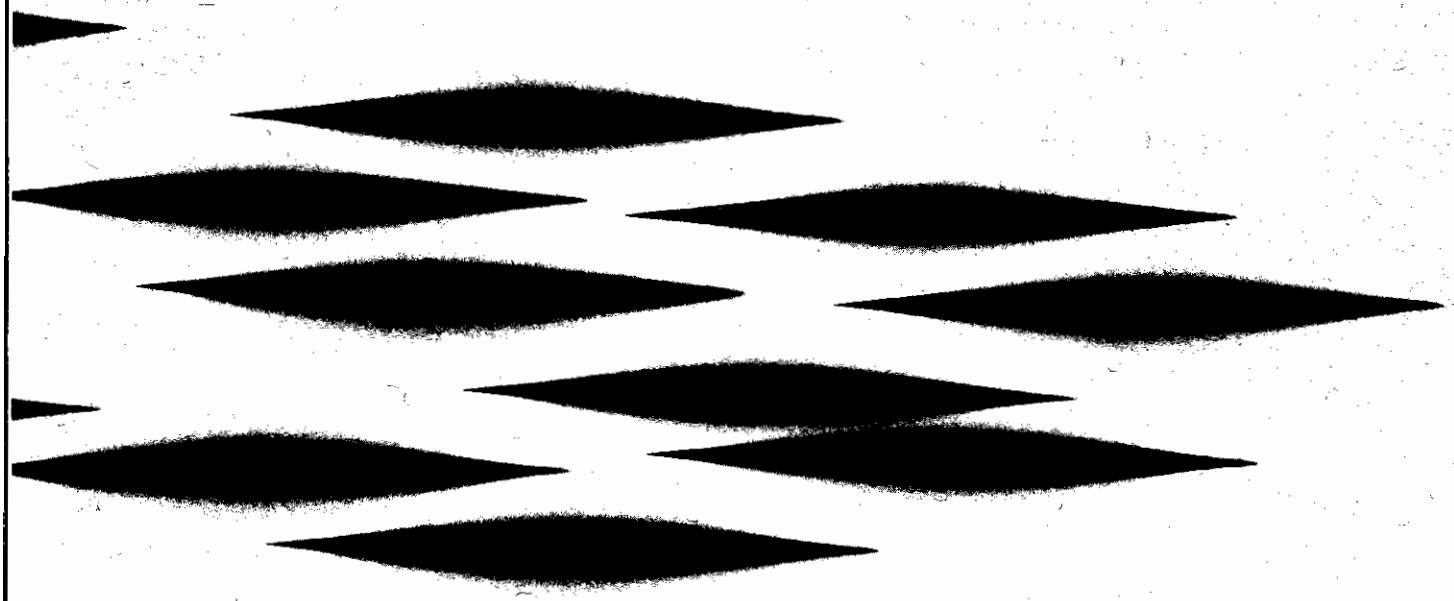
ISSN 0066-071X
ISBN 0-910329-01-X

STUTTERING AND LARYNGEAL BEHAVIOR: A REVIEW



ASHA MONOGRAPHS
NUMBER 21 A PUBLICATION OF THE AMERICAN SPEECH-LANGUAGE-HEARING ASSOCIATION

STUTTERING AND LARYNGEAL BEHAVIOR: A REVIEW

STUTTERING AND LARYNGEAL BEHAVIOR: A REVIEW

C. Woodruff Starkweather
Temple University
Philadelphia, Pennsylvania

ASHA Monographs Number 21 (ISSN 0066-071X)
(ISBN 0-910329-01X)

AMERICAN SPEECH-LANGUAGE-HEARING ASSOCIATION

Rockville, Maryland 20852

July 1982

Preface

One of the most challenging questions in speech-language pathology is: What causes stuttering to develop in some children, whereas others either acquire fluent language directly or pass through a brief period of disfluency? Compelling as it is, the question of what originally causes stuttering has remained unanswered despite vigorous efforts that began in the earliest years of the profession. The desire to solve this problem is possibly even stronger today than it was 40 years ago, and the strength of this desire has occasionally been so great that scientific caution has been forgotten and premature claims of solution have been announced. Typically, premature claims of having found the answer to this question are quickly recognized as unwarranted, but another error more often goes unnoticed. This more insidious error is the over-interpretation of research results, and it seems to occur when a series of results are published that support a particular hypothesis about what causes stuttering. One feels, from a review of the literature, that the quarry has been sighted, and we are all going to run it down. Minor obstacles, such as alternative explanations of results, can be brushed aside. More serious ones, like established facts that contradict the hypothesis, may be leaped over entirely. The force of this rush to find the answer is extremely compelling, and I do not lay claim to being immune to it. In fact, it was my realization that I was caught up in such a rush that led me to reexamination the research on stuttering and vocalization and ultimately to the writing of this monograph.

