FEEDBACK OF STUTTERERS' ELECTROMYOGRAPHIC ACTIVITY

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Abstract

Five studies were performed to explore the feasibility of treating severe chronic stuttering with EMG feedback. EMG spiking from the throat was found to correlate with stuttering, and to differentiate between stuttering and fluent speech. EMG spiking tended to disappear when stuttering was reduced by metronome-paced speech and by speech therapy. Likewise, when EMG spiking was reduced by feedback training, stuttering was concurrently reduced. Presentation of the feedback tone without instructions or information produced no reduction in stuttering or EMG spiking. Pseudofeedback was also generally ineffective. It was concluded that the feedback effect is apparently not an artifact of instructions, masking, distraction, adaptation, slowing of speech, or of a stutter-contingent aversive tone, nor is it a Hawthorne or placebo effect. Implications of the results are discussed.

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CHAPTER 1

INTRODUCTION

Stuttering

Stuttering is one of the most thoroughly researched topics in psychopathology, with over 2000 references in the psychological, medical, and speech literature since 1900. Despite this vast amount of research and the apparent simplicity of the disorder, there is as yet no generally accepted theory or treatment for stuttering, nor even a standard definition.

Stuttering appears to be an universal phenomenon, unrelated to intelligence and socio-economic status. Slight differences in incidence figures within and across cultures probably reflect the lack of a standard definition of stuttering, plus the possibility that it is continuous with normal disfluency. Whether it arises out of normal childhood disfluency is not known. At any rate, nearly all stuttering has its onset between the ages of two and seven (Van Riper, 1971). Male stutterers outnumber females by about three to one, for unknown reasons. The prognosis for child stutterers is good: about 80% remit spontaneously (Sheehan & Martyn, 1966). The prognosis for adult stutterers is relatively poor (Beech & Fransella, 1968).

It is common to speak of a genetic predisposition to stuttering, but this hypothetical mechanism has not been identified and thus has no explanatory value. Genetic research on stuttering is scanty, but it has been established that a child will have a disproportionately high risk of becoming a stutterer if he has relatives who stutter (Andrews & Harris, 1964). The possible modelling effect of contact with such relatives has usually not been controlled. Adequate modern studies of concordance are lacking.

Stuttering usually consists mainly of syllable repetitions and hesitations, plus obvious tension and diverse motor behaviors (e.g., blinking, head jerking, fist clenching) which are often considered to be superstitious operants. Stuttering can take many forms across individuals, and within individuals over time. Van Riper (1971) reviewed 63 nosological studies of this disorder and concluded that definite subgroups have not yet been defined. Nevertheless, the possibility that there are distinct types of stutterers remains an enticing one, for it would help account for the many inconclusive and contradictory results in the huge stuttering literature.

Stutterers can predict with considerable accuracy the words on which they will stutter (Bloodstein, 1972). Stuttering occurs most frequently at points where a normal speaker is likely to hesitate or pause, e.g., between phrases. It is not related to word length, grammatical form, or initial sound, but it is positively related to the propositionality (meaningfulness) of the message being communicated and to the perceived status of the listener. Stuttering tends to occur during manding, a state in which the speaker is experiencing deprivation or other aversive stimulation (Skinner, 1957).

It is well-established that stuttering tends to decrease "spontaneously" over time in a relatively constant stimulus situation (Beech & Fransella, 1968). This phenomenon is commonly called the adaptation effect. It has been attributed to extinction due to anxiety reduction (Johnson, 1959), although there are some differences between extinction and adaptation curves (Wingate, 1966). Gray and Brutten (1965) found adaptation occurred without a decline in palmar sweating, an index of anxiety, and concluded that adaptation is due to the accumulation of reactive inhibition. This interpretation is consistent with

the fact that after adaptation and a short rest period, during which time reactive inhibition could dissipate, stuttering tends to return to its baseline level. Another possibility is that adaptation is simply a special case of stimulus habituation (Van Riper, 1971). The adaptation phenomenon is not utilized in therapy, since it is very transient and does not transfer to extra-clinical situations. Instead, it is considered a nuisance variable which must be controlled, lest it raise false hopes in the naive researcher. Adaptation and spontaneous recovery can largely be eliminated by the simple expedient of changing speech stimuli rather than exposing the subject repeatedly to the same material, and by using continuous speech rather than allowing breaks.

A minority of adult stutterers overcome their handicap without speech therapy. For those who do not remit spontaneously, the clinical picture remains relatively bleak in spite of a vast amount of research. Stutterers have been treated with almost every conceivable variety of psychotherapy and behavior therapy, medicated with almost everything from ataractics to amphetamines, and even treated with surgery, ECT and acupuncture. A number of speech therapies which have no counterparts in psychology or medicine have also been developed specifically for stutterers. The various therapies have been thoroughly reviewed by Van Riper (1973), the undisputed dean of stuttering research and therapy, who after 40 years in the field remains a stutterer himself. Van Riper is pessimistic about the likelihood of a "cure" for stuttering, preferring instead to desensitize the stutterer to his problem so that he may learn to live with it. For Van Riper and his numerous followers, successful therapy involves turning a tense, anxious stutterer into a more relaxed, "fluent"

stutterer who no longer avoids the situational and phonemic cues which precede stuttering.

Three of four recently developed therapies appear to have considerable potential, however. One of these is rate-control therapy (e.g., Perkins, Note 1) in which behavioral techniques are used to train the stutterer to speak at a slow rate. Nearly all stutterers can be made fluent this way, but maintenance of fluency involves continuing to speak abnormally slowly at times. In a six-month follow-up study, Perkins found that 92% of his stutterers had retained some improvement and 53% maintained "normal" fluency, although speech rate data were not given. Adequate independent follow-up studies of rate control therapy remain to be done.

It has been known for centuries that stuttering is reduced dramatically when the stutterer paces his words or syllables with an iterated stimulus, e.g., a metronome. This effect is not due to a deficiency in the perception or production of rhythm on the stutterer's part (Rotter, 1955). Rhythm per se is not an essential property of the stimulus: rhythmic and arhythmic beats of equal predictability are equally effective in suppressing stuttering (Brady, 1969). Nor is the metronome effect merely a variant of rate control therapy, for its effect has been found to be independent of speech rate (Hanna & Morris, Note 2). Van Riper (1971) suggested that the metronome acts as a timing device to integrate the motor patterning of speech, but it is not known how this occurs. Unfortunately, the metronome effect is apparatus-bound: it wears off in a matter of minutes. Still, it may have therapeutic potential.

A great deal of stuttering research and therapy has been based on the assumption that stuttering is symptomatic of neurosis. The extreme state-

ment of this position is the orthodox psychoanalytic view (Fenichel, 1945), wherein stuttering is a symptom of a pregenital conversion neurosis which involves a symbolic upward displacement of the anal sphincter. Stuttering thus allows the speaker to smear his listener with symbolic feces. Other psychodynamic interpretations are less entertaining but equally unconstrained by data. Although psychotherapy remains one of the most common treatments for stuttering, its value remains to be demonstrated (Perkins, 1973; Van Riper, 1973). Moreover, reviewers of the personality literature have consistently concluded, sometimes to their own surprise, that the case for stuttering as a neurosis has not been made. There is little unequivocal evidence that stutterers as a group are more neurotic or maladjusted than nonstutterers (Beech & Fransella, 1968; Sheehan, 1970). According to Van Riper, "most of our stutterers have been pretty normal individuals -- except when they had to say something" (1971, p. 272). In the relatively few cases where neurotic symptoms are present, they probably represent the outcome of years of communicative frustration and social penalties. The popular stereotype portrays the stutterer as a neurotic introvert, as exemplified by P-P-Porky P-Pig. While some stutterers undoubtedly fit this description, as a whole they do not. There is no evidence to suggest that the stutterer has a particular personality profile or group of traits that differentiates him or her from the nonstutterer. The bulk of a huge body of research has failed to demonstrate that stutterers as a group have basically different personalities from normal speakers, even after years of stuttering (Beech & Fransella, 1968; Van Riper, 1971).

Much of the recent psychological research on stuttering has been concerned with the possibility that it is essentially a learned behavior.

Brutten and Shoemaker (1967) have proposed an elegant two-factor theory of stuttering. Stress is known to cause fluency failure in normal speakers. Through repeated exposure to stress, negative emotion may become classically conditioned to certain stimuli, e.g., speaking to an authority figure. The pattern of autonomic activity which defines negative emotion causes disintegration of the precisely-timed motor behavior of speech. Thus the hesitations, repetitions, breathing abnormalities etc. which constitute the core behaviors of stuttering are thought to be respondents. In the second stage of the development of stuttering, secondary symptoms are acquired by some individuals through escape and avoidance conditioning, e.g., head-jerking that occurs during stuttering may be reinforced by eventual fluent utterance and by the termination of self-generated and listener-generated aversive states. Of course, the fact that such operants are reinforced intermittently makes them highly resistant to extinction. Van Riper (1973) developed such gross head-jerking behavior that his stuttering was once mistaken for an epileptic seizure.

As therapy, Brutten and Shoemaker prescribe reciprocal inhibition therapy to weaken the core of classically conditioned negative emotion, plus the optional treatment of secondary symptoms through unreinforced massed performance. Although some early reports of reciprocal inhibition therapy for stutterers were not encouraging (e.g., Lazarus, 1963; Wolpe, 1961), there have been some positive results recently (Adams, 1971; Webster, 1970). Lanyon (1969) and Tyre, Maisto, and Companik (1973) reported a decrease in both stuttering and state anxiety after reciprocal inhibition therapy, but Gray and England (1972) found that these occurred at different times. Stuttering was reduced early in treatment when anxiety, measured in terms of skin conductance

scores, was high. Anxiety seems to have been alleviated a few sessions after stuttering had decreased. This is inconsistent with the two-factor theory.

The two-factor theory predicts that the core behaviors of stuttering would be increased by punishment, through the production of disruptive autonomic activity. There are reports that random punishment does indeed increase stuttering (Siegel & Martin, 1965; Brookshire & Eveslage, 1969), although this effect was not found by Biggs and Sheehan (1969). Contingent punishment, however, has usually been found to increase stuttering. Such contingent aversive stimuli have included verbal reprimand (Quist & Martin, 1967), very loud noise (Flanagan, Goldiamond & Azrin, 1958; Siegel & Martin, 1965), electric shock (Daly & Cooper, 1967), and even the blinding flash of a photographer's 100-candlepower gun (Brady, 1968). On balance, the bulk of recent research on punishment supports a purely operant view of stuttering, as opposed to the two-factor theory.

Stuttering has other operant properties as well. It has been temporarily instated in normal speakers through positive and negative reinforcement (Van Riper, 1973; Flanagan, Goldiamond, & Azrin, 1958). Reinforcement of fluency reduces stuttering (Van Riper, 1973; Perkins, Note 1), as do response cost (Halvorson, 1971) and stutter-contingent time-out from speaking (Haroldson, Martin & Starr, 1968). Once established, a stutterer's fluent speech can be brought under stimulus control (Martin & Siegel, 1966).

If stuttering is an operant, its acquisition and maintenance schedules are not fully understood (Sander, 1975). Many operant researchers assume that normal speech includes stutters, but at such a low frequency that no problem is apparent (Goldiamond, 1965). In some children, repetitions and other dis-

fluencies that occur during manding may be unintentionally reinforced by parents and other listeners through attention and noninterruption. Furthermore, repetitions and hesitations may serve to postpone the aversive consequences of stuttering (Shames & Sherrick, 1963). However, self-generated and listener-generated punishment probably occur prior to reinforcement (i.e., fluent utterance), and thus it is difficult to understand how stuttering is maintained. Halvorson (1973) has suggested that stuttering and its punishment may be the initial components of a behavior chain terminating with reinforcement for fluency. At any rate, the value of the operant approach is illustrated by therapies which have successfully utilized shaping (Perkins, Note 1), stimulus control (Azrin, Jones, & Flye, 1968), self-recording (LaCroix, 1973), time out (James & Ingham, 1974), social reinforcement for fluency and punishment for stuttering (Ryan, 1971; Quist & Martin, 1967), and even token economies (Ingham & Andrews, 1973b).

Although most researchers probably would agree that many of the symptoms of stuttering are learned, some suspect that the essential cause of the disorder is a disturbance of auditory feedback. "Artificial stuttering" may be induced in a large proportion of normal speakers be feeding back their tape-recorded speech at a delay of about .20 seconds (Lee, 1951). Apparently this delayed auditory feedback (DAF) disrupts the precise timing required for the simultaneous and successive contractions of the dozens of antagonistic muscles involved in speech. However, DAF has a paradoxical effect on many stutterers: it makes them "artificially fluent". DAF is not a viable treatment at this time because there is little carry-over of fluency from lab to life, but it has spawned several intriguing models of stuttering as a feedback

disturbance (e.g., Gruber, 1965; Mysak, 1960; Sklar, 1969). Speech is a finely-integrated motor activity which presumably involves at least three kinds of feedback:

1.) airborne auditory feedback

2.) bone-conducted and tissue-conducted auditory feedback, and

3.) somesthetic (i.e., tactile, kinesthetic, and proprioceptive) feedback from the speech musculature.

Left-right feedback asynchrony, a possible source of instability, has been found in some stutterers (Stromsta, Note 3). Another source of feedback distortion might be temporal differences between competing channels, and indeed this has been demonstrated for channels 1 and 2 (Stromsta, 1962). In other words, some stutterers may have an organic DAF system. Stress presumably causes this unstable feedback system to fail, e.g., Timmons and Boudreau (1972) hypothesized that anxiety produces changes in the contour of the oral cavity, which lead to further feedback distortion, greater anxiety, and so on. The stutterer carries his own stressers with him, in the form of negative emotion conditioned to situational and phonemic cues. Each failure to be fluent presumably adds to future stress.

DAF must usually be at least as loud as the speaker's own speech to be effective, and it is most disruptive when loud enough to mask bone conduction, i.e., greater than 50 dB above threshold (Butler & Galloway, 1957). Yates (1963) hypothesized that nonstutterers whose speech is not disrupted by DAF actually monitor their speech via channel 2 and/or 3. Indeed, Goldiamond, Atkinson and Bilger (1962) found that nonstutterers can become relatively resistant to DAF when instructed not to listen to it.

The defective feedback theory is supported by another phenomenon as well: many stutterers become artificially fluent when listening to white noise at a loudness of approximately 90 dB above threshold (Cherry & Sayers, 1956). Speech therapists call this treatment "masking noise" because it minimizes or eliminates the stutterer's hearing of his speech via channels 1 and 2. Like DAF, masking noise generally has no long-lasting therapeutic effect but it is of considerable theoretical interest because it apparently forces the stutterer to monitor his speech through channel 3.

The therapeutic implications of the feedback model of stuttering are clear: If channels 1 and 2 are somehow defective, therapy should involve teaching the stutterer to rely on channel 3 (Mysak, 1960; Gruber, 1965). This could be achieved through masking channels 1 and 2 by applying loud white noise to the ears (Sklar, 1969). An alternative strategy, which is the essence of this dissertation, involves enhancing proprioceptive information by feeding it through the airborne auditory feedback channel.

Biofeedback

Biofeedback training is based on the fundamental learning principle that we can learn to make a given response when we receive information that we have made that response, or an approximation to it (Lazarus, 1975). Immediate feedback is the most effective, e.g., feedback from the eyes facilitates control of the striate muscles involved in writing a sentence. Under ordinary conditions, we do not receive much feedback about certain physiological processes, e.g., the fine activity of the posterior cricoarytenoid muscle in the larynx, but biofeedback can make this information more salient.

In the past decade, biofeedback has been shown to have important theoretical ramifications as well as potential for the treatment of a host of disorders. According to Budzynski (1973):

> "An accumulating body of clinical and research evidence suggests that biofeedback represents a relatively effective technique for the shaping of self-control over certain physiological events. These events are usually autonomous in that they tend to occur automatically and below the level of awareness. When these internal events fall outside the normal range of functioning, they constitute maladaptive behaviors that can lead to feelings of anxiety, or the appearance of such stress-related disorders as migraine and tension headaches, certain cardiovascular problems, and sleep-onset insomnia, to name a few. Through feedback training, patients learn to maintain their physiology within a normal range of functioning." (p. 546).

In view of the possibility that stuttering is the result of defective feedback, an investigation of the effects of feedback training on stuttering seems warranted.

Some of the earliest and most important biofeedback research, from a theoretical point of view, involved operant conditioning of autonomic responses. These included reward and avoidance conditioning of smooth muscle responses such as heart rate (Miller & DiCara, 1967) and vasomotor behavior (Snyder & Noble, 1968); and of glandular responses such as salivation (Miller & Carmona, 1967) and GSR (Snyder & Noble, 1968). Miller, DiCara and colleagues curarized and artificially respirated their animal subjects in order to control for the possibility that the autonomic responses were actually mediated by skeletal muscles under voluntary control. Under these conditions their subjects were able to increase and decrease their heart rate and blood pressure, produce localized blood flow in one ear without producing such a change in the other ear, modify their stomach and intestinal contractions, and vary

their rate of urine secretions. Results like these, if they can be replicated, provide the basis for a reinterpretation of psychosomatic symptoms in humans, viz., operant conditioning of autonomic responses.

Another early line of biofeedback research has involved attempts to teach human subjects to produce or suppress alpha waves (Kamiya, 1969). Facilitation of alpha was initially touted as a royal road to altered states of consciousness and other ill-defined experiences. It now appears that alpha activity is experienced only as normal relaxation. Moreover, it appears that facilitation and blocking of alpha did not involve operant conditioning at all, as originally claimed. Alpha can be readily blocked by eye movement and facilitated by lack of eye movement. Thus what was first thought to be evidence of operant conditioning of EEG activity now appears to be an artifact of responses which were already in the repertoire, viz., oculomotor activity. Moreover, the magnitude of EEG changes did not exceed the limits of baseline fluctuations (Peper & Mulholland, 1970).

In spite of the fact that some early results have not been substantiated, biofeedback does appear to have considerable clinical potential. Auditory feedback of GSR has been used in the treatment of agoraphobia (Lader & Wing, 1966) and generalized phobia (Lader & Mathews, 1968). Sargent, Green and Walters (1973) taught their subjects to control the difference in skin temperature between the forehead and the right index finger. This study has implications for the treatment of migraine headaches, since skin temperature is directly related to blood flow. Elder, Ruiz, Deabler and Dillenkoffer (1973) taught essential hypertension patients how to reduce their diastolic blood pressure by attending to a visual signal. Rosen (1973) used a similar

signal, viz., a red light to inform his subjects when they had excessive penile tumescence. This study has potential for the treatment of psychogenic impotence, fetishism and other sexual disorders. Lovibond and Caddy (1971) used feedback of blood alcohol level to teach alcoholics to drink without becoming profoundly intoxicated. Other disorders currently being treated with biofeedback include epilepsy, premature ventricular contractions, cardiac arhythmias, stomach acidity, and excessive perspiration (Miller, Barber, DiCara, Kamiya, Shapiro & Stoyva, 1974).

Feedback of electromyographic activity (EMG) is particularly promising. Self-control of EMG responses can be achieved relatively easily (Green, Green & Walters, 1971), presumably because it involves striate muscle functions. Using indwelling electrodes, Basamajian (1963) has demonstrated that humans can make fine adjustments of single motor units in the thumb muscles. Surface EMG has been used to treat several disorders which involve excessive muscle tension. Hardyck, Petrinovitch and Ellsworth (1966) eliminated subvocalization in students with reading problems by providing them with auditory feedback of laryngeal muscle activity. Jacobs and Felton (1969) used visual EMG feedback to induce relaxation in neck-injured patients. Auditory feedback of frontalis muscle activity has been used to treat tension headaches (Budzynski, Stoyva, Adler & Mullaney, 1973) and chronic anxiety and insomnia (Raskin, Johnson & Rondestvedt, 1973). Netsell and Cleeland (1973) reduced lip hypertonia in a Parkinsonian patient by means of EMG feedback and Cleeland (1973) also applied this paradigm to the modification of spasmodic torticollis. A general review of the current state of EMG feedback is provided by Basmajian (1972).

Biofeedback is a remarkable form of behavior therapy in that subjects often achieve strong positive results in a single session, especially when the striated musculature is involved. This learning can be achieved without reinforcement other than knowledge of the results, i.e., the feedback itself. Curiously, subjects usually are unable to verbalize how they exerted control over a given physiological response, any more than one could describe how to move a finger.

In order to apply the promising biofeedback paradigm to stuttering, it was necessary to select a psychophysiological correlate of this disorder. Several such correlates have been investigated, although the amount of research in this area is perhaps less than one might expect from the immense volume of the stuttering literature in general. Severe stuttering is often accompanied by a host of obvious bodily movements, e.g., eyeblinks, gasping, swallowing, head nodding, lip tremors and gross movements of the limbs. There may also be increases in palmar sweating (Brutten, 1963), increases in GSR (Kline, 1959), abnormal EEG activity (Knott, Correll & Shepherd, 1959), and pupil dilation (Luchsinger & Arnold, 1965). Williams (1955) monitored masseter EMG and found that action potentials from this site were significantly higher during stuttering than during fluency. Spikes appeared on the EMG record during stuttering, but disappeared when stutterers deliberately made relatively relaxed repetitions of syllables. Moreover, when normal speakers faked stuttering, spiking was produced. Williams concluded that stuttering is accompanied by excessive tension in the jaw muscles. Sheehan and Voas (1954) found that masseter tension peaked before stuttering. Shrum (Note 4) failed to replicate this finding but did find more tension in the neck, chest

and jaw during stuttering than during fluency.

EMG appeared to be the most reactive physiological parameter during stuttering and thus was a promising modality for feedback training. It was assumed that EMG would probably discriminate stuttering from fluency in a large proportion of subjects, although it was also realized that each stutterer has his own profile of responses.

After some pilot research, the masseter site was rejected in favour of electrode placement near the larynx, for several reasons: First, since the majority of stutterers are male, the masseter site is often covered with sideburn hair. Second, movement artifacts are common at the masseter site but less common near the larynx. More spiking was recorded from the layrngeal site than from the masseters in pilot research. And finally, there is evidence that the larynx may be a locus of the stuttering block (Schwartz, 1974). Thus I decided to investigate the effect of feeding back stutterer's EMG activity from a site near the larynx.

Stuttering research and biofeedback research are both notable for initially positive results that tend not to be substantiated later (Van Riper, 1973; Blanchard & Young, 1973). Green, Green and Walters (1971) touted feedback training as a cure for everything from warts to cancer. Budzynski (1973) warned against such high expectations and urged that carefully controlled laboratory studies be done before clinical studies are attempted. In stepwise research, demonstration of a treatment effect precedes the evaluation of that treatment. The intent of the present research was not to devise a new speech therapy at this time, but rather to observe the effect of EMG feedback in a controlled laboratory situation. If it were

shown that biofeedback did suppress stuttering, then biofeedback would have potential as speech therapy. However, the development of an effective therapeutic program was not within the scope of the present research.

CHAPTER II

GENERAL METHODOLOGY

Subjects

Severe chronic stuttering is relatively rare in adults. The incidence of stuttering in the general population is less than 1% (Beech & Fransella, 1968), but even this figure may lead one to underestimate the difficulty of procuring subjects for research. The prevalence of stuttering is undoubtedly much lower than the incidence, because 1) the rate of spontaneous remission in young stutterers is about 80% (Sheehan & Martyn, 1970), and 2) even in adults, stuttering is often an intermittent disorder which may disappear for days or weeks and then recur. Probably the prevalence of severe chronic stuttering in adults is a small fraction of 1%.

Stuttering research has always been hampered by small sample sizes. The median sample size in 70 articles on stuttering published in the Journal of Speech and Hearing Disorders and the Journal of Speech and Hearing Research from 1970 through 1974 was 14. Some researchers cope with this problem by having fluent subjects simulate stuttering (e.g., Williams, 1955; Freeman & Ushijima, Note 5). A more valid solution to the problemmof subject scarcity is the use of single-subject designs (e.g., Martin, 1968).

A total of 14 adult stutterers (11 males and three females, which approximates the usual sex ratio) participated in various phases of the present series of studies. This sample was very heterogeneous with regard to socio-economic status, ethnicity, education and age (range 17-49 years; mean age 27.3). All had received speech therapy or psychotherapy for their stuttering, without much success, and were on a waiting list for rate-control

therapy. Most reported that their stuttering was a severe social and occupational handicap. In general, these subjects seemed to be underachievers relative to their level of education (e.g., a 36-year-old PhD in physics was employed as a sawmill laborer). Most seemed deficient in social skills.

Five subjects participated in a single study, eight in two studies, and two in three studies. The exact assignment of subjects to studies is given in Appendix A. There were 30 single-subject studies of stuttering, 30 studies of EMG and 23 studies of speech rate in the present research. Statistical analysis was based on the number of observations per subject rather than on the number of subjects.

Dependent Variables

Stuttering. Is stuttering in the ear of the beholder, or is it sufficiently distinct from fluency that it can be defined and measured reliably? In a lengthy review of this issue, Van Riper (1971) argued:

> Just because stuttering is occasionally difficult to distinguish from normal disfluency in its early or minor forms (and we must remember that stutterers also have normal disfluencies) we need not deny that stuttering exists as an entity. (p. 15)

Van Riper concluded by defining stuttering as "a word improperly patterned in time and the speaker's reactions thereto" (p. 15).

While Van Riper's definition is certainly correct, it is not sufficiently specific. For the purpose of the present research, stuttering was defined as including:

1. Sound or syllable repetitions. These comprise the largest proportion of stutters for most subjects. "Kuh-kuh-kuh-Katy" would be counted as one stutter.

2. Abnormal prolongations of sounds. "Abnormal" would be in the order of about one second at the beginning of a word, or less than one second if the prolongation occurred within a word.
3. Abnormal hesitations. These are accompanied by the strange hissing noises called "vocal fry" (Moser, 1942) or by other indications of excessive tension and respiratory abnormalities, and are thus distinguishable from normal hesitations that result from not knowing what to say.

Thompson (1971) used a similar definition of stuttering ("hesitations, prolongations, part-word repetitions, undue pauses") and achieved a reliability coefficient of $\underline{r} = .87$ when two raters who had been trained to use this definition scored tapes for stuttering. Martin (1968), also scoring tapes, reported an inter-rater reliability of $\underline{r} = .98$. MacDonald and Martin (1973) used untrained students to count stutters and other disfluencies from videotapes, without giving them any a priori definitions of these two response categories. Nevertheless, they found high intra- and inter-rater reliability, in terms of percent agreement, and concluded that stuttering is an unambiguous and reliable response class even to untrained judges.

Azrin, Jones and Flye (1968) measured stuttering in terms of percent of words stuttered. This measure is satisfactory if all subjects use the same words (e.g., in a reading task) but it is clear that if two subjects have the same percentage score, the subject who used longer (polysyllabic) words will actually be more fluent. For this reason James and Ingham (1974) advocate percent syllables stuttered. Although this is not a direct measure of the duration of each stutter, judgements of the severity of stuttering

are highly correlated with frequency of stuttering and with speech rate (Young, 1961; Sander, 1961). Percent syllables stuttered is a measure of frequency, and controls for differences in speech rate, e.g., between adjacent one-minute segments of speech. According to Ingham and Andrews (1973a):

> ... changes in the severity of the disorder may be suitably assessed from frequency counts of moments of stuttering and possibly from measures of the rate of speaking. These two indices can also be measured "on line" with high reliability (Ingham and Andrews, 1971b), thereby providing useful indices of a subject's speech behavior. (p. 407)

EMG spiking. Since the auditory feedback tone used in this research was activated by the same electrical activity which produced spikes and other pen deflections in the EMG record, a count of EMG spikes was chosen as an appropriate measure of the effect of feedback training on EMG activity.

Because of differences in basal levels, the threshold at which the feedback tone was activated was determined individually for each subject during the baseline period (Appendix C). Depending on the subject's own pattern of EMG activity and on the sensitivity level at which the polygraph was set, fluent speech produced pen deflections representing not more than .20 mV. Stutters, on the other hand, were often (but not inevitably) accompanied by conspicuous spikes representing up to .50 mV. Signals attributable to yawning, swallowing and movement artifacts were readily distinguishable from stutter spikes and were not counted.

Spikes, like stutters, were expressed relative to speech rate. Thus the degree of spiking was expressed as the number of spikes per 100 syllables for each minute of speech.

Speech rate. When normal speakers are required to speak abnormally

fast, they become disfluent. Stutterers tend to speak faster than normals and can become more fluent by slowing down (Van Riper, 1971, 1973). This trade-off between rate and fluency is the essence of rate-control therapy and may be involved in other therapies as well. In order to determine that a biofeedback effect was independent of rate control, it was necessary to monitor speech rate, which is usually expressed in syllables per minute (SPM). It should be noted that even if a biofeedback effect were accompanied by or even produced by a retarded speech rate, this would not necessarily detract from its effectiveness.

It is important to note that SPM is only an estimate of true articulatory rate, since it does not take into account pauses between moments of speech. It is a conceivable paradox, although not a likely one, that an increase in SPM could be produced by a retarded articulatory rate if slower speech were accompanied by a decrease in time-consuming pauses, hesitations and repetitions. That is, syllable prolongation could occasionally be more than offset by a decrease in the time occupied by stutters. A true measure of articulatory rate would include speech only, and eliminate all pauses. In practice, it would be so difficult to calculate a true articulatory rate that SPM is virtually always used instead. SPM is an adequate estimate of articulatory rate for our purposes, and the possibility that it could occasionally be misleading is outweighed by the probability that it would not be misleading in several replications.

Observer Agreement on the Dependent Variables

One subject was selected at random from each of the five studies in this dissertation, and a 10-minute sample of speech was randomly selected

from each of these subjects. Stuttering and speech rate data were calculated from tape recordings, and EMG spikes were counted from the corresponding polygraph records. These data were tallied on hand counters by myself and a practicing speech therapist who was unaware of the experimental hypotheses. Stuttering was defined according to the definition described earlier in this chapter. An EMG spike was defined as a sharp downward deflection of the EMG pen trace, which exceeded a certain cutoff (viz., the feedback threshold). Syllables that were repeated during stuttering were only counted once. Before scoring the data, both raters practiced on an additional speech sample, in order to ensure that the definitions of the dependent variables were fully understood.

Inter-rater reliability data are given in Appendix B. Mean inter-rater reliabilities of .89, .93, and .95 were achieved for stuttering, spiking, and speech rate, respectively, using Fisher's Z-transformation. All reliability coefficients were significant at the .01 level (critical value of $\underline{r}(8) = .77$). However, a high degree of reliability does not necessarily imply agreement on specific instances, so an additional analysis was performed. Agreement between raters was calculated according to the formula

$$\% \text{ agreement} = \frac{\text{no. of agreements}}{\text{no. of agreements \& disagreements}} \times 100$$

Using this more conservative measure, mean agreements of 80%, 92% and 97% were found for stuttering, spiking, and speech rate, respectively (Appendix B). A lesser degree of agreement was consistently found for stuttering and spiking than for speech rate. This is in part due to the fact that the former variables were expressed relative to a denominator of 100 syllables,

and thus included the error variance of the speech rate measure. Less agreement was found for stuttering than for spiking, probably because the low base rate of the former (e.g., 2%-20% syllables stuttered) minimized observer agreement due to chance alone. Moreover, the distinction between stuttering and fluency is sometimes a subtle one.

The high degree of agreement between the two raters indicates that the data are not seriously contaminated by random error and experimenter bias, i.e., nonrandom error which is confounded with the treatment effect. This tends to inspire confidence in the results of the studies that follow.

Design Design

Time series methodology has been used for decades in the experimental analysis of behavior, and recently has gained increasing acceptance in other areas of behavioral research (Sidman, 1960; Chassan, 1967; Leitenberg, 1973). Campbell and Stanley (1970) define a time-series design as

0 0 0 I 0 0 0

where each 0 represents a regular observation of the dependent variable and I represents the experimental intervention. Actually the basic design used in the present series of studies is better illustrated as

0 0 0 I0 I0 I0

because the intervention continues throughout the treatment period. A summary of designs used in the present research is given in Table I.

Time-series designs appear to be particularly appropriate in psychophysiology (Johnson & Lubin, 1972) and stuttering research (Martin, 1968).

Table 1

Designs Used in the Present Series of Studies

Study	Schematic	Intervention
1	$O_1\ I_1\ O_2$	I_1 = rate control therapy
2 (a)	$O_1\ O_2 \cdots O_{15}\ I_2O_{16}\ I_2O_{17} \cdots I_2O_{30}$	I_2 = metronome paced speech
(b)	$I_2O_1\ I_2O_2 \cdots I_2O_{15}\ O_{16}\ O_{17} \cdots O_{30}$	
3 (pilot)	$O_1O_2 \cdots O_9$ /break/ $I_3O_{10}\ I_3O_{11} \cdots I_3O_{18}$ $O_{19}\ O_{20} \cdots O_{27}\ I_3O_{28}\ I_3O_{29} \cdots I_3O_{36}\ O_{37}$ $O_{38} \cdots O_{45}$	I_3 = feedback
3	$O_1\ O_2 \cdots O_{15}\ I_3O_{16}\ I_3O_{17} \cdots I_3O_{30}$	
4 (a)	$O_1\ O_2 \cdots O_{15}\ I_4O_{16}\ I_4O_{17} \cdots I_4O_{30}$	I_4 = feedback without instructions
(b)	$I_4O_1\ I_4O_2 \cdots I_4O_{15}\ O_{16}\ O_{17} \cdots O_{30}$	
5 (pilot)	$O_1\ O_2 \cdots O_9$ /break/ $I_3O_{10}\ I_3O_{11} \cdots I_3O_{18}$ $O_{19}\ O_{20} \cdots O_{27}\ I_5O_{28}\ I_5O_{29} \cdots I_5O_{36}\ I_3O_{37}$ $I_3O_{38} \cdots I_3O_{45}$	I_3 = feedback I_5 = false feedback
5 (a)	$O_1\ O_2 \cdots O_{15}\ I_3O_{16}\ I_3O_{17} \cdots I_3O_{30}$ /break/ $O_{31}\ O_{32} \cdots O_{45}\ I_5O_{46}\ I_5O_{47} \cdots I_5O_{60}$	
(b)	$O_1\ O_2 \cdots O_{15}\ I_5O_{16}\ I_5O_{17} \cdots I_5O_{30}$ /break/ $O_{31}\ O_{32} \cdots O_{45}\ I_3O_{46}\ I_3O_{47} \cdots I_3O_{60}$	

A time-series analysis is an economical and efficient way to begin researching a new area, such as in the present instance, because it can be used with a single subject. Where subjects are rare, this method is almost inevitable. The power of a time-series analysis is a function of the number of data points rather than the number of subjects. Another advantage of this approach is its flexibility. In stepwise research, early results can be used to determine the course of subsequent studies without seriously depleting a small sample of rare subjects. (This is illustrated by the way in which Studies 3 and 5 differ from their pilot studies.)

A related advantage of single-subject designs is that they permit replication with a minimum of subjects. Inter-subject replication may be more valuable than inter-group replication where the reliability and generality of a treatment effect are of interest. In group designs, replication of changes in central tendency requires relatively large samples, and may obscure within-group differences (Sidman, 1960).

Within-group differences are particularly common in stuttering (Van Riper, 1971) and psychophysiology (Lacey, 1967). I once had a client who stuttered on 77% of his syllables. Consider the effect of grouping this subject with five others who each stuttered severely on 5% of their syllables: a strong biofeedback effect on the latter five subjects could be obscured partially or completely by a weak effect on the former subject, and vice-versa. In exploratory investigations like the present research, individual differences need to be scrutinized closely rather than ascribed to error variance. It makes a great deal of sense to use each subject as his own control. An untreated control group is an unattainable luxury where

subjects are scarce, and seems particularly inappropriate in the present research where all subjects have been stuttering severely for 15-45 years. (However, in Study 5 each subject participated in a pseudotreatment control condition as well as serving as his own control.)

Another unique advantage of the time-series design is its longitudinal perspective. Time-series research involves continuous measurement rather than only pre- and post-treatment measures, thus permitting process as well as outcome research. The time series illustrates the immediacy and duration of treatment effects, plus changes in treatment effects over time. This kind of information is clearly of great interest in biofeedback and therapy research.

Of course, the time-series design has certain potential disadvantages as well. One of these is historical invalidity, where a variable other than the treatment may account for any effect (or lack of effect) that occurs after the point of intervention (Campbell & Stanley, 1970). Historical invalidity is a major problem in ex post facto analyses, e.g., investigations of archival data. In the present planned time series analyses it is not a relevant problem, because the interval between pretreatment and posttreatment observations is only 60 seconds. Replication is a recognized defense against historical invalidity.

Another potential problem is regression to the mean (Glass, Willson, & Gottman, 1972). This is of major concern in true clinical research where subjects come for treatment during a personal crisis. Any spontaneous change back toward baseline would appear to be an effect of therapy. In stuttering research a regression artifact could occur if the treatment coincided with

a large spontaneous fluctuation in stuttering in either direction. A subsequent decrease in variance would tend to look significant, even in the absence of a change in level or slope of the time series. This could be controlled statistically through the use of an integrated moving average, so that no extreme value occurred at the end of the baseline period. Replication would be an additional precaution.

Where two or more treatments are applied during a single time-series experiment, there is a risk of multiple treatment interference (Campbell & Stanley, 1970). This possibility can be controlled by counterbalancing, by appropriate statistical analysis, and by a rest period between treatments (e.g., Study 5).

Until recently, the time-series design has been used without benefit of statistical analysis. Few would argue that the experimental analysis of behavior has been held back because of a disregard of statistics; nevertheless, mere visual inspection of the data can markedly increase the risk of inappropriate inferences (Gottman, 1973).

Chassan (1967) suggested that a standard *t*-test could be applied to pre- and post-intervention data. Others have used analysis of variance. It is not difficult to imagine instances where such analyses might be misleading, e.g., a strong adaptation effect would tend to produce a spuriously significant difference in pre- and post-intervention means. Moreover, stuttering data (and most time-series data, for that matter) are dependent and thus violate a basic assumption of *t*-tests and analysis of variance, viz., that errors associated with observations must be independent. Although the time series is basically an extension of the pretest-posttest design, it

raises unique statistical problems.

Fortunately, techniques have recently been developed which permit valid statistical inference from the time-series data (Glass, Willson & Gottman, 1972). The analysis used in the present research is based on a program developed by Maguire and Glass (1967). It uses an integrated moving average model with deterministic drift to transform the pre- and post-intervention data and test for significance between them. This model can accomodate some instability in the baseline data. The least-squares estimate of the level of the time series has a t distribution with N-3 degrees of freedom when divided by its standard error, where N equals the number of observations in the time series. An abrupt shift in the level of the series at the point of intervention implies a treatment effect. A delayed treatment effect would be relatively unlikely to reach statistical significance. Abrupt or delayed changes in the drift and variance of the time series are not analyzed, but fortunately they are of little interest here. Since multivariate statistics for time series analysis have not yet been perfected, each dependent variable must be analyzed separately.

CHAPTER III

STUTTERING AND LARYNGEAL HYPERTENSION

Laryngeal Involvement in Stuttering

As mentioned previously, a physiological correlate of stuttering is a necessary prerequisite to a biofeedback treatment. Laryngeal EMG was selected because of accumulating evidence of laryngeal involvement in the stuttering block.

It has long been known that stuttering is accompanied by abnormalities in respiration (Van Riper, 1971). Since air-flow (usually exhalation) is a necessary condition for phonation, some speech pathologists are of the opinion that what the stutterer calls a "block" is indeed a physiological block, i.e., a temporary occlusion of the airway. According to Van Riper (1971), such a block could occur at one or more of four loci: at the lips, at the front or rear of the tongue, or at the larynx. Van Riper suggested that the site of the stuttering block need not be the same for all stutterers.

Surprisingly, the role of the larynx in stuttering has largely escaped scrutiny until recently. Laryngeal involvement in airway blockage is certainly plausible, for the glottis is the smallest aperture along the airway. Freeman and Ushijima (Note 5) used depth electrodes to take EMG recordings from four intrinsic laryngeal muscles during the speech of a single stutterer and a normal speaker who faked stuttering. It was found that fluent speech was accompanied by a precise reciprocal integration of adductor and abductor muscles, but that this reciprocity was not present during stuttered speech. In other words, stuttering is literally a disintegration of speech. These researchers also found generally higher levels of laryngeal muscle activity

during stuttering than during fluency, presumably because stuttering involves simultaneous antagonsitic adductor-abductor activity.

Schwartz (1974) provided the definitive statement of the position that stuttering is a disorder of laryngeal hypertension. During normal respiration the vocal folds abduct slightly with inhalation and adduct slightly with exhalation. This abduction and adduction is presumably due to the contraction or inhibition of the posterior cricoarytenoid (PCA), one of the strongest intrinsic muscles of the larynx. Uninhibited contraction of the PCA would probably prevent phonation by blocking or constricting the airway. Schwartz argued that the normal supramedullary control of the PCA is disrupted during psychological stress. Stress produces abnormalities of respiration and the PCA responds to these with a vigorous, inappropriate abductive response. An individual could attempt to overcome this response in several ways, e.g., forceful contraction of other laryngeal muscles or of the lips, tongue and jaw. Unfortunately, these responses produce air pressures which only perpetuate the excitation of the PCA. Eventually, however, the individual does manage to speak. The consequent reduction in stress is thought to reinforce the maladaptive struggle behaviors.

While the above hypotheses about laryngeal involvement in stuttering are plausible, they are based on insufficient data at this time. Further research is required to demonstrate that laryngeal hypertension is indeed intimately related to the stuttering block. During pilot research I noted that stutters were often accompanied by EMG spiking from the larynx, whereas fluency was accompanied by an absence of spiking. Similar results were found by Williams (1955), who placed electrodes on the masseters. I origi-

nally attempted to investigate the temporal relationship between spikes and stutters by recording laryngeal EMG on one polygraph channel, and using the adjacent channel to record speech. Any noise at the approximate frequency of human speech caused pen deflection away from a silent baseline. Unfortunately, sounds other than human speech caused artifacts which were not readily distinguishable from speech. Moreover, stuttering was often characterized by silent hesitations and thus was not always accompanied by pen deflections. In short, the speech channel did not permit discrimination between fluency and stuttering, or even between speech and silence. As an alternative a tape recorder was synchronised with the EMG record so that the temporal relationship between stuttering and spiking could be investigated with an accuracy of a fraction of a second.

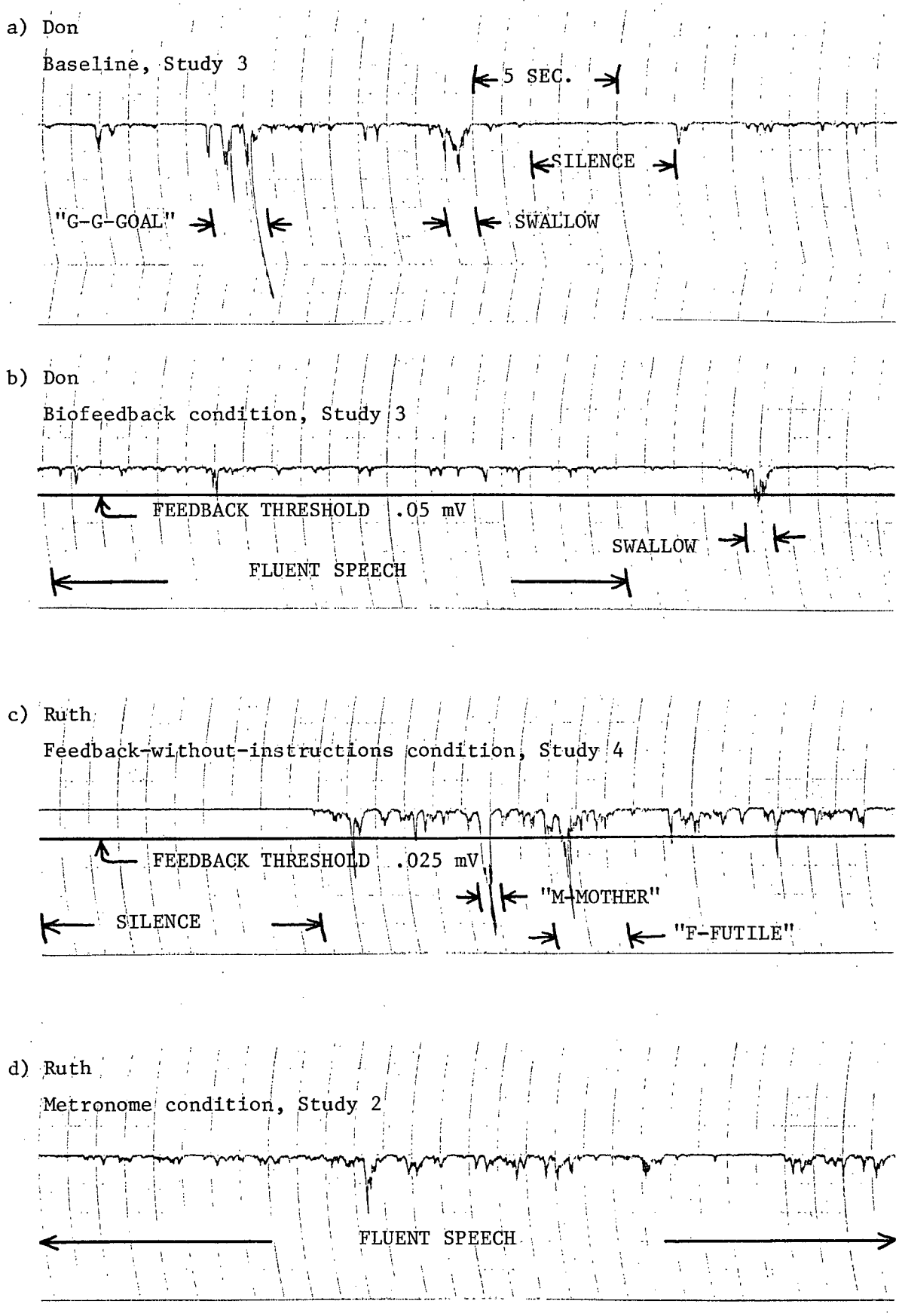
The results were striking. I found that often hesitations, repetitions, and prolongations which were counted as stutters were not accompanied by spiking, but most pen deflections not attributable to artifacts (e.g., yawning, swallowing) did occur during moments of stuttering. Depending on its severity, a single stutter may be accompanied by no spiking, by a single spike, or by more than 100 spikes. Some illustrations of the stutter-spiking relationship are given in Figure 1.

These pilot data were highly encouraging. Subsequently, two related studies were performed to explore the relationship between stuttering and EMG spiking.

Study 1

Stuttering can be reduced dramatically by rate control therapy, at

Figure 1. Sample EMG recordings.



least temporarily (Perkins, Note 1). It was predicted that if stuttering and spiking are indeed intimately related, suppression of stuttering would be accompanied by a similar reduction in spiking.

Method

Three subjects participated in this study. Each received an intensive course of speech therapy from myself and four other therapists. Five two-hour therapy sessions were held daily for three weeks (excluding week-ends). The therapy emphasized rate control, which was achieved by calculating speech rate and percent syllable stuttered and feeding this information back to the subject at the end of every five minute period of speech. Increases in fluency were rewarded monetarily, e.g., one dollar for every 10% reduction in the percentage of syllables stuttered. Stutters were punished by means of a loud, aversive tone. Transfer and maintenance of fluency were emphasized during the third week of treatment, e.g., the subjects were required to move out of the clinic and engage strangers in conversation while recording their own disfluencies covertly on golf counters. No attempt was made to induce the subjects to relax their speech muscles or any other muscles.

Measures of disfluency and laryngeal tension were taken at the beginning and end of the three-week treatment. Two surface EMG electrodes were attached 2 cm bilateral to the body center-line, approximately 1 cm above the thyroid prominence. A ground electrode was attached to the left wrist. The subjects sat in a padded armchair in a sound-proof room, while I recorded integrated EMG data on a Beckman R411 polygraph in an adjacent room. Head movement did not appear to produce EMG artifacts.

Cards from the Thematic Apperception Test (TAT) were used as stimuli

Table 2

Reduction in Stuttering as a Function of Rate Control Therapy

Subject	Percent syllables stuttered	
	Pre	Post
~~Frank~~		
Frank	12.04	0
Rick	14.65	3.12
John	17.91	2.80

Table 3

Reduction in EMG Spiking as a Function of Rate Control Therapy

Subject	EMG spikes per 100 syllables	
	Pre	Post
Frank	50.07	3.56
Rick	71.18	5.19
John	131.54	0.97

for speech. The subject's task was to speak for three minutes about the situation portrayed on each card. Each subject was exposed to five randomly-selected cards. The same procedure was repeated at the end of the three-week treatment, except that different cards were used. No attempt was made to interpret the TAT responses, as theseedo not differentiate stutterers from normal speakers (Sheehan, 1970).

Results

Stuttering and spiking data are presented in Tables 2 and 3 respectively. Time-series analysis was not used in this study because the three-week interval between the pre- and post-treatment measures allowed for historical confounding. Instead, a one-tailed t-test for related measures was used, yielding $t(2) = 11.52$, $p < .005$, for stuttering and $t(2) = 3.19$, $p < .05$, for spiking. Stuttering and spiking were positively correlated for all three subjects (Table 4). The mean correlation, using Fisher's Z-transformation, was $r(28) = .72$, $p < .01$.

Study 2

Stuttering can be almost totally suppressed by the simple expedient of instructing the subject to speak in time with a metronome beat (Brady, 1969; Hanna and Morris, Note 2). The rationale behind this study was similar to the previous one: if stuttering and EMG spiking are related, they should covary.

Method

The subject (David) performed the TAT task for 30 minutes while laryngeal EMG was recorded as in Study 1. During the last 15 minutes the subject

Table 4

Correlation Between Percent Syllables Stuttered and EMG Spikes per 100 Syllables in Study 1

Subject	r
Frank	.79*
Rick	.78*
John	.52*

* $p < .01$

Table 5

Correlation Between Percent Syllables Stuttered and EMG Spikes per 100 Syllables in Study 2

Subject	r
David	.15
Ruth	.37*
Don	.85**
Ray	.33

* $p < .05$

** $p < .01$

spoke in time with a metronome set at 120 beats per minute. This study was replicated with another subject (Ruth); then two other stutterers (Don and Ray) performed the speech tasks in reverse order. This balancing strategy was used to offset the adaptation effect.

Results

Stutters and EMG spikes were calculated from tape recordings and polygraph records for each minute of speech. David's data are presented graphically in Figures 2 and 3. Data for all four subjects are summarized in Table 6. Speech rate data were not analyzed in their own right, because the metronome setting of 120 beats per minute clearly produced a slowing of speech. Stuttering and spiking were significantly correlated for two of the subjects (Table 5). The mean correlation, using Fisher's Z-transformation, was $r(28) = .49$, $p < .01$.

Discussion of Studies 1 and 2

Two major conclusions can be drawn from these data. First, the rate control and metronome treatments were dramatically effective in the short run. (An informal three-month follow-up of the three rate control subjects revealed that one had relapsed completely, another had retained some improvement, and the third retained substantial improvement. The metronome effect, which had been even more striking, wore off within minutes.)

Second, and more to the point, stuttering and EMG spiking appear to be positively correlated. The relationship is close for some subjects, moderate for others, and negligible for two subjects, but on the whole it is significant. Possibly the correlation would have been higher except for the fact that the number of spikes is related to the duration of a stutter, which

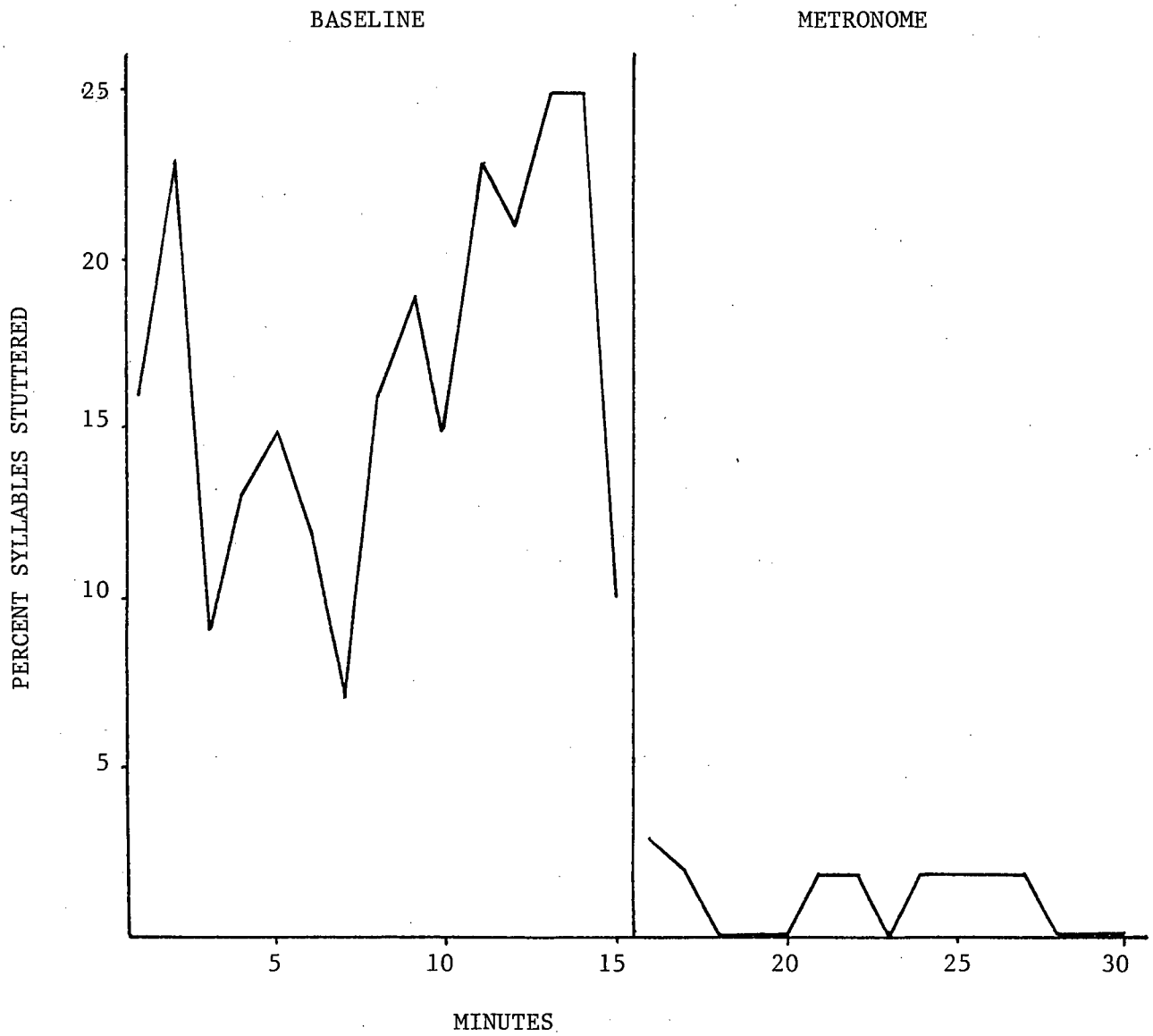


Figure 2. Effect of metronome on stuttering: David ($p < .005$).

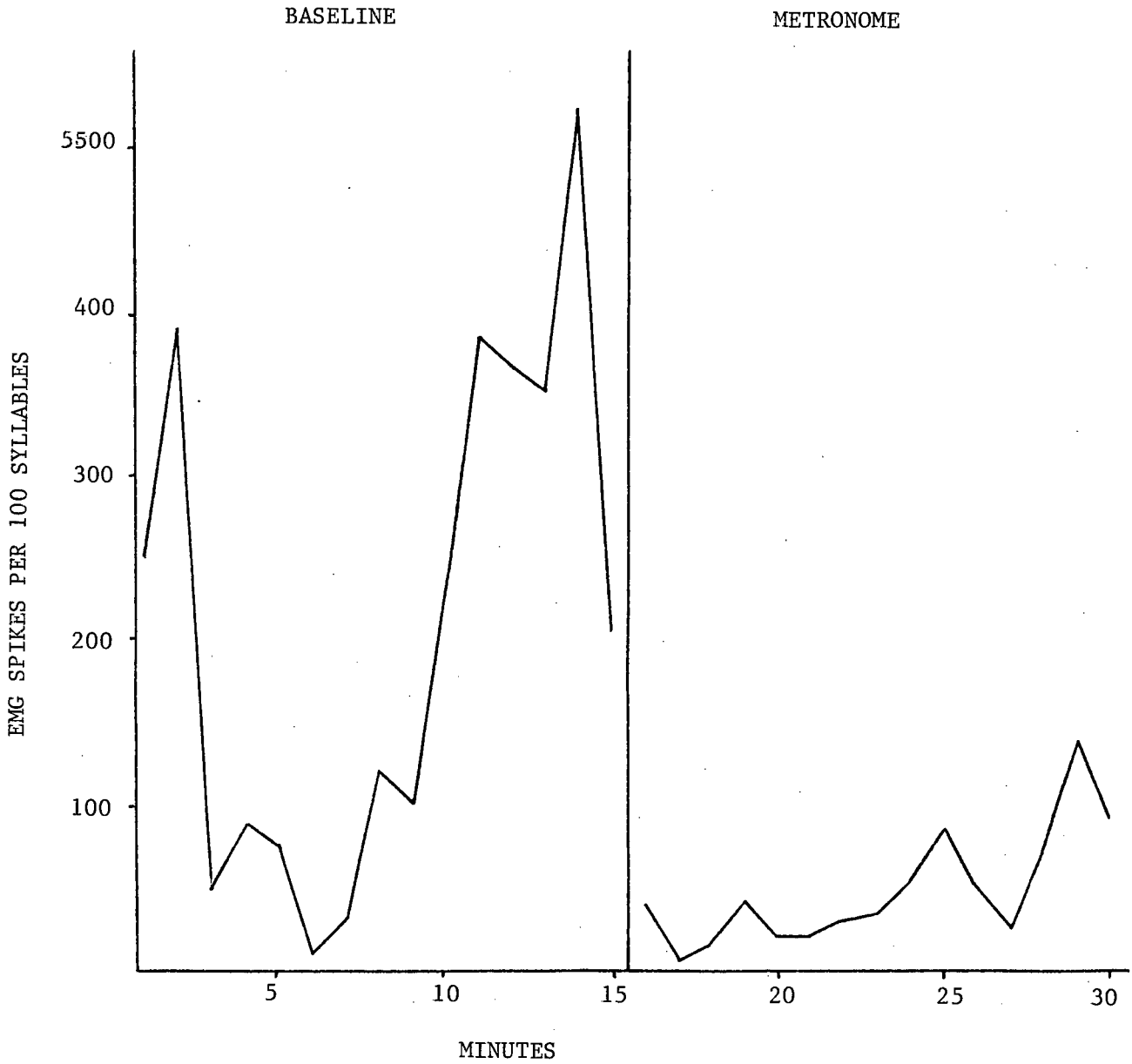


Figure 3. Effect of metronome on EMG spiking: David ($p < .005$).

Table 6

Suppression of Stuttering and EMG Spiking by Metronome Treatment

Subject	$t(27)$ for change in level: Percent syllables stuttered	$t(27)$ for change in level: EMG spikes per 100 syllables
David	5.93**	2.86**
Ruth	2.85**	2.18**
Don	.88	.79
Ray	2.59*	1.17

*$p < .05$

**$p < .005$

is highly variable within subjects. At any rate, the correlation between stuttering and EMG spiking is consistent with the laryngeal hypertension model.

Laryngeal hypertension is not necessarily a cause of stuttering, although this would make theoretical sense. It could just as well be an effect of the stress of being a stutterer. Correlational data is not sufficient to demonstrate causality; nevertheless, the degree of concomitant variation I have found was sufficient to justify further research in this vein. The next step was an attempt to demonstrate the converse of what had been achieved here, viz., to reduce EMG spiking in the hope that stuttering would be concurrently reduced.

CHAPTER IV

FEEDBACK OF ELECTROMYOGRAPHIC ACTIVITY

At least three independent lines of research suggest that it may be possible to reduce stuttering with biofeedback. The first of these, which was the most influential in initiating the present series of studies, is simply that EMG feedback has already been applied successfully to a host of stress-related disorders (e.g., Raskin, Johnson & Rondestvedt, 1973). Additional impetus and direction was provided by the laryngeal hypertension model (Schwartz, 1974). Finally, the possibility that stuttering involves defective feedback (Mysak, 1960) implies that enhanced proprioceptive feedback might suppress stuttering.

It was anticipated that if biofeedback were ef[illegible] a greater (more direct) effect on [illegible]MG spiking than on stut[illegible]

Pilot for Study 3

Method

A single subject (John) participated in this study. EMG was recorded from the throat as described in Study 1, while the subject performed the TAT task. After a baseline period of nine minutes of speech, auditory feedback was presented through a speaker at the subject's side. The feedback consisted of a tone whose frequency varied in proportion to the amplitude of the EMG signal. The feedback apparatus is illustrated in Figure 4.

The subject was told that the feedback tone reflected tension in his speech muscles: the greater the tension, the higher the pitch of the tone. His task was to produce a low frequency tone as often as possible, just as

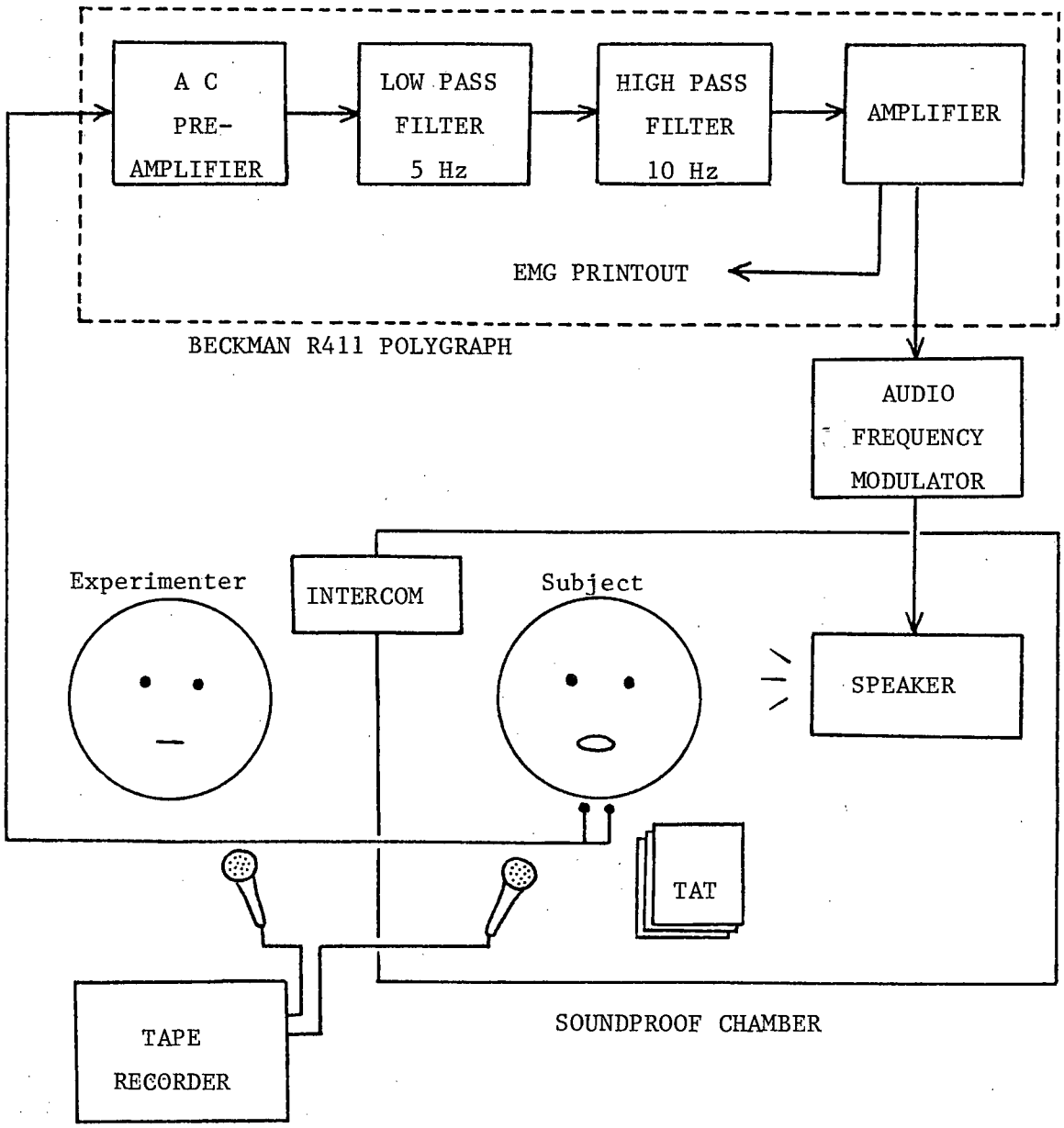


Figure 4. EMG feedback system.

one learns to control the amount of pressure exerted on an automobile accelerator by listening to the sound of the motor. The subject was not told how to relax his speech muscles; he was merely instructed to try to do so. Stuttering was not mentioned explicitly.

The biofeedback effect was demonstrated by having the subject swallow several times. He practiced tensing and relaxing his throat without speaking for about a minute and a half, until he was able to vary the pitch of the tone at will. Then the subject practiced saying his name, address, etc, several times, noting changes in the pitch of the tone as he blocked on these words. Within two and a half minutes, he was able to chat without stuttering severely and without greatly increasing the frequency of the tone. After this brief practice period the subject resumed the TAT task as before, with the tone on. He was told that the feedback would be turned on and off intermittently while he spoke. A complete on-off cycle comprised six cards (18 minutes). The session was concluded after the subject had completed speaking about the fifteenth card.

Results and Discussion

Stuttering data are illustrated in Figure 5, and statistical analyses for all three dependent variables are summarized in Table 7. The first comparison, viz., the first baseline vs. the first feedback period, is atypical in that a four minute interval occurred between these conditions. Fortunately, the risk of historical confounding during such a brief interval is slight. The second comparison must also be interpreted cautiously because of the possibility of carryover of the biofeedback effect from the first feedback period into the second baseline period. If this occurred it would tend to

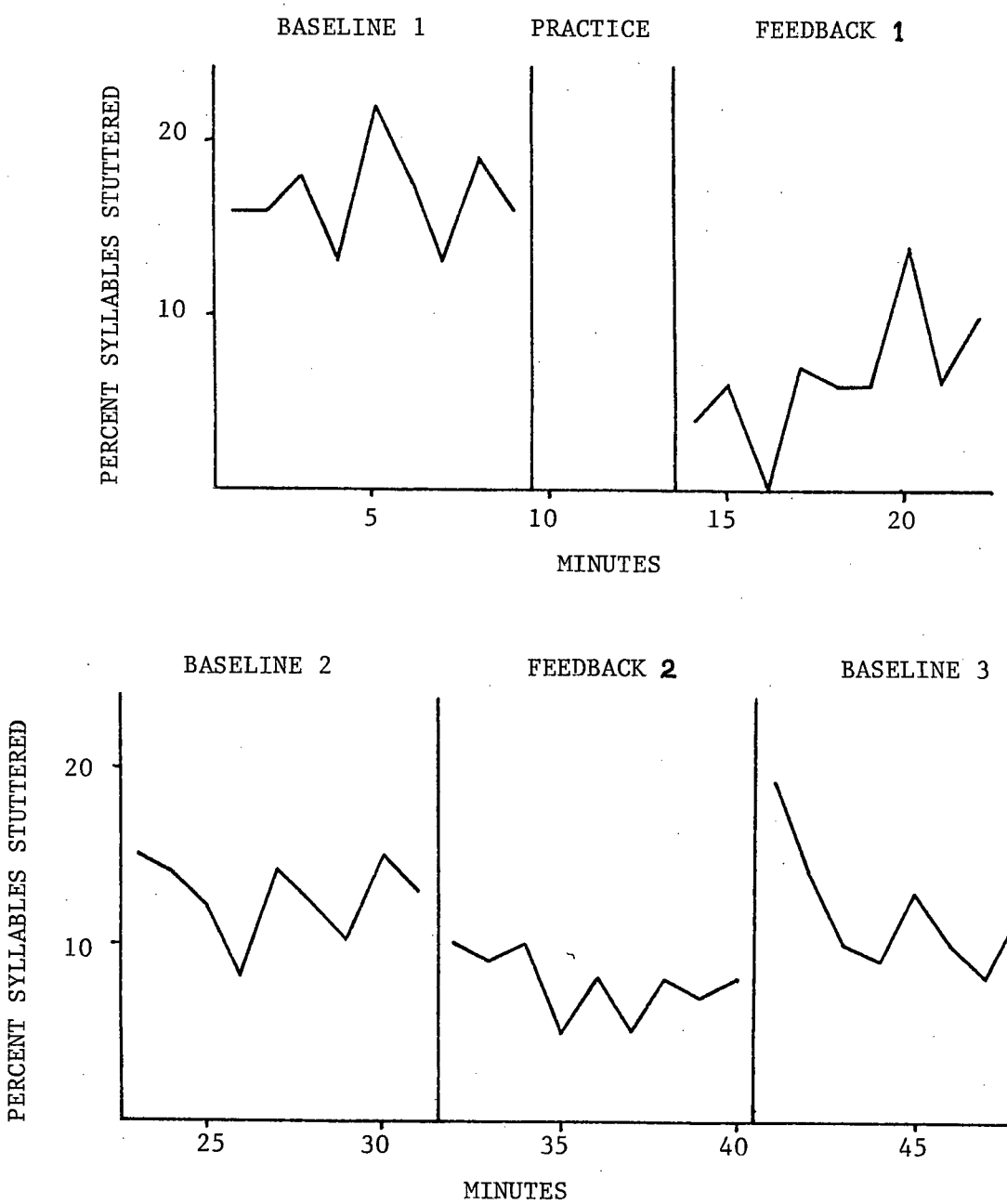


Figure 5. Effect of EMG feedback on stuttering: John (From Hanna et al., 1975).

Table 7

Effects of EMG Feedback: Pilot Study

Comparison	$t(15)$ for change in level: Percent syllables stuttered	EMG spikes per 100 syllables	Speech rate (SPM)
Baseline 1 vs. Feedback 1	4.66**	2.31*	1.86*
Baseline 2 vs. Feedback 2	1.80*	1.14	.43

* $p < .05$

** $p < .0005$

obscure the reversal effect, and thus the time series statistic may be conservative. In spite of these qualifications, it appears that the feedback treatment was accompanied by a significant decrease in stuttering both times. EMG spiking was significantly reduced the first time, but not the second. The subject's speech rate appeared to increase the first time, but did not increase during the second application of feedback. It can be concluded tentatively that the feedback effect was not dependent on slowing of speech.

Other time series comparisons of lesser interest, e.g., the second feedback period vs. the third baseline, were not analyzed statistically because of the problems inherent in using the *t* distribution to make multiple comparisons among means.

Study 3

The results of the pilot study were sufficiently encouraging that an attempt was made to replicate these findings several times. The reversal design used in the pilot study was abandoned because of the difficulty of interpreting carryover effects. The practice interval between the baseline and feedback periods was also eliminated. Each period was lengthened to 15 minutes in order to increase the power of the time series statistic.

Method

The general procedure was similar to that used in the pilot study. The subject (Don) was told that after a baseline period of 15 minutes of speech, an auditory feedback tone would be presented through a speaker at his side. Again, the feedback tone increased in frequency in proportion to the amplitude of the EMG signal. This was accomplished by using the ampli-

fied EMG signal to power a tone generator. Below a certain amplitude, the tone shut off entirely. It was explained to the subject that the tone reflected tension in his speech muscles: the greater the tension, the higher the pitch of the tone. The subject's task was to try to turn the tone off, or at least to produce a low-pitched tone, as often as possible. These instructions had to be given at the beginning of the experiment, rather than at the end of the baseline period, because the time-series design did not permit an interruption between baseline and treatment periods. (Verbatim instructions are given in Appendix D.) Laryngeal EMG was recorded as in the previous studies while the subject performed the TAT task. The study took 30 minutes: 15 minutes of baseline speech, followed immediately by a 15 minute period during which the feedback was in operation.

This study was replicated with four other subjects.

Results

Data for the first subject (Don) are illustrated graphically in Figures 6 and 7. Biofeedback resulted in a significant reduction in stuttering, but not in EMG spiking, for this subject. The covariation in stuttering and spiking is noteworthy nevertheless. Statistical analyses of the stuttering and spiking data for all five subjects are summarized in Table 8. Ruth's stuttering data (Figure 8) illustrate the value of testing the significance of time series data. The treatment effect appears highly significant because there was no stuttering at all in the biofeedback period. However, closer inspection reveals that there was a high degree of spontaneous fluctuation in the baseline data, and that seven of the 15 baseline observations were zero. In short, biofeedback did not reduce Ruth's stuttering

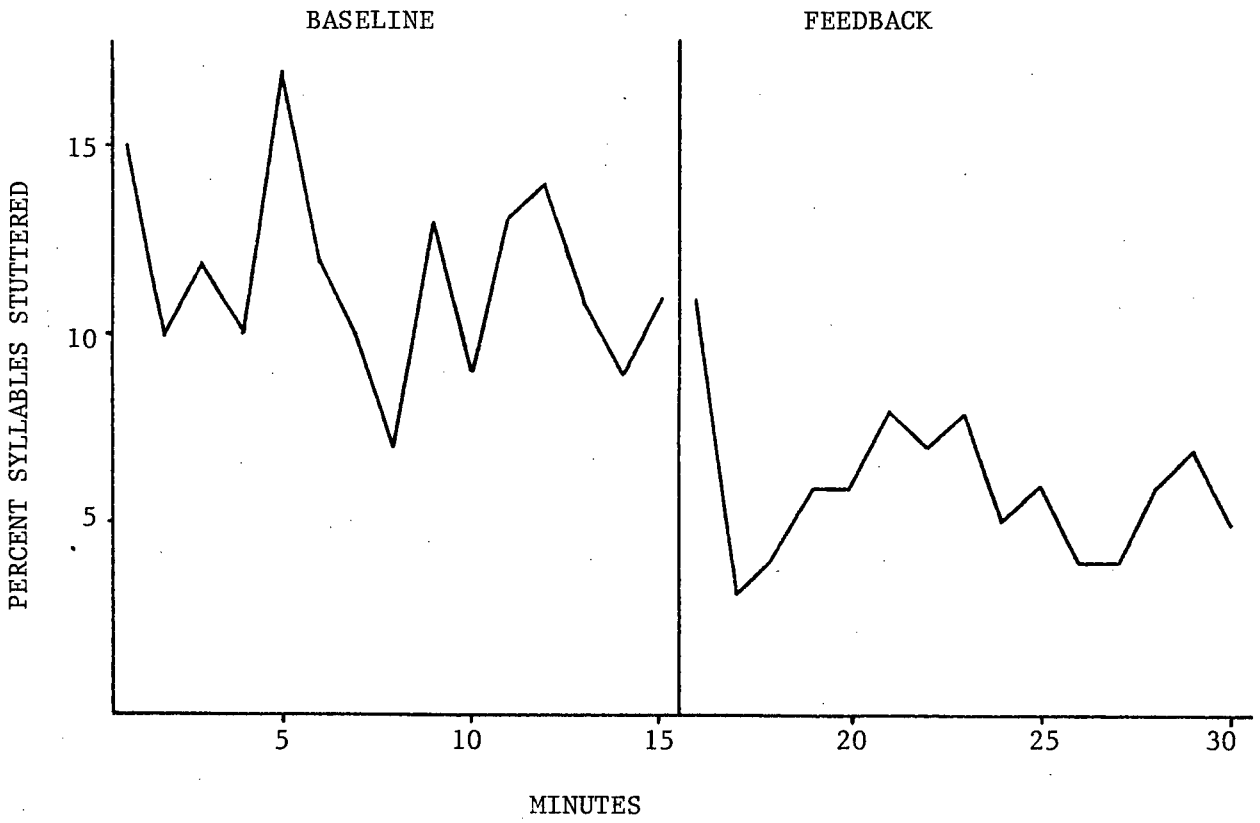


Figure 6. Effect of EMG feedback on stuttering: Don ($p < .05$).

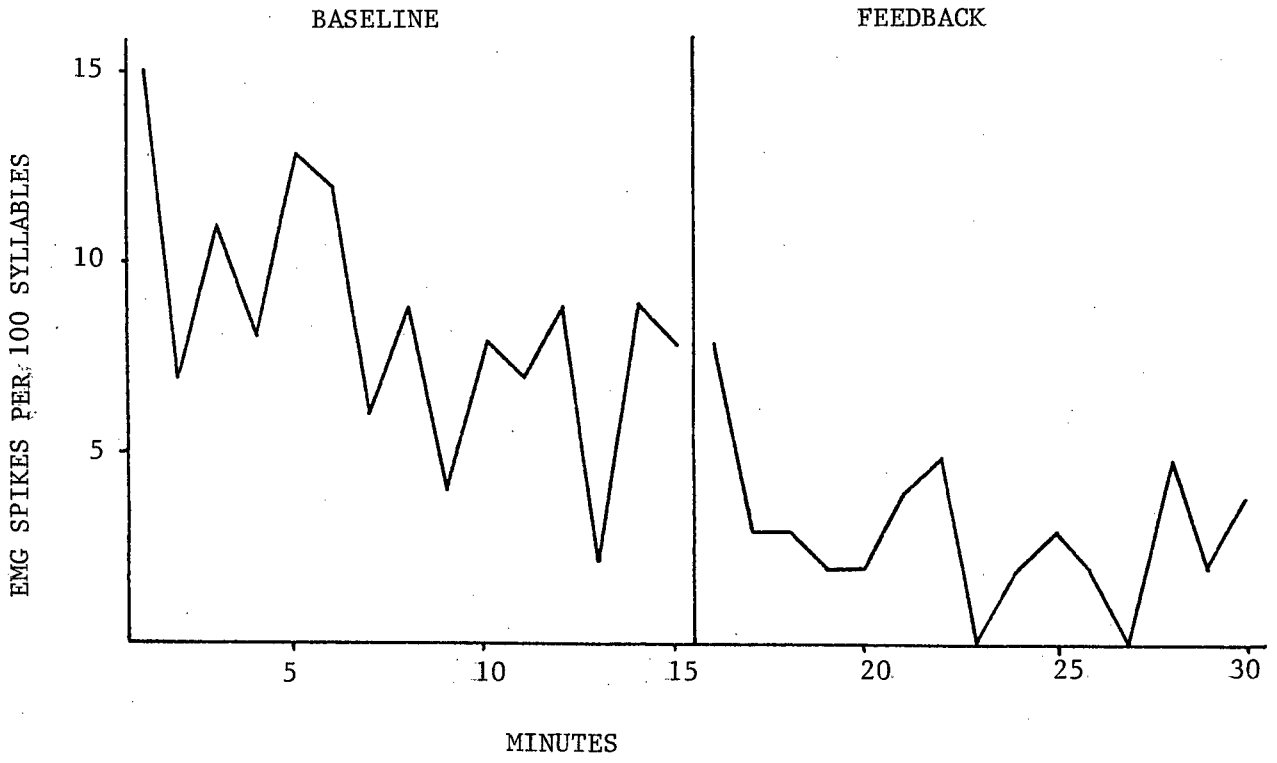


Figure 7. Effect of feedback on EMG spiking: Don (Not significant).

Table 8

Effect of EMG Feedback on Stuttering and Spiking

Subject	$t(27)$ for change in level	
	Percent syllables stuttered	EMG spikes per 100 syllables
Don	2.24*	.91
David	2.61*	2.97**
Ray	.86	.30
Ruth	.63	3.37**
Larry	1.27	6.32**

*$p < .05$

**$p < .005$

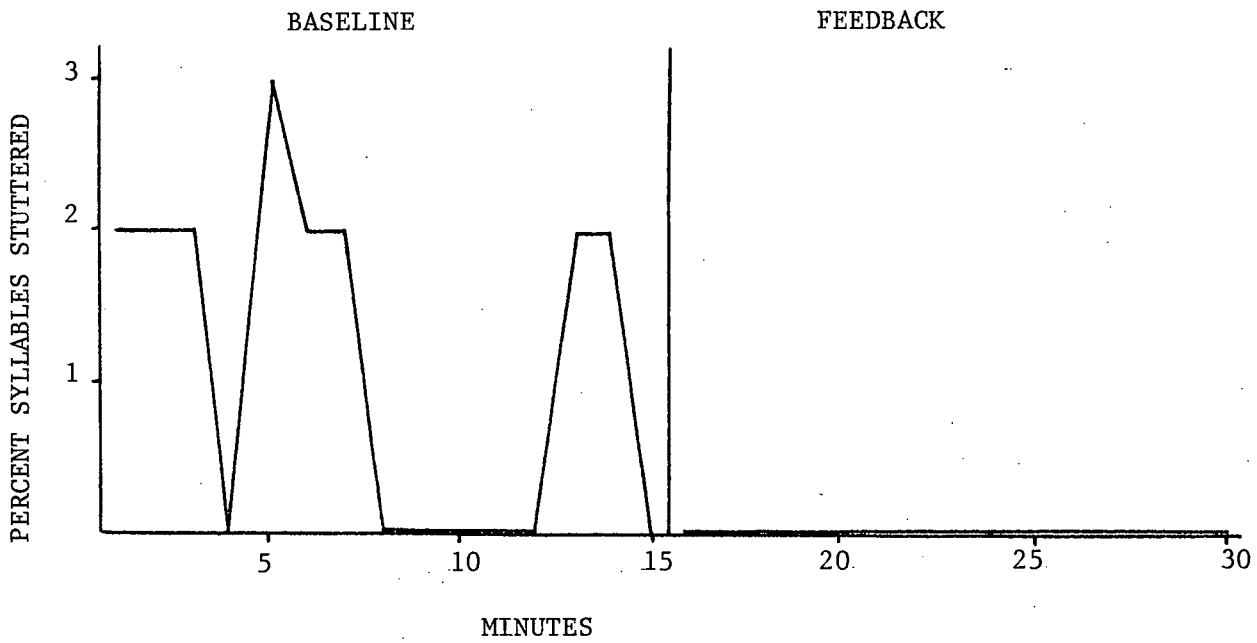


Figure 8. Effect of EMG feedback on stuttering: Ruth (Not significant).

significantly.

Nevertheless, stuttering was significantly reduced by biofeedback in two out of five cases. Spiking was reduced in three cases.

Speech rate data are given in Table 9. One of the five subjects showed a significant reduction in speech rate during the feedback condition.

Discussion

The biofeedback effect appears to be a promising one: four out of six subjects (including the pilot subject) achieved reductions in spiking in a single session and three achieved reductions in stuttering. This effect is particularly surprising in view of the fact that all six subjects were chronic stutterers who had been treated unsuccessfully in the past. Moreover, the last five subjects did not have the advantage of a practice interval between the baseline and feedback periods, yet three of them showed an abrupt treatment effect. This learning effect was achieved without reinforcement other than knowledge of the results.

Only one subject appeared to slow his speech during the feedback treatment. This may have been a response to the novelty of the feedback task. It can be concluded tentatively that the feedback effect was not an artifact of rate control.

Further replications of the feedback effect may be found in Study 5.

Table 9

Effect of EMG Feedback on Speech Rate

Subject	$t(27)$ for change in level Speech rate (SPM)
Don	1.09
David	1.80*
Ray	.39
Ruth	.73
Larry	.66

*$p < .05$

CHAPTER V

FEEDBACK WITHOUT INSTRUCTIONS

Study 4

It appears that feedback training can be used effectively to modify a given physiological response, even when the subject has not been informed which response he is supposed to modify. Beatty (1972) reported that merely instructing his subjects to increase the loudness of a tone (which was related to EEG activity) resulted in significant changes in the proportion of alpha and beta activity, although the subjects were not informed that EEG was the dependent variable of interest. Ascough and Sipprelle (1968) and others have reported similar results with heart rate conditioning. It would be of relatively little interest to replicate this "feedback without information" effect in the present series of studies. However, a related and unexplored problem concerns the effect of instructions (to reduce the pitch of the tone) as distinct from information (that the tone is related to muscle tension). Instructions are presumably a necessary component of the feedback effect reported in Study 3, if it is indeed authentic. Feedback without instructions should not be accompanied by a reduction in stuttering and EMG spiking. If it were, we should have to conclude that the "biofeedback" effect was an artifact of the presentation of the tone itself, rather than being due to enhanced information about laryngeal muscle activity.

It has been reported that stuttering can be reduced by presentation of a loud aversive tone (Barr & Carmel, 1969; Biggs & Sheehan, 1969; Flanagan, Goldiamond & Azrin, 1958). However, the fact that this effect occurs whether

the tone is presented at random or contingent upon stuttering suggests that it may be an artifact of masking. Masking is not a feasible explanation of the present biofeedback effect, because the EMG feedback tone is too high-pitched and insufficiently loud to mask the frequencies of speech.

Method

The subject (Rick) performed the TAT task for 30 minutes while EMG and stuttering data were recorded as usual. At the end of the 15 minute baseline period, I announced: "From now on you will hear an intermittent high-pitched tone while you are speaking. Continue talking about the pictures as before." When 30 minutes of speech had been recorded, the subject was questioned to see if he had perceived the function of the tone (see Appendix E). Finally the subject was debriefed about the true purpose of the experiment.

This procedure was repeated in two more single-subject studies. (The subjects were Frank and Ruth. One week later, Ruth participated in Study 3). Three other subjects (Teresa, Doug, Ron) received the feedback-without-instructions treatment first, followed by a 15 minute period without feedback. (This precaution against the adaptation effect may have been unnecessary, in view of the fact that the time-series statistic does attempt to accomodate trends in the data.

Results

Stuttering and spiking data for the first subject (Rick) are given in Figures 9 and 10 respectively. Data for all six subjects are summarized in Table 10. Results were significant for two subjects: Frank showed an increase in stuttering and spiking during the feedback condition and Teresa

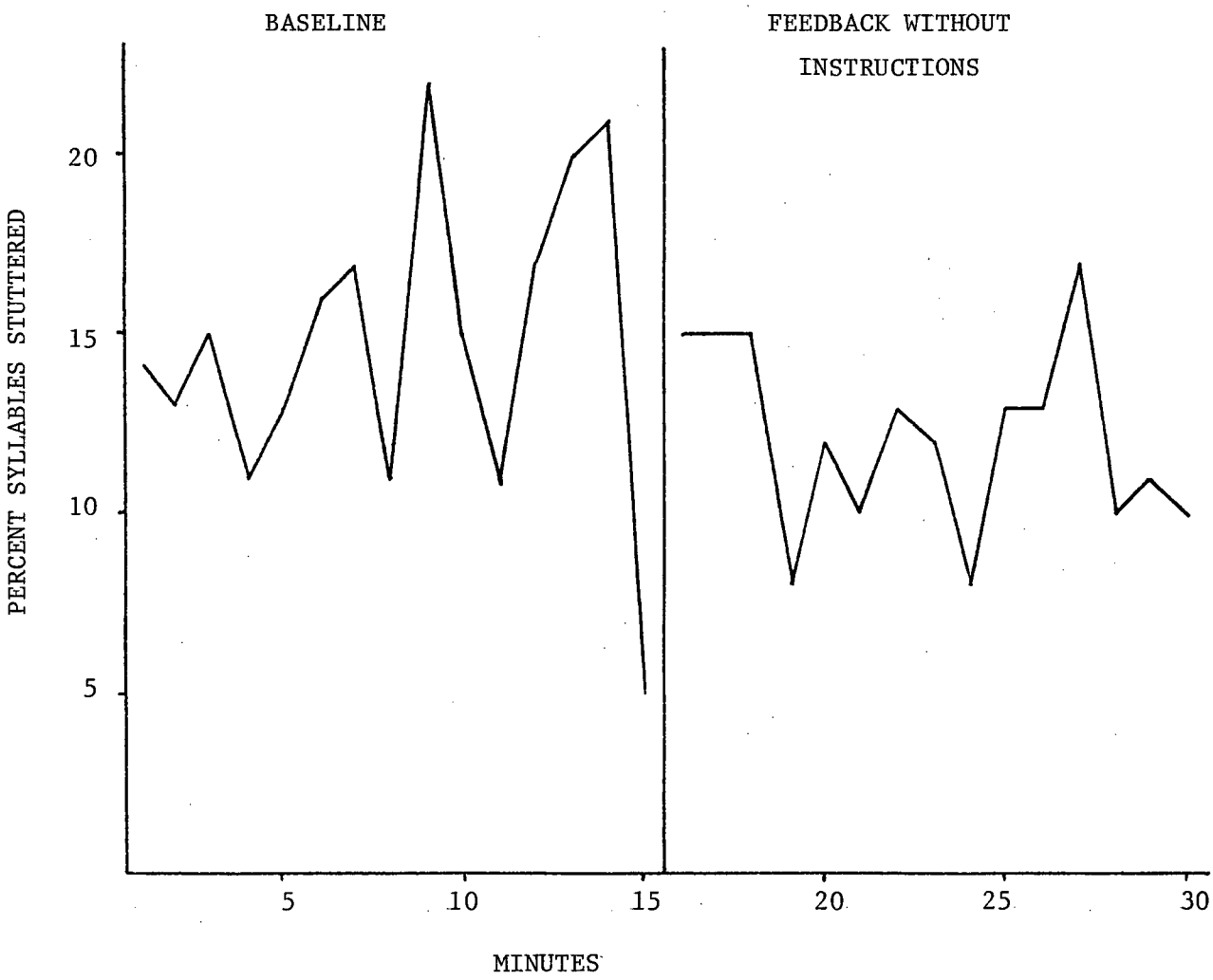


Figure 9. Effect of feedback without instructions on stuttering: Rick (Not significant).

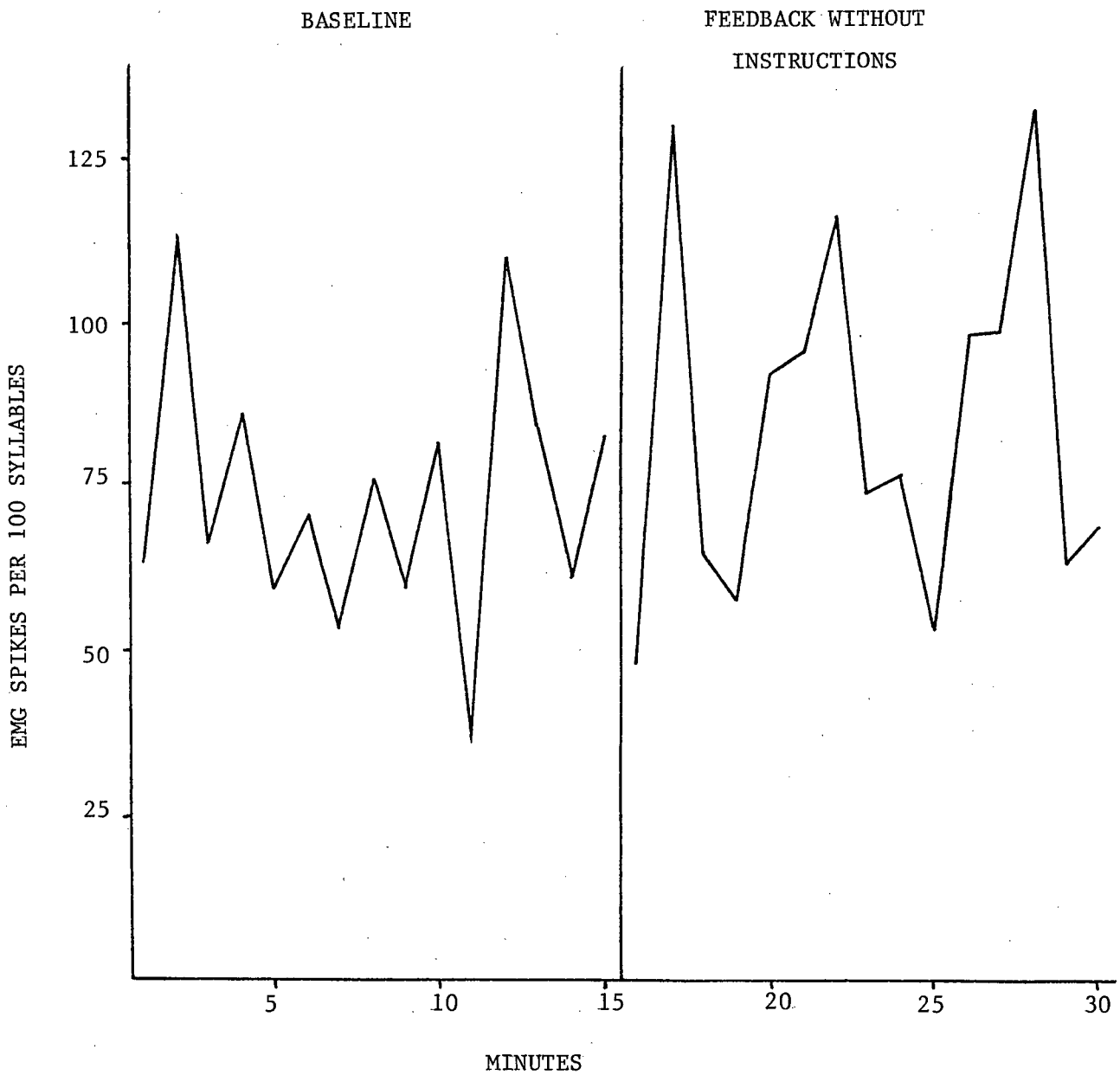


Figure 10. Effect of feedback without instructions on EMG spiking: Rick (Not significant).

Table 10

Effect of Feedback Without Instructions on Stuttering and EMG Spiking

Subject	$t(27)$ for change in level: Percent syllables stuttered	$t(27)$ for change in level: EMG spikes per 100 syllables
Rick	.83	.45
Frank	1.78*	2.01*
Ruth	.06	.52
Teresa	1.23	1.83*
Doug	.66	.19
Ron	.66	.88

*$p < .05$

showed an increase in spiking. The insignificant results for the other five subjects were in both directions: there were two increases and three decreases in stuttering during the feedback condition, and three increases and one decrease in spiking.

Speech rate data are given in Table 11. Feedback without instructions produced no significant change in speech rate for any of the six subjects.

None of the subjects were aware that the tone reflected laryngeal muscle tension. One subject (Ruth) commented, "It squeaks when I swallow." None of the subjects reported that the tone made any difference to their speech.

Discussion

Instructions appear to be a necessary component of the feedback effect reported in Study 3. Feedback without instructions did not produce a reduction in spiking and stuttering, in fact, it apparently tended to increase them. This may have been due to the alarming nature of the tone per se, or to the fact that its function was not explained. If the former hypothesis is correct, feedback by means of a visual signal might be less alarming and more effective than auditory feedback. At any rate, the feedback effect appears not to be an artifact of masking or of the presentation of a loud, aversive tone.

The findings that feedback without instructions produced no significant changes in speech rate, regardless of its effect on stuttering and spiking, supports a similar finding in Study 3.

We have concluded that instructions are necessary. The final study in this series addresses itself to the question: "Are instructions sufficient?"

Table 11

Effect of Feedback Without Instructions on Speech Rate

Subject	$t(27)$ for change in level Speech rate (SPM)
Rick	.47
Frank	1.19
Ruth	.25
Teresa	.59
Doug	.64
Ron	.52

CHAPTER VI

FALSE FEEDBACK

The relative ease with which stuttering can be manipulated in the laboratory has given rise to numerous false leads in the search for an effective treatment (Van Riper, 1973). Martin and Siegel (1966) demonstrated that merely instructing their subjects to "Read more carefully" and to "say each word fluently" resulted in a reduction in stuttering. Likewise, simply calling a stutterer's attention to his disfluency will sometimes decrease it temporarily (Wingate, 1959). Thus the effect of instructions needed to be investigated in the present series of experiments.

Likewise, the placebo effect had to be controlled. The initial "success" of several questionable treatments used in the past suggests that the attention given and hope aroused during any novel treatment for stuttering may well have an effect on fluency. A related problem concerns the Hawthorne effect, i.e., the possibility that changes in stuttering and/or spiking may be due to the subject's awareness of the experimentation rather than to the specific nature of the treatment. The aim of the present research was to establish a potentially permanent psychophysiological effect rather than a transient, purely psychological effect. The stutterer's fund of hope is too small to be wasted.

Instruction, placebo and Hawthorne effects could all be ruled out if pseudofeedback were found to be ineffective.

Pilot for Study 5

Method

The subject in this study (John) had had previous experience in feedback training, i.e., he had participated in the pilot for Study 3 a week earlier. Stuttering and spiking data were recorded as usual while the subject performed the TAT task. After a nine minute baseline and a brief practice period, genuine feedback was given for nine minutes. This was followed by a second nine minute baseline, then nine minutes of false feedback and finally nine more minutes of genuine feedback. False feedback was accomplished simply by recording EMG from another subject in an adjacent room and presenting this person's feedback to John, ostensibly as his own.

Results and Discussion

Statistical analyses for all three dependent variables are summarized in Table 12. Changes in the frequency of John's stuttering are illustrated in Figure 11. It appears that stuttering was reduced to about 50% of baseline during the first feedback period. Spiking was concurrently reduced. False feedback reduced stuttering and spiking slightly, but not significantly. This slight reduction may have been due to the fact that the subject, who had already had some exposure to feedback training in the pilot for Study 3, was essentially receiving random signals to relax his throat muscles during the false feedback condition. Moreover, he kept complaining during the latter period that "this thing isn't adjusted right", which suggests that he had indeed acquired some degree of awareness of his laryngeal muscle activity.

The subject appeared to increase his speech rate during the first genuine feedback period, but not during the false feedback period. This is

Table 12

Effects of Genuine and False Feedback: Pilot Study

Comparison	$t(15)$ for change in level: Percent syllables stuttered	EMG spikes per 100 syllables	Speech rate (SPM)
Baseline 1 vs. Feedback 1	2.86**	1.91*	3.96*
Baseline 2 vs. False Feedback	1.30	1.59	1.04

* $p \leq .05$

** $p \leq .01$

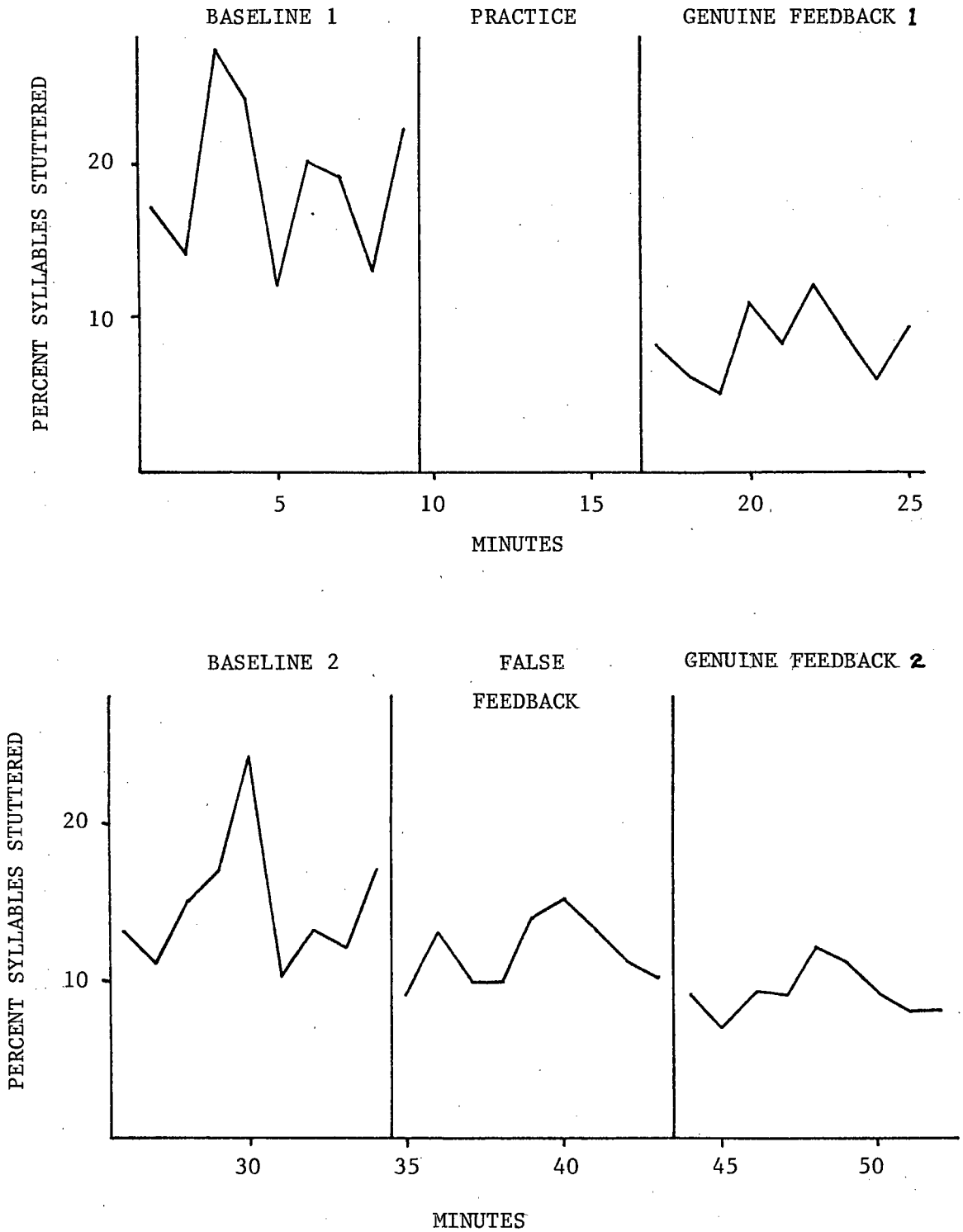


Figure 11. Effect of genuine and false feedback on stuttering: John (From Hanna et al., 1975).

consistent with earlier findings that feedback-induced fluency is not accompanied by a reduction in speech rate.

These conclusions are subject to the qualifications discussed in the pilot for Study 3.

Study 5

The results of the pilot study were encouraging but inconclusive, so I attempted to replicate these findings several times, with methodological refinements.

Method

EMG and stuttering were recorded as usual while the subject (Eric) performed the TAT task. A 15 minute baseline was followed immediately by 15 minutes of genuine feedback. (No practice interval occurred between these periods, as in the pilot study.) Following a 10 minute break, there was a second baseline period followed by 15 minutes of false feedback. The 10 minute break was given in order to reduce the possibility of multiple treatment interference, i.e., carryover of fluency from the genuine feedback session into the false feedback session. The subject received exactly the same instructions for false feedback as for genuine feedback (Appendix D).

False feedback was achieved by presenting each subject with feedback of his own EMG activity, but with a delay of 30 seconds. This was readily accomplished manually: I simply copied each subject's EMG record on a second channel of the polygraph, at a lag of 30 seconds, by manipulating the "centre" control. Deflections of the pen on this artificial EMG channel powered the tone generator. The fidelity with which the false EMG record resembled the

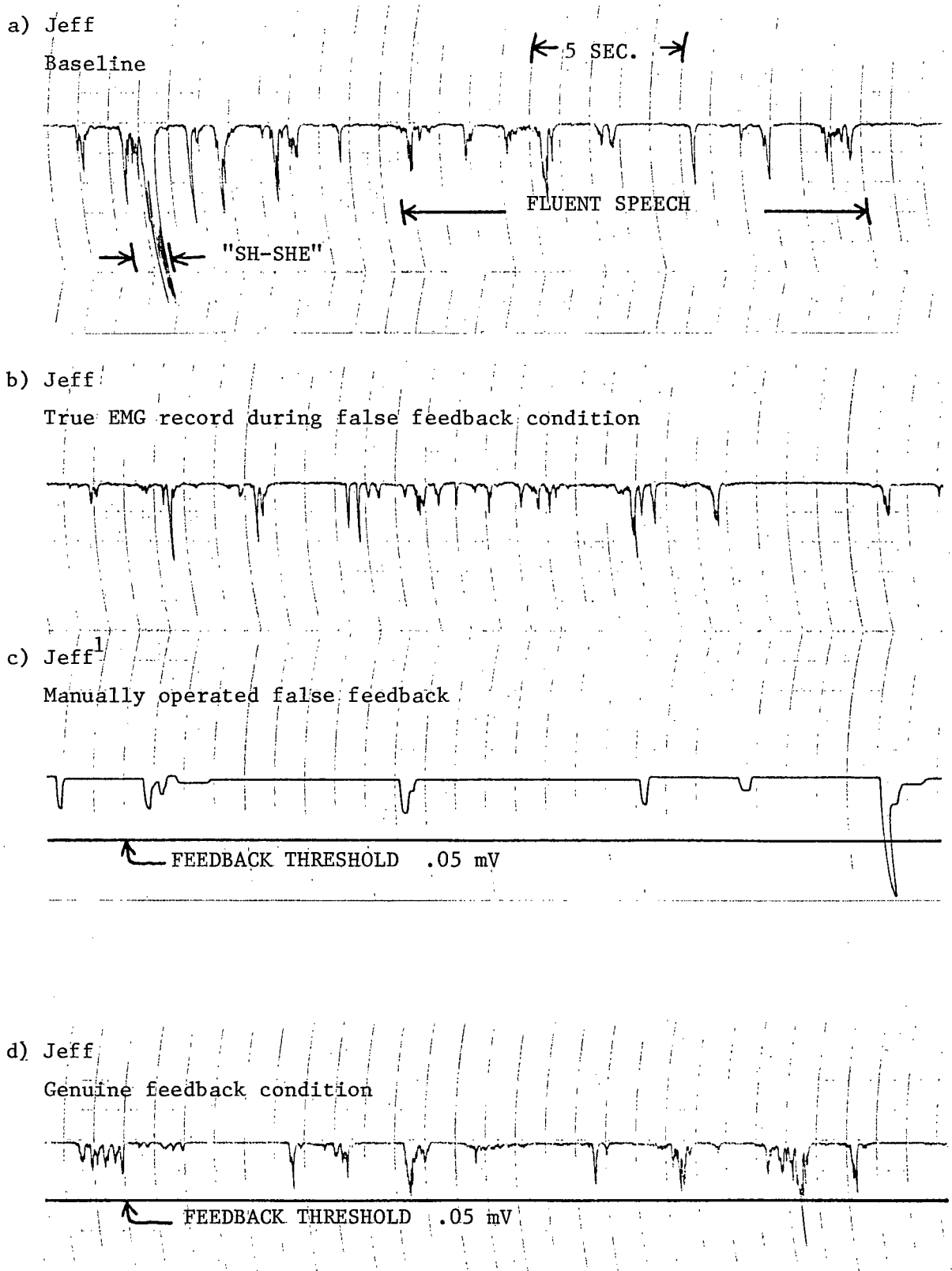
genuine record is illustrated in Figure 12. The auditory similarity was even more striking. While listening to tapes of this experiment, I found it difficult to differentiate between genuine and false feedback conditions on the basis of sound alone. This type of false feedback was superior to that used in the pilot study in that it resembled genuine feedback more closely, and did not require a confederate subject.

The subject was questioned after this study in order to ascertain his degree of awareness of the experimental manipulation (Appendix E), and then was debriefed about the true nature of the experiment. The procedure was repeated in the same order for two other subjects (Teresa and Doug), and then replicated three more times (Jeff, Ron and Susan) in counterbalanced order, i.e., false feedback preceded genuine feedback.

Results

The effects of genuine and false feedback were tested for significance against their respective baselines rather than against each other, because of the possibility of historical invalidity and multiple-treatment interference. The 10 minute break at half time appears to have been sufficient to allow stuttering and spiking to return approximately to baseline (e.g., Figure 13). Doug reduced both this stuttering and spiking with genuine feedback (Table 13). The reduction in Eric's EMG spiking during genuine feedback fell short of significance: $t(28) = 1.64$, $p < .06$. Genuine feedback was sometimes effective when it preceded false feedback, but never when it followed false feedback. False feedback was remarkably ineffective: it reduced spiking only once in six cases, and did not reduce stuttering at all.

Figure 12. Sample EMG recordings from Study 5.



1. In this illustration the feedback tone was activated only once, i.e., when the pen deflection exceeded the threshold.

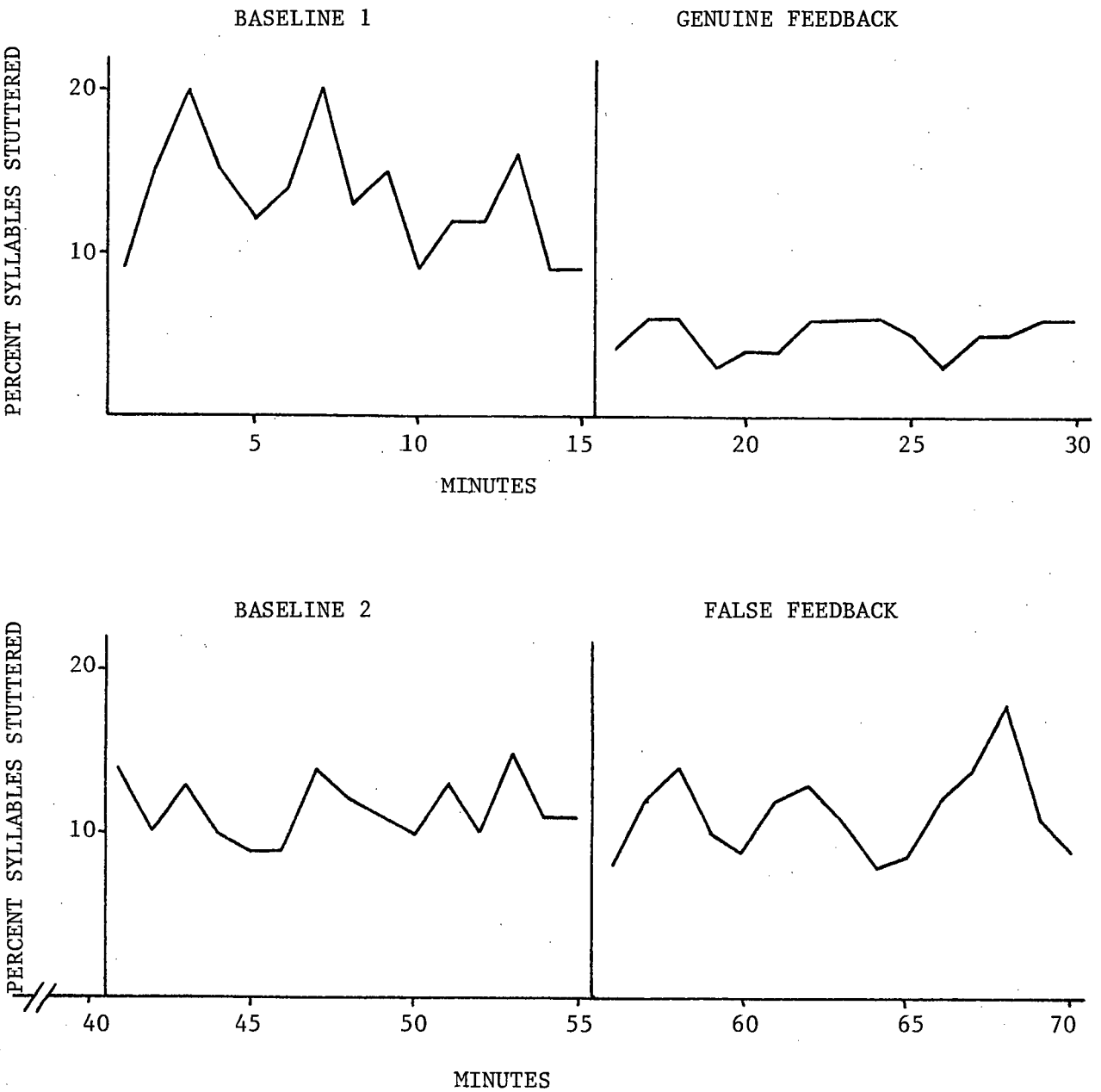


Figure 13. Effect of genuine and false feedback on stuttering: Doug ($p \leq .005$ for genuine feedback; false feedback not significant).

Table 13

Effect of Genuine and False Feedback on

Stuttering and EMG Spiking

Subject	Order of treatments	t(27) for change in level: Percent syllables stuttered	t(27) for change in level: EMG spikes per 100 syllables
Eric	genuine	1.21	1.64
	false	1.05	.77
Teresa	genuine	.04	.45
	false	.06	.04
Doug	genuine	3.32**	1.75*
	false	.70	1.35
Jeff	false	1.26	2.27*
	genuine	.95	.88
Ron	false	.39	.20
	genuine	.93	.77
Susan	false	1.31	.39
	genuine	.70	.32

* $p < .05$

** $p < .005$

Speech rate data are given in Table 14. Only two of the 12 comparisons were significant: Eric's speech rate decreased under false feedback, while Ron's increased under genuine feedback. Generally, neither type of feedback had a consistent effect on speech rate.

Two subjects (Doug and Eric) detected a difference between genuine and false feedback. Doug stated, "Even when I was relaxed the second time, that thing still squeaked ... I couldn't control it as well the second time." Eric claimed that it was equally difficult to control the genuine and false feedback tones. The other four subjects reported that the tones made no difference to their speech, which was generally true. Susan claimed that she was able to control the tone, which was not true. The subject who was able to control the genuine feedback tone successfully was unable to articulate how he did so, except in vague terms, e.g., "I just concentrated on it and tried to relax" (Doug).

Discussion

This study (and its pilot) demonstrated that a brief period of genuine feedback training was sometimes effective in reducing stuttering and EMG spiking. And if any more evidence were needed, the feedback effect appeared independent of speech rate. These conclusions are consistent with those of Study 3.

False feedback was generally ineffective. Moreover, genuine feedback was ineffective in the three cases in which it was preceded by false feedback. This suggests that false feedback may have interfered with subsequent genuine feedback training, because of negative transfer. Unfortunately, the data are not sufficiently strong to support this line of speculation.

Table 14

Effect of Genuine and False Feedback on Speech Rate

Subject	Speech rate (SPM) under genuine feedback	Speech rate (SPM) under false feedback
	$t(27)$ for change in level	
Eric	.29	2.87**
Teresa	1.29	.55
Doug	.35	1.53
Jeff	.30	.63
Ron	1.83*	1.19
Susan	.04	.03

*$p < .05$

**$p < .005$

The false feedback control condition allows us to discount the possibility that any changes in stuttering or spiking were due to concentration on the task of controlling the tone, or to distraction of the subject's attention away from his speech. Although distraction has often been invoked as an "explanation" of treatment effects in the stuttering literature, it has rarely been defined operationally. Moreover, Thompson (1971) demonstrated in a series of unpublished studies that distracting tasks do not reeuce stuttering significantly.

In summary, the false feedback control condition allows us to conclude that the biofeedback effect was not attributable to a placebo or Hawthorne effect, nor was it an artifact of instructions, masking distraction or rate control.

CHAPTER VII

CONCLUSION

In retrospect, it may be seen that there were several indications that feedback of EMG activity should have worked as well as it did. EMG feedback has already been applied effectively to several other stress-related disorders, e.g., insomnia, tension headaches and chronic anxiety. Moreover, one of the leading theories of stuttering suggests that stutterers suffer from defective feedback about their own speech, and thus enhanced feedback of laryngeal muscle activity would seem to be an appropriate remedy. Finally, the fledging model of stuttering as a disorder of excessive laryngeal muscle tension certainly implies that feedback-induced relaxation might be therapeutic.

The results of this series of studies support the laryngeal hypertension model in particular. EMG spiking from the throat, which differentiated between stuttering and fluency, was found to correlate positively with stuttering (Studies 1 and 2). Suppression of stuttering as a result of rate-control therapy and metronome-paced speech resulted in a parallel suppression of EMG spiking (Studies 1 and 2). The difference in stutterers' EMG activity before and after these treatments was striking; the post-treatment EMG records (e.g., Figure 1(d)) are indistinguishable from those of fluent speakers. Suppression of EMG spiking with biofeedback resulted in concurrent reductions in stuttering (Studies 3 and 5). These data certainly implicated the larynx as a locus of the stuttering block, although not necessarily the only locus: airflow blockage could also occur at the lips or at the front or back of the tongue (Van Riper, 1971).

The present results are also consistent with the feedback model of stuttering: several subjects did become more fluent when their somesthetic feedback was enhanced. Moreover, this effect can be interpreted within an operant framework, e.g., the proportion of low amplitude EMG signals was increased through negative reinforcement, viz., termination of an aversive auditory stimulus. The spontaneous comments of some subjects leave little doubt that the tone was aversive for them.

A total of twelve subjects received feedback training in Studies 3 and 5. Four subjects achieved significant reductions in stuttering, and five reduced their EMG spiking significantly. The feedback effect was also replicated twice within one subject, with positive results. These results may be conservative, because the time-series statistic is sensitive to immediate changes only, i.e., changes occuring in the first minutes of feedback training. Indeed, the strongest effect was achieved by the pilot subject, who was allowed to practice manipulating the feedback tone for a few minutes between baseline and feedback periods. Nevertheless, some subjects attained positive results rapidly without this advantage.

There are additional reasons why these results may be conservative. For one thing, the length of each EMG spike was not taken into account. In theory, a subject could have reduced the amplitude of his EMG spikes by 50% during the feedback treatment, yet this would not necessarily be reflected in the frequency of spikes which exceeded the feedback threshold. It would be a tedious task to measure the lengths of about ten thousand spikes, so this hypothesis has not been tested. However, visual inspection of the EMG records of some subjects does not discourage this line of speculation.

The strength of the feedback effect may also have been limited by the fact that the subjects were drawn from a waiting list for a therapy about which they had high expectations. This might have tended to limit their enthusiasm for the biofeedback treatment. Moreover, the subjects were all chronic and severe stutterers who had histories of unsuccessful speech therapy. Laryngeal hypertension during stuttering must be a very minute and grossly overlearned response, yet nearly half of the subjects were apparently able to make fine adjustments of their speech musculature on the basis of a mere 15 minutes of auditory feedback. This is indeed a promising trestment effect.

It is not immediately apparent why some subjects were able to master the biofeedback task while others were not. There are no well-established prognostic indicators in the biofeedback literature. Perhaps the most likely explanation is that the 15 minute feedback period was simply too brief for some subjects.

The EMG feedback effect appears to be an authentic one, rather than an artifact of some other condition that reduced stuttering. The importance of monitoring speech rate during any treatment for stuttering has been stressed by Perkins (1975). In Studies 3 through 4, 27 tests of significance for change in speech rate were performed. Two subjects were found to increase their speech rate, while two others decreased theirs. It can be concluded that feedback effect is not generally accompanied by, much less a result of, slowing of speech.

Information that the feedback tone is related to laryngeal muscle tension, plus instructions to reduce the pitch of the tone, appear to be necessary (Study 4), but not sufficient (Study 5). Thus the feedback effect

is not attributable to instructions alone, nor is it merely a case of reduction of stuttering by means of a contingent aversive tone. False feedback did not reduce stuttering for any of the seven subjects who received it, nor did it reduce EMG spiking in six out of seven cases. It can be concluded, then, that the feedback effect is not a placebo or Hawthorne effect, nor is it an artifact of distraction or masking (Study 5).

The relatively high degree of observer agreement on the dependent measures inspires some confidence in these results. Moreover, the statistical tests which support the above conclusions were not based on the small number of subjects in each study, but rather on the 30-60 data points in each time series. This is an example of the ways on which a small number of relatively rare subjects can be used efficiently. I disagree with the opinion of Johnson and Lubin (1972) that we need not use significance tests in single-subject psychophysiological experiments. The ease with which errors of inference could arise from visual inspection of the data is illustrated in Figures 7 and 8.

At this point, biofeedback can be said to have joined several other treatments which reduce stuttering. Similar laboratory effects have been demonstrated for masking noise (Cherry & Sayers, 1956), delayed auditory feedback (Goldiamond, 1965) and metronome-paced speech (Fransella & Beech, 1965). Unfortunately, none of the latter treatments have much carryover from laboratory to life, and thus they have not fulfilled their therapeutic promise.

The prospects for biofeedback as speech therapy appear more encouraging. Probably the feedback effect could be made stronger and more durable.

Shaping (i.e., varying the EMG amplitude at which the feedback tone is activated or becomes high-pitched) could be used to make feedback training easy at first, then progressively more challenging. As feedback was faded out, the stutterer would presumably be forced to rely increasingly on somesthetic cues. Manipulation of the volume and frequency range of the feedback tone might also enhance feedback training. Non-auditory modalities (e.g., a visual display of red, green, and amber lights) might be used instead of, or in conjunction with, the auditory tone. Indeed, there is a slight indication that the aversive feedback tone itself may have tended to disrupt the speech of some subjects (Study 4). Back-up reinforcers could be used to make the feedback task more rewarding. And of course, feedback of several parameters (e.g., GSR, respiration, heart rate) could be used to teach the stutterer to moderate his general arousal level. Biofeedback training could also be used in conjunction with other treatments for stuttering, e.g., rate control therapy, or with flooding or systematic desensitization to feared situational and phonemic cues. The laboratory situation could be made more realistic by having subjects converse spontaneously with each other, or with a videotaped speaker. EMG might prove useful in the diagnosis of incipient stuttering, if EMG spiking were found to discriminate true stuttering from normal childhood disfluency. Young stutterers could perhaps be given preventitive feedback training.

Now that a treatment effect has been established, the next step in this line of research might involve a comparison of the effectiveness of biofeedback against the best existing therapy, viz., rate-control therapy. If a sufficient number of matched subjects could be found, a group design

would be appropriate for this purpose. Otherwise, time-series analysis could be used profitably, e.g., each data point might represent the mean frequency of stuttering during a one-hour weekly treatment session. Subjects could be followed up covertly after the therapy had been completed, e.g., they could be telephoned on some plausible pretext by an accomplice of the experimenter. The use of nonreactive measures and naturalistic observation in the evaluation of stuttering therapy has been advocated by Andrews and Ingham (1972).

It might be possible to construct a miniature EMG feedback unit (consisting of a pocket-sized amplifier plus electrodes and an earplug) which could be worn as a prosthetic. Similar devices are already being developed. Brady (1972) has patented a miniature metronome which can be worn like a hearing aid, but unfortunately it imparts a staccato quality to the wearer's speech which is as conspicuous as a severe stutter. A portable masking noise generator is available (Trotter & Lesch, 1967) but it produces fluency in the wearer at the cost of temporarily deafening him, and thus is difficult to use in conversational situations. Portable delayed auditory feedback units have been constructed but are not suitable for clinical use at this time (Van Riper, 1973).

Bailey (Note 6), following the lead of Hanna et al. (1975), is presently testing a take-home feedback unit for stutterers. The advantage of a take-home feedback unit are obvious: the stutterer could learn control of his laryngeal muscles in his own time and at his own place, rather than during regular visits to the clinician's office. A take-home device could also be useful in a crisis situation when access to the clinic would be im-

possible. The cost and quality of portable EMG feedback equipment is becoming more attractive (Paskewitz, 1975). The success of such a device relies on the possibility that the biofeedback effect is not apparatus-bound, but will transfer to real life speaking situations.

Biofeedback as a treatment for stuttering deserves to be investigated in the context of an intensive treatment program emphasizing transfer and maintenance of fluency. Although this treatment requires relatively sophisticated instrumentation it is conceivable that after sufficient training, a stutterer could learn to attend to proprioceptive cues of laryngeal tension in the absence of biofeedback equipment. Indeed, this has been demonstrated in other biofeedback applications. Biofeedback may be the technique "... that will prevent or eliminate the core disruptions of the motor sequences that perhaps comprise the heart of the (stuttering) problem" (Van Riper, 1973, p. 203).

CHAPTER VIII

ADDENDUM

Guitar (Note 7) has independently performed a series of experiments which resemble Study 3. Guitar trained three stutterers to reduce their muscle action potentials (MAPs) at four sites:

1) over the obicularis oris superior (lip),

2) over the anterior belly of the digastric (chin),

3) just above the thyroid cartilege (larynx),

4) over the frontalis muscle (forehead).

EMG feedback consisted of a tone which increased in frequency in proportion to the voltage of the EMG signal. Guitar's and Hanna's EMG feedback were apparently very similar, because both were based on the electrode site and feedback apparatus used by Hardyck, Petrinovich and Ellsworth (1966). Following a baseline period, feedback training focused on one muscle group at a time. Shaping was used to facilitate feedback training. Results are presented in Table 15. Although significance levels are not given, all three subjects appeared to show substantial improvement with feedback from the laryngeal site. Guitar also found that subjects 1 and 3 showed improvement with feedback from the lip as well, and concluded that the stuttering block may not be localized at the larynx alone for all stutterers. The degree of change shown in Table 15 may be an overestimate because of the possibility of a strong adaptation effect during repeated reading of lists of stimulus words. Indeed, the decrease in stuttering during feedback from the frontalis site probably reflects some degree of adaptation, as well as general head relaxation.

Table 15

Changes in Muscle Action Potential (MAP) Levels and Stuttering Frequency (From Guitar, Note 7)

Subject	Site	Percent decrease in MAP level	Percent decrease in stuttering
1	frontalis	19	18
	lip	76	89
	chin	24	1
	larynx	51	44
2	frontalis	52	0
	lip	39	6
	chin	13	2
	larynx	56	25
3	frontalis	41	28
	lip	87	57
	chin	73	32
	larynx	37	55

Guitar claimed that two of his three subjects achieved "partial" and "considerable" generalization of fluency outside the laboratory, but presented no data to this effect. Subsequently, EMG feedback from the chin was used as therapy for a fourth subject. Stuttering was reduced to a negligible level after only three sessions of feedback during conversational speech. Five weeks later the subject reported that he was "satisfactorily fluent in all situations", and did not stutter during a videotaped conversation with a stranger. At a nine-month follow-up, the subject was reported to be speaking fluently at a normal rate.

The similarity between the results of Guitar's research and Study 3 is encouraging, as is Guitar's exploratory treatment study.

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APPENDIX A

Assignment of Subjects to Studies

Subject	1	2	3	4	5
Rick	X			X	
Frank	X			X	
John	X		X		X
David		X	X		
Don		X	X		
Ray		X	X		
Ruth		X	X	X	
Larry			X		
Teresa				X	X
Doug				X	X
Ron				X	X
Eric					X
Jeff					X
Susan					X
N =14	3	4	6	6	7

APPENDIX B

Dependent Variables: Inter-Rater Reliability and Percent Agreement

Study	Subject	Dependent variable: Percent syllables stuttered	Dependent variable: EMG spikes per 100 syllables	Dependent variable: Speech rate (SPM)
1	Frank	.87 81%	.90 92%	.94 96%
2	David	.84 76%	.92 90%	.90 94%
3	Ray	.89 85%	.94 93%	.97 98%
4	Ruth	.93 77%	.95 90%	.96 97%
5	Doug	.89 83%	.92 94%	.94 98%

APPENDIX C

Minimum Pen Deflections Defining an EMG Spike

Subject	Polygraph sensitivity (mV/cm)	Feedback activation threshold (mV)
David	.05	.025
Don	.10	.05
Ray	.05	.05
Ruth	.05	.025
John (pilot studies)	.10	.10
(Study 1)	.20	.20
Rick	.10	.05
Frank	.10	.05
Larry	.10	.05
Teresa	.05	.075
Doug	.05	.075
Ron	.05	.05
Eric	.05	.025
Jeff	.05	.05
Susan	.02	.02

APPENDIX D

Instructions for Studies 3 and 5

(Before baseline, after TAT instructions)

When you have spoken about the cards for 15 minutes, you will hear a high-pitched tone coming from the speaker at your side. The tone indicates the tension in your speech muscles. The higher the pitch of the tone, the more tense you are. When you are speaking in a relaxed way, the tone will shut off completely. Your job is to try to shut it off, or at least to keep the pitch of the tone as low as possible. I can't explain how you could do this, any more than I could tell you how to lift your arm. Just try anything you can to prevent the tone from coming on, or being high-pitched, while you speak about the cards. Do you understand?

(At the end of the baseline period)

The tone will be in operation from now on. Do anything you can to prevent the tone from coming on, or from being high-pitched, while you speak. Take a new card and continue speaking.

APPENDIX E

Questions Used to Investigate Awareness of Feedback Conditions

Study 4 (Feedback without instructions)

1. What do you think that tone was for?
2. Do you think the tone made any difference to your speech? (If so, what?)

Study 5 (False feedback)

1. Do you think the tone made any difference to your speech? (If so, what?)
2. Did you do anything to try to prevent the tone from coming on? (If so, what?)
3. Do you think you did any better atccontrolling the tone the first time it was on, or the second time?
4. Do you think the tone was the same each time?