions.

Chapter IV

DISCUSSION

Excessive physiological tension is associated with many maladaptive behaviors. As a result, researchers and clinicians have employed various techniques to reduce or control some of this muscular tension, including hypnosis, meditation and relaxation (Barber, 1963; Null et al., 1974; Jacobson, 1938; Wolpe, 1969). Stuttering is a complex speech disorder that frequently results from or causes excessive physiological tension. The most widely accepted therapeutic program which focuses upon reducing physiological tension in stutterers is systematic desensitization using relaxation as the reciprocal inhibition to anxiety (Adams, 1972; Burgraff, 1974; Gray and England, 1972). These techniques are aimed at reducing general body tension. Although stutterers may have higher overall tension levels than non-stutterers during speech activities, each stutterer has a specific pattern of excessive physiological tension. That is, particular muscle groups may exhibit more obvious abnormalities than other muscle sites. Recently, muscular biofeedback has been introduced to reduce tension from certain areas in the body. It has been found to be an effective and efficient means of procuring relaxation (Haynes, et al., 1975; Reinking et al., 1975; Coursey, 1975). These biofeedback procedures have been used occasionally in attempts to reduce stutterers' muscular tension (Guitar, 1975; Alexander, 1975; Lanyon, 1976). Based upon the limited research in this potentially useful therapy for stutterers, this experiment

was designed to explore the effectiveness of EMG biofeedback training to reduce muscular activity and stuttering behavior.

Generally, the results from this investigation are consistent with previous research in that the reduction of excessive muscle tension in the speech musculature was a basic factor in reducing stuttering behavior (Guitar, 1975; Lanyon, 1977). All four subjects in the present study demonstrated a reduction in electrical activity and stuttering behavior after biofeedback training in one of the two muscle sites. For subjects F-S2, S-F1, and S-F2 the therapy sessions involving specific muscular sites produced the most positive effects, whereas the best results for subject F-S1 occurred during the frontalis sessions. As in all of the previous EMG biofeedback studies, the therapeutic gains varied dramatically. This is demonstrated by the pre- and post- biofeedback dysfluency percentages from the muscle site that was most beneficial F-S1 (28% to 7%), F-S2 (17% to 11%) S-F1 (38% to 36%), S-F2 (6% to 4%). It should be noted that F-S1, who demonstrated the most drastic dysfluency reduction across the biofeedback sessions, was the only subject of the four for whom the frontalis training was beneficial. Examining the data reveals that F-S1 was the only subject who demonstrated frontalis EMG readings between the 20-40 μv. range during speech activities. The other three subjects had fluctuating frontalis EMG recordings from 5-15 μv. Thus, while the frontalis biofeedback training sessions were not beneficial for those illustrating low levels from this site, for subject F-S1 the frontalis muscle was the site that had the greatest potential for tension reduction.

The dysfluency reductions during the most beneficial biofeedback site are slight (except for F-S1) compared to other therapeutic programs. There are several factors that may account for this. Cumulative effects of

biofeedback training were noted from the subjects. That is, there was little, if any, carryover of reduced muscular activity when the experiment changed from one muscle site to the next. Thus, any therapeutic gains that occurred during muscular training from one site were not beneficial during the training of the second muscle site. These results are consistent with other EMG biofeedback studies indicating that generalization from such therapeutic procedures is difficult (York, 1977; Alexander et al, 1977; Legewie et al. 1975). Thus, the present behavior changes must be evaluated in terms of single-site training (i.e., six 20-minute biofeedback session). In addition, spontaneous speech was the only speech behavior that was evaluated in this investigation. This represents the most complex speech activity and therefore is usually the most difficult for stutterers. Other therapeutic programs frequently report behavioral data indicating dramatic changes in speech utterances that are less complicated, such as words, short phrases, short sentences, and reading. Therefore, considering the high level of communicative skills that were under investigation and the limited amount of training (two hours), the reduction of dysfluency across the therapeutic setting was encouraging.

There were some data that were unexpected. It was assumed that the subject would exhibit a greater degree of relaxation (less electrical activity) during the baseline recordings than during conversational speech, where the muscle being monitored would play an active role. However, subjects F-S2 and S-F2 demonstrated the reverse pattern on several occasions. This is illustrated in Figure 6 during the pre-test conditions of sessions 1, 2, 3, 4, and 6 and in Figure 11 during the pre-test sessions 2, 3, 5, and 6. The most logical explanation concerns the order in which the baseline and speech activities were measured. During the pre-test

the baseline EMG level was recorded first and was followed by the measurement of the EMG activity during speech. It is very probable that because the baseline level was always measured first, any overall arousal level that the subject had entering the therapy session would get reflected in the first value obtained. Therefore, if the individual had been anxious during the day, the higher arousal level might have been apparent during the first few minutes of baseline recordings. By the time the EMG speech data were taken (about 10 minutes after the subject had entered the room), the individual may have become generally more relaxed, especially since that was the general goal of the therapy sessions. Therefore, on these few occasions from these two subjects, the speech EMG data were lower than the baseline measure that was obtained several minutes earlier. This explanation is the most plausible since this reversal of EMG values only occurred during pretesting and NEVER occurred after the biofeedback session was completed. That is, the post-testing data always illustrated that the subject had higher EMG levels for speech compared to non-speech.

A more perplexing phenomenon occurred concerning the EMG levels during speech activity. Three of the four subjects, to varying degrees, demonstrated greater muscular activity during conversational speech after biofeedback training compared to pre-therapy levels. That is, the data suggest that the subjects became more tense as a result of individual sessions and after the entire six session therapy program was completed. Subject S-F1's data during the frontalis feedback training illustrate this point in Figure 9. The post-therapy EMG level was 16 μv . compared to 11 μv. level before any training had commenced. In addition, during five of the six therapy sessions (the exception is session 5) the posttest

EMG activity was higher than the pretest level. Since this type of data has not been illustrated in other biofeedback studies and since the subjects reduced their dysfluency percentage across the therapy programs, factors within the experimental designs were reviewed for possible explanations.

The biofeedback signals were controlled by the subjects. They could receive visual feedback and auditory feedback simultaneously and increase the amplitude of the auditory signal. Subject S-F1, whose data most exemplified this reversal of EMG levels across biofeedback training, indeed increased the audio signal during the feedback sessions. This subject reported that he did not have control of the EMG activity unless this high amplitude was continuously used. It is feasible that some subjects were being overstimulated with biofeedback, a situation that may have hindered relaxation. This could have been achieved by the subjects to obtain feedback from both auditory and visual channels simultaneously and/or by intensifying the pure tone from the speaker. This could have raised the general tension level of the body so that by the end of the therapy session the actual muscular activity was greater than the level preceding therapy.

The data acquisition procedure used to determine the EMG levels could have contributed to higher posttesting values compared to pretesting measures. The data were obtained by averaging the levels from three 60-second samples during continuous speech. These intervals were initiated by the experimenter, and throughout the time period the EMG activity was being accumulated by a digital counter. The equipment automatically limited counting the muscular activity after 60 seconds. Therefore, any pauses that occurred during this 60 second interval were

included in the continuous speech segment. Some muscular activity for speech is greater than during non-speech activity. The 60-second segments that contained the least speech would be biased toward a lower EMG reading. Frequently the subjects had difficulty at the beginning of a therapy session engaging in continuous speech. However, by the end of the 20 minute biofeedback training session, they were more likely to have continuous discourse without any further questions from the experimenter. Thus, the pretest EMG data for speech was probably abnormally low in many instances, which suggested that the individual had actually increased his muscular activity across the biofeedback training.

Recommendations

Several points have been raised during this experiment that should serve as a caution to researchers and clinicians who might consider EMG biofeedback as a therapeutic technique. Indiscriminate application of these procedures is inefficient, and an extended diagnostic process should be considered to determine which candidates would benefit most from the program. Clients who demonstrate obvious muscle tension would be considered for biofeedback training. Several sessions might be necessary to determine a specific muscle site that would produce the most beneficial results for each individual. According to this study, three of the specific muscle sites seemed appropriate for such training, whereas the frontalis produced the most drastic effects from the other subject. In addition, the type of feedback should be examined individually, since some subjects benefit more from visual feedback, whereas others are better attuned to the auditory mode. Also, it is cautioned that the client should not have free control to modify the feedback provided since loud auditory stimuli may interfere

with the intent of relaxation training.

Ideally, an instrumentation configuration that could record two EMG sites while the subject is being trained by one would be beneficial. This would assist in determining whether muscular tension from one muscle was systematically having a controlling influence over the other muscles. If quantifiable EMG measurements are needed for comparison with speech output, then a synchronization between the two must occur. This can be accomplished by triggering the initiation and termination of muscular activity accumulation by the onset and offset of voicing.

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