In the past 10 years, a number of articles have been written on the relation between stuttering and laryngeal behavior. The idea that this relation is special, or that by understanding it we may answer the question of what causes stuttering, is essentially attributable to the work of Wingate (1976). This idea is fresh and exciting, but it may nonetheless be incorrect. The purpose of this monograph is to look critically at the experiments, reviews, and theories about the relation of stuttering to laryngeal behavior. Also, to see if there have been over-interpretations of research results, or if there are alternative explanations to results, and through this discovery, clarify what is known to be true about the relation of stuttering to the vocal mechanisms or to the process of phonation.

I am eager to thank Hugo Gregory for his generous expressions of encouragement, for his support, and for a number of helpful suggestions. I am also indebted to Marty Adams for his thoughtful and constructive criticisms. These men should not be held responsible, however, for the opinions expressed in this monograph, which are mine.

C. Woodruff Starkweather
Temple University

Stuttering and Laryngeal Behavior: A Review

C. Woodruff Starkweather
Temple University

Abstract

This monograph is a critical review and analysis of recent writings on the laryngeal behavior of stutterers. Theories and hypotheses related to laryngeal behavior are described first. Then experimental studies are reviewed. These studies are grouped by topic and method, as follows: the physiology of stuttering, the adaptation effect, reaction time, voice onset and termination times, feature analysis, and fluency enhancement. An account is then given of data that are not explained adequately by vocalization hypotheses. A few general conclusions are then drawn. Finally, the clinical implications of the data are examined.

Although interest in the area of laryngeal behavior in stutterers has been high, the data have not adequately supported theoretical accounts. This is so because in much of this research, alternative explanations have been overlooked and the results overinterpreted. Despite this, it seems evident that clinical methods of evaluation and treatment should be modified to accommodate laryngeal behavior.

Introduction

To begin with, it will be helpful to consider two different hypotheses about the relation between laryngeal behavior and stuttering. The first, which we might call the weak vocalization hypothesis, is simply that the larynx is involved during stuttering; the stutterer stutters with his entire vocal tract. According to this "weak" hypothesis, there are laryngeal stuttering behaviors as well as oral articulatory ones (not to mention respiratory ones). This first hypothesis tells us very little about what causes stuttering, and is consequently called the weak vocalization hypothesis, although it has important clinical and diagnostic implications that will be described at the end of this monograph.

The second hypothesis is theoretically stronger. It is that laryngeal stuttering is *primary,* with the implication that oral articulatory stuttering is *secondary.* The primariness of laryngeal stuttering may be of two types—(a) it occurs first and is perhaps causative in the sequence of stuttering behaviors, or (b) it occurs first and is perhaps causative in the sequence of development. In either sense of *primary,* laryngeal behaviors may not only occur first but also cause or precipitate nonlaryngeal stuttering behaviors. Regardless of which interpretation of *primary* is used, this second hypothesis has great theoretical importance. If true, it could be explained in two ways: (a) stutterers differ from nonstutterers in the anatomy or physiology of their larynges or of the central nervous system (CNS) structures serving laryngeal behavior, or (b) laryngeal behavior is inherently more "susceptible" to stuttering. It may be for example, that the larynx has less variety of gesture available to it than other structures, as Perkins, Rudas, Johnson, & Bell (1976) have suggested. The second of these two explanations seems more plausible, and it permits laryngeal stuttering to be *primary* (either behaviorally or developmentally) without being directly related to what *causes* stuttering. But the first explanation is more exciting, because it begins to answer the question of what causes the disorder.

THE ORIGINS OF VOCALIZATION THEORY

Wingate's Review of the Conditions That Enhance Fluency

Although there were some earlier publications describing laryngeal anomalies in stutterers (Chevrie-Müller, 1963; Stromsta, 1959), vocalization theory first appeared as an explanatory concept in the works of Wingate (1969, 1970, 1976). Wingate (1976) reviewed the conditions that were known to make stutterers more fluent, and he reviewed in detail the conditions that had the most powerful fluency-enhancing effects—rhythmic stimulation as in metronomically paced speech, singing, choral speaking, shadowing, hearing loss, masking noise, and delayed auditory feedback. He concluded that these conditions all reduced the rate of speaking and that the vowel lengthening that occurred in slower speech was responsible for the enhancement of fluency.

For the metronome effect—the tendency of stutterers to speak more fluently when pacing their speech to a rhythmic stimulus—Wingate described four explanations that had been given by others, and he explained why they failed to account for the effect. The first explanation was distraction. Distraction could not account for the effect because several researchers (Azrin, Jones, & Flye, 1968; Brady, 1969; Fransella & Beech, 1965) had found that arhythmic pulses, which would presumably be more distracting, were less effective fluency-enhancers than rhythmic pulses. Furthermore, Fransella (1967) and Brady (1969) had found that distracting tasks by themselves had no fluency-enhancing effect.

The second explanation was that stutterers spoke slower when pacing their speech to a metronome, and slower speaking was known to promote fluency. However, Barber (1940) had found that rhythm reduces stuttering even at fast rates, and Fransella and Beech (1965) had found that although slower speaking rate and metronomic rhythm both reduce stuttering, the two effects were independent. Finally, when stutterers speak at their normal rate to the metronome they are more fluent even though rate has not been reduced (Brady, 1969).

The third explanation was that the metronome produced a masking noise, which, although it was brief and intermittent, served to mask the stutterer's speech from his own hearing. Wingate found this explanation unacceptable because of the finding (Meyer & Mair, 1963) that fluency could be enhanced by pacing speech to a rhythmic stimulus that was presented visually or tactually.

The fourth explanation was that the metronome caused the stutterer to speak more regularly. It induced regularity. Wingate argued that the stutterer's speech is not "regularized" by the metronome, and he offered evidence that showed there was indeed considerable irregularity of rhythm in the speech of stutterers as they spoke to the beat of a metronome. However, Wingate may have overlooked the fact that a stutterer's speech is considerably *more* regular when paced to a metronome than when speaking under normal conditions. Consequently, the regularity induced by the metronome could be responsible for the increased fluency. Wingate erred in assuming that perfect regularity was required to explain the effect. Wingate noted also that stutterers can be trained to speak to a metronome with several syllables or words per beat. In this case, Wingate commented, speech is only slightly regularized, if at all, and yet stutterers may still speak quite fluently under this condition. Wingate failed to note, however, that it requires training to speak in this way, therefore this condition does not produce immediate fluency the way speaking one syllable to a beat does. Consequently, even though speech is less regularized than when it is paced at a rate of one syllable to a beat, the training given to the stutterer may compensate for the additional irregularity. Of the untrained, syllable-timed metronome effect, Wingate says "the effect is immediate and continuous as well as pronounced" (p. 181). In fact, as we have just noted, it is *only* immediate and continuous when a substantial amount of regularity is induced. As Wingate noted, the stutterer times the stressed syllables to coincide with beats of the metronome, and does this by "placing emphasis on syllable nuclei" (p. 181). Wingate cited Boomsliter and Hastings' (1971) finding that the "instant" of the syllable is *almost exclusively* within its vowel section, and from that Wingate concluded that this action of placing emphasis on syllable nuclei is the means by which the guiding function of rhythm sets in motion and supports the central element of the rhythm effect—emphasis on phonation. Syllable nuclei are predominantly vowels, which are the major phonatory component of speech. Expressive emphasis of these components produces primarily an increase in their duration and some increase in volume. Wingate's conclusion is that the rhythm effect reduces stuttering because it causes the stutterer to emphasize vowels. Allen (1972) has shown that when nonstutterers are asked to locate the "beats" of a natural sentence, they place them in advance of the vowel by an amount proportional to the duration of the preceding consonant. "Natural beats" of a language may thus be located in the consonants that introduce stressed syllables or within the transition from consonant to vowel, but not within the vowel portion itself. We don't know how stutterers align their speech with a metronome as far as the consonant or the vowel of the stressed syllable is concerned, so the conclusion that the rhythm effect depends specifically on louder or longer vowels, and not on a general slowing of rate, which would include lengthened consonants as well, must be considered hypothetical until evidence is produced in support of it.[1]

[1]Brayton and Conture (1978) determined a significant negative correlation between vowel duration and the frequency of stuttering in metronomically paced oral reading, but they did not measure the duration of any consonants in this study, nor

Wingate noted further that there is a reduction of stress contrast in speech that is timed to one syllable per beat, and indeed normally unstressed syllables are produced as if stressed, so that all syllables are given their full color, intensity, and duration. Since stuttering occurs far more on stressed than on unstressed syllables (Brown, 1938), one would think that rendering all syllables into stressed form would increase, not decrease stuttering. Another way of making this point is to ask the following question: If it is longer and louder syllables that explain the rhythm effect, why do stutterers in unpaced speech stutter almost exclusively on stressed syllables, which are longer and louder than unstressed ones? The answer may be that the rhythm effect is caused not by more emphasis on phonation but by the reduction of stress contrast. When stress contrast is present, rapid changes in the speed of articulatory movement are necessary to change from longer durations of both the consonants and vowels associated with stressed syllables to the shorter consonant and vowel durations associated with unstressed syllables (Umeda, 1975, 1977). With a reduction of stress contrast, there is less need for changes in the speed of articulatory movement.

Wingate also said that there is a corresponding reduction in consonant articulation during metronomically paced speech, but there is no evidence of this. Since all syllables are pronounced as if stressed, metronomically paced speech should, if it has any effect, enhance on consonants by lengthening the ones that introduce syllables that would ordinarily be unstressed.

The second fluency-enhancing condition that Wingate reviewed is singing. The major effects of singing on speech, he says, are lengthening of the vowels, rate reduction, and greater vocal intensity. He suggests that these are all primarily vocalic changes. He argued that singing does not induce regularity in the rhythm of speech because the beat of the song does not correspond with the syllables of the lyrics. Of course it is true that not every syllable of a song is placed on a beat of the music, but the syllables, particularly the stressed syllables, are always located with regard to the music's rhythm—on beats, half beats, etc., so the timing of every syllable of a song is determined by the song's rhythm, which is regular. The syllables of a song may be said to be regularized, although they do not occur one to a beat. This may not be true of recitativo and rubato singing, where the song's rhythm becomes irregular, but we don't know what the effects of these types of singing are on fluency.

Wingate's argument about singing is that the effects are primarily vocal. However, they are not entirely vocal. The consonants of sung speech are also lengthened according to the data Wingate supplies. Of course, being shorter to begin with, they are not lengthened as much as the vowels, but proportionally the effect of singing on consonants seems to be nearly as great, and possibly even greater, than that on the vowels. Actually, what is lengthened most in singing are the steady-state portions of speech—vowels, silences, semivowels, nasals—portions in which there is relatively little change in frequency and intensity. In stutterers' fluent speech, nonsteady-state portions may be lengthened, although the data substantiating this point (Starkweather & Myers, 1978) are still quite limited, and they are disputed by some (Hand, Note 8). Familiarity with the words of a song may also account for a portion of the fluency-enhancing effects of singing (Healey, Mallard, & Adams, 1976).

The third fluency-enhancing condition Wingate discusses is choral speech, in which a stutterer reads in unison with another speaker. It seems clear, as Wingate argued, that the effect is not due to distraction, reduced communicative responsibility, or auditory masking. Wingate also rejected pace-setting (syllabic regularity) because "it is difficult to see how the accompanist can be considered to set the pace or lead" (p. 200). However, choral speech, whatever way it may be accomplished, is slower,[2] as Wingate acknowledged. He theorized that the vowels are more lengthened than the consonants by this rate reduction. Although it is probably true that the vowels are lengthened more than the consonants, the consonants are probably lengthened too and this change may consequently contribute to or be responsible for the fluency-enhancement. Klatt (1974) noted that speech sounds are changed in duration by any lengthening or shortening influence in proportion to their inherent duration. It is not clear, however, whether rate of speech in words per minute (WPM) is slower in choral speech because additional time is added between words, or because articulatory rate is slower. In the latter case, both vowels and consonants are probably lengthened. Recently Adams and Ramig (1980) have demonstrated that choral speaking can enhance fluency in the absence of rate reduction. However, in this demonstration the speaker clearly sets the pace. Consequently, fluency enhancement in choral speaking may be attributed either to lengthened segments or to pace-setting.

A fourth fluency-enhancing effect results from shadowing, in which the stutterer immediately repeats another speaker. It is much like choral speech except that there is a brief lag between the lead speaker and the "shadower." The original explanation of fluency in this condition was that the stutterer listened to the speaker's voice rather than his/her own (Cherry, 1953), but it is not just listening that is changed in shadowing. The rate is slower (Cherry, Sayers, & Marland, 1956), as Wingate

examine the rate of the subjects' speech in any way other than the duration of vowels. So, although there is a relation between vowel duration and fluency enhancement in rhythmic speech, the same relation may exist for consonant duration or coarticulation, or syllables per minute, and we cannot conclude that the fluency enhancing effect depends on vowel duration exclusively.

[2]This is true only when the model is present so that his speech can be affected. Stutterers speed up in order to follow a taped model (Adams & Ramig, 1980).

noted. The intelligibility of shadowed speech is reduced (Sargeant, 1961), giving it a "mumbled" quality. Clearly, consonants as well as vowels must be produced differently to give speech a mumbled quality, but it is unclear whether the time, intensity, or frequency characteristics of consonants and vowels are changed.

A fifth fluency-enhancing condition is hearing loss. Although the quality of the information is low because it is based on survey research (Wingate, 1970), it seems likely that some hearing-impaired people do stutter. Therefore, the relation between hearing loss and stuttering is not a basic one, an important point that Wingate made. Typically, "scanning" speech occurs, which involves a slower rate with lengthened vowels and poorly articulated consonants. Again, the rate itself, not necessarily the lengthened vowels, may be responsible for the fluency. There is no evidence that consonants are lengthened, although they are changed in some way.

A sixth fluency-enhancing condition is masking noise. It seems clear that the fluency that results from the stutterer's not being able to hear his/her own voice does not result from a reduction of anxiety, since the fluency-enhancement takes place in the absence of changes in the level of autonomic arousal (Adams & Moore, 1972). Wingate contends that the fluency-enhancing effects of masking noise lie in the changes that the noise makes in the stutterer's speech, that is, the changes induced by the Lombard Effect. In nonstutterers, the Lombard Effect includes increased fundamental frequency, increased vocal intensity, and reduced rate. Ringel and Steer (1963) found longer phonation and longer syllables, but words per minute, although slower, was not significantly slower. Ringel and Steer, however, used as subjects 13 females speaking sample sentences. It seems reasonable to assume, in the absence of much evidence, that both consonants and vowels are lengthened as rate is reduced in an attempt to improve intelligibility in the presence of the masking noise. Vowels are lengthened more in absolute terms because of their longer inherent duration, but proportionally, there may be no difference in the lengthening of vowels and consonants under masking noise. In any event, there is no reason to attribute the effect solely to changes in vowels when consonants are probably affected too.[3]

The final condition Wingate reviewed is delayed auditory feedback (DAF), in which the stutterer's voice is fed back electronically with a slight delay. Wingate argued that fluency in this condition is not attributable to the feedback change itself, but to changes in the manner of speaking. Reduced rate is one of those changes. In nonstutterers, both vowels and consonants are lengthened in DAF, and it is the inherent duration of the sound that determines the extent to which it will be lengthened (Agnello, 1970). Furthermore, McKay (1968) found that nonstuttering subjects who spoke slower without delayed feedback showed their maximum disruption at longer delays than those who spoke more rapidly without delay. Therefore, the extent of change that DAF produces on speech varies directly with the duration of the sounds and syllables being produced.

Following his review of the various conditions that enhance fluency in stutterers, Wingate concluded that reduced rate is common to all the conditions. One of the characteristics of speech spoken at slower rates is increased vowel duration. In one case—metronomically paced speech—lengthened vowels are not known, but only supposed to occur. Furthermore, it is known that the fluency-enhancing effects of metronomically paced speech are independent of rate reduction in the same condition. Wingate deduced that vowels are lengthened in paced speech, independent of rate changes, from the observation (Boomsliter & Hasting, 1971) that the "beats" of speech rhythm are located within the stressed vowel. Others, however (Allen, 1972) have more precisely located the beats of English within the consonant preceding the stressed vowel. In either event, the idea that vowels are lengthened during metronomically paced speech is an inference. The idea that the consonants are "subordinated" is a second inference derived from the first.

Reduced rate has a number of other characteristics besides lengthened vowels:

1. Speech is physiologically simplified because there are fewer gestures per unit of time.
2. Consonants are lengthened as well as vowels (Klatt, 1974).
3. Coarticulatory overlap is decreased (Stevens & House, 1963).
4. There is full vowel color on stressed vowels, at least at normal rates as compared to fast rates where vowel color is diminished (Gay, 1978).
5. There is probably more flexibility in the timing of stressed syllables, since it is known that there is less flexibility at fast rates (Folkins, Miller, & Minifie, 1975).

Wingate's conclusion that what the fluency-enhancing conditions have in common is vowel lengthening explains fluency enhancement only if vowel lengthening is the only characteristic these conditions have in common. Other characteristics, also shared by all of these conditions, diminish Wingate's explanation. Since vowel lengthening is only one aspect of reduced rate, the other aspects of reduced rate are equally likely explanations. But there is one condition that enhances fluency without reducing rate—metronomic pacing. Wingate's analysis is unburdened by this fact because he inferred that vowels are lengthened in metronomically paced speech. For this inference to have explanatory power, it is necessary for vowels to be lengthened even in speech that is paced at normal rate and for the *stressed* vowels to be lengthened, since they are the ones that would be stuttered. Wingate doesn't make these extensions in his review, but without them the vocalization hypothesis fails to account for one of the strongest fluency-enhancers. Of course, the question is an empirical one—are stressed vowels lengthened in speech paced at normal rates? To

[3]See pages 29-30 for more discussion of the masking effect.

date, this question has not been the subject of experimental investigation as far as I know.[4] In the absence of any facts, the inference seems unlikely to me. A better explanation is that pacing speech enhances fluency either by inducing regularity of timing, that is, by producing less variation in the time interval between stressed syllables, or by inducing less variation between stress levels. When the *durational* aspect of stress is considered alone, reduced variation in rhythm and reduced variation in stress level may be the same thing. Of course, it is possible that *both* rate-reduction and pacesetting induce fluency, either independently or because of some common characteristic.

On first analysis, then, it would seem that there may be two different explanations for fluency-enhancement—reduced rate, or some aspect of reduced rate on the one hand, and induced regularity on the other.

The use of two variables to explain a phenomenon is less desirable than using one variable on the principle (Occam's Razor) that the more parsimonious explanation has a better chance of being correct. It is consequently desirable (although be no means necessary) to seek a more parsimonious explanation.

In normal speech, articulatory rate changes frequently, speeding up and slowing down at certain locations. Specifically, these locations where the speed of articulatory movement changes are identified with the following observations:

1. Vowels are shortened at the beginnings of words and at the beginnings of sentences (Oller, 1973; Umeda, 1975).
2. Vowels and consonants are both shortened in longer utterances (Jones, 1948; Lindblom, 1968).
3. Consonants are shortened after a pause (Umeda, 1977).
4. Consonants initiating a word are longer than those in mid-word or in final position (Umeda, 1977).
5. Consonants and vowels are both longer in stressed than in unstressed syllables (Klatt, 1974; Umeda, 1975).
6. Consonants are shorter in words of greater length (Klatt, 1974).
7. /s/, at least, is lengthened in words that occur more rarely in the language (Umeda, 1977).

These locations of rate change in normal speech are also the locations at which stuttering and normal nonfluencies are likely to occur (Brown, 1938; Brown & Moren, 1942; Conway & Quarrington, 1963; Hahn, 1942; Hejna, 1955; Ronson, 1976; Soderberg, 1966; Taylor, 1966; Tornick & Bloodstein, 1976; Wall, 1977).

Speech rate is typically close to maximum. Typical adult speech rate is 5-6 syllables per second (Malécot, Johnston, & Kizziar, 1972; Miller, 1951; Walker & Black, 1950), while maximum diadochokinetic rate is 6-7 movements per second (Daniloff, 1973), although diadochokinesis is probably more difficult to coordinate than speech and is not highly correlated with conversational rates (Lass & Sandusky, 1971). A better estimate of maximum articulatory rate may come from reaction time experiments. Netsell and Daniel (1974) found that reaction time for the lips (the slowest articulator) is 200 msec, corresponding to five movements per second. It seems evident that in speaking we move our articulators nearly as fast as they can be moved. Stutterers as a group have slower vocal and articulatory reaction times than nonstutterers (Adams & Hayden, 1976; Adler, 1977; Starkweather, Hirschman, & Tannenbaum, 1976; MacFarlane, Note 14). Therefore, stutterers may speak closer to their maximum rate of articulation or they may speak more slowly, or both.[5]

If stutterers speak at a rate close to their maximum rate of articulation, then speaking at a slower rate of articulation may make the stutterer more fluent because it provides a wider range of rate changes. The rules of the language require a speaker to speed up at some points, slow down at others. Both can be done if the stutterer is speaking at a slower rate to begin with. Because the articulators are already moving nearly as fast as they can, only slowing can be accomplished. Slower rate may enhance fluency by providing the stutterer with this flexibility. Similarly, regularized speech may enhance fluency by calling for fewer rate changes in the same period of time, since rate changes associated with stress, syntactic boundary, and length of sentence, at least, would be eliminated. Consequently, it may be that both regularized and slower speech enhance fluency because they demand less of the stutterer in the way of rate adjustment, the former because it requires fewer of these adjustments, the latter because it allows more room to make them.

Two other facts about stuttering are related to Wingate's conclusion that fluency-enhancement results from lengthened vowels and subordinated consonants. In free speech, stuttering tends to occur at some locations where vowels are shortened—the beginnings of utterances, the beginnings of words, and on longer words. It makes sense that lengthened vowels would enhance fluency, if shortened vowels enhance stuttering. Unfortunately, there are other locations where lengthened vowels and/or shortened consonants occur and where stuttering is likely. Vowels are lengthened on stressed syllables, and consonants are shortened after a pause and in longer utterances.

The second fact is that there is a major fluency-enhancer, whispering, in which phonation is greatly reduced or even entirely eliminated. The timing of whispered speech is altered by reducing rate and thereby lengthening the duration of both vowels and consonants

[4]Brayton and Conture (1978) established that vowels are lengthened in stutterers' paced oral reading and that the change in vowel duration is related to changes in fluency, but they did not report other aspects of rate.

[5]It is difficult to assess the rate of speech in stuttering independent of the stuttering itself. Even when perceptible stutterings are excised, covert or "subacoustic" stutterings may remain.

in normal speakers (Parnell, Amerman, & Wells, 1977). Far from stressing vowels and subordinating consonants, vowels in whisper are subordinated in intensity and consonants are probably over-articulated. Yet fluency is definitely enhanced, although not so much as in paced speech, singing, or choral speech (Wingate, 1976).

In summary, Wingate has reviewed a number of the conditions under which stuttering is ameliorated. On the positive side, he has effectively dismissed from consideration explanations based on distraction, changes in audition, or reduced communicative responsibility and focussed attention on the changes these conditions produce in speech rate. A difficulty arises in that the metronome effect is known to ameliorate stuttering even when speech is produced at normal rate. To deal with this problem, Wingate extrapolated one change associated with reduced rate, lengthened syllable duration, and describes this change as an emphasis on vowels and a subordination of consonants. He then inferred, without supporting evidence, that metronomic pacing also involves an emphasis on vowels and a subordination of consonants. Consonants, however, are not subordinated at slow rates but are instead lengthened along with vowels to a degree proportional to their inherent duration, at least in nonstutterers (Klatt, 1974). Furthermore, in some conditions that ameliorate stuttering, for example, under masking noise, consonants may well be over-articulated, although in others, such as shadowing, they may be "mumbled."

Wingate contended that metronomic pacing cannot be due to "induced regularization" because paced speech is not perfectly regular. It is, however, more regular than unpaced speech, so regularization induced by the pacing could be responsible for the effects.

Characteristics of reduced rate other than vowel lengthening, consonant lengthening, fewer gestures per unit time, decreased coarticulation, and increased flexibility in the timing of stressed syllables are overlooked as explanations of fluency enhancement.

An explanation of fluency enhancement more closely aligned with the facts is that the effect is achieved because fewer gestures are required per unit of time as a result of reduced rate in the case of masking noise, DAF, shadowing, choral speaking, and induced regularization in metronomic pacing and singing. In addition to reduced rate, choral speaking, and shadowing, involve alterations of timing and the provision of a timing model or template that may also diminish the complexity of the speech act, but the mechanism by which this might be achieved remains unclear.

Finally, Wingate's vocalization hypothesis fails to account for whispering, a condition known to ameliorate stuttering in which speech is physiologically simplified by requiring fewer gestures per unit of time but in which vowels are definitely subordinated. The vocalization explanation also fails to account for the fact that stuttering is *more* likely to occur at stressed syllables, where vowels are emphasized, and in longer utterances and after a pause, where consonants are shortened.

Schwartz's Theory

In 1974, Schwartz published a theory that attempted to explain stuttering as a disorder of the vocal mechanism, specifically as a hyperexcitability of vocal reflexes. Schwartz's theory is centered around the airway dilation reflex (ADR). This reflex is a rapid opening of the glottis during inspiration in response to rising subglottic air pressure, as might occur in the presence of an airway obstruction or a need for extra air. The glottis is opened by contraction of the posterior cricoarytenoid (PCA). Schwartz's idea is that the receptors that initiate the ADR in response to rising subglottal air pressure function during expiration as well as during inspiration. However, subglottal pressure is 14-16 times greater during speech than during free exhalation. Therefore, the PCA must be inhibited during speech in order to achieve phonation. Without such inhibition, the glottis would stay open as a result of the ADR occurring in response to the additional air pressure. The matter is complicated further by the fact that during intervocalic intervals (IVI) containing voiceless consonants, the inhibition of the PCA must be released so that voicing can stop and then the inhibition must be quickly restored so that voicing can continue again. Therefore, alternate inhibition and disinhibition of PCA must characterize normal speech. The control over these rapid changes in excitability of the PCA is supplied, according to Schwartz's theory, by supra-medullary control centers that "dominate" the medullary ADR. Under stress, Schwartz contended, supra-medullary control is lost in addition supra-tidal respiratory demands may be made, and the ADR is disinhibited. The theory is succinctly stated by Schwartz, "According to this model, both factors are probably always present at the moment of a stuttering block; that is, psychological stress produces a requirement for supratidal volumes and a tendency to misinterpret the air pressures required for speech as an airway obstruction, to produce a vigorous *abductive* response of the larynx" (p. 173). (Italics mine.) To overcome the speech difficulties produced by the disinhibited ADR, Schwartz suggested that an individual might forcefully contract the laryngeal musculature, which might completely block phonation or produce hypertense phonation. Or the individual might display hypertense postures of the lips, tongue, jaw, etc., to "release the abducted larynx" (close the glottis). These additional blockages make matters worse by further obstructing the airway and raising subglottal pressures. He noted that there might also be other sites of compensation—respiratory activity or movements of the limbs or face—that are learned in an attempt to deal with the laryngeal problem. Avoidance responses are also learned.

Clearly, this hypothesis falls into the second of the two categories described earlier. That is, it is a "strong" hypothesis, suggesting that laryngeal behaviors are primary both in the sequence of stuttering behaviors and in the sequence of development. Furthermore, although Schwartz does not explicitly say so, the implication is

fairly clear that the primariness of laryngeal stuttering behavior results from a difference between stutterers and nonstutterers, specifically in their capacity to control rapid sequences of inhibition and disinhibition of the ADR, and not from laryngeal behavior being inherently more susceptible to stuttering. In other words, Schwartz is suggesting an etiology of the disorder.

Zimmermann and Allen (1975) take issue with Schwartz on several points. They pointed out that there is no evidence for a loss of PCA inhibition in stutterers, and that consequently this crucial aspect of the theory is speculative. They noted also several predictions that can be derived from the theory that seem to be contradictory to fact:

1. There will be no stuttering on voiceless consonants, since inhibition of the PCA is not necessary for these sounds.
2. In CV syllables initiated by a voiced consonant, voicing should either stop or at least be breathy.
3. Stutterers should stutter only under stress.

In addition to these false predictions, Zimmermann and Allen contend that the theory does not account for repetitions. That is, there is no explanation in the theory for why stuttering behaviors are often repetitive in form.

Adams (1975) also takes issue with Schwartz, suggesting that the "coping strategies" Schwartz describes must, by his own description, fail most of the time. Why then does the stutterer continue to use them? Adams, agreeing with Zimmermann and Allen, suggests also that the theory fails to account for the repetitive behavior of stuttering and adds that it also does not explain prolongations, particularly the audible prolongation of voiced continuants, which, according to the theory should be less likely to occur in stutterers than in nonstutterers.

Schwartz (1975), replying to Zimmermann and Allen's contention that the model predicts no stuttering on voiceless consonants, says that the ADR results in *more* abduction than is "normal" for these sounds, making the transition to subsequent sounds aphonic or breathy. Not permitting this, or not wanting it to happen, the stutterer repeats and prolongs these sounds. This answer responds incidentally to the objections raised about the form of stuttering and is an elaboration of the theory. From this elaboration, one might infer that stutterers prefer to repeat sounds than to have aphonic or breathy transitions. Although this seems unlikely, in view of the low regard stutterers seem to have for repetitions, there are no data to my knowledge that confirm or deconfirm this inference. Another prediction than can be derived from Schwartz's response is that on voiceless consonants, stutterers must either be able to predict stuttering or show dysphonia. Again, there are no data to confirm or deconfirm this prediction.

In response to Zimmermann and Allen's criticism that in CV transitions from voiced consonants, voicing should either stop or be breathy, Schwartz replied that "laryngeal lock-up" occurs. The stutterer, anticipating the ADR, jams the folds together to prevent their abduction. Again, a prediction derivable from Schwartz's reply is that in the production of CV transitions from voiced consonants, stutterers must either be able to predict stuttering or show breathy phonation, if not abducted aphonia. No data confirm or deconfirm the prediction.

Another set of criticisms of Schwartz's theory have been made by Freeman, Ushijima, and Hirose (1975). They questioned Schwartz's description of the physiology of respiration. Specifically, they asserted that the PCA is *not* involved in expiratory airway regulation, only in inspiratory regulation. Furthermore, the subglottic receptors, reacting to rising expiratory air pressure, which Schwartz uses to explain the reflex, do not exist. The receptors are sensitive only during inspiration to dilate the airway. They play no role in suppressing ADR during expiration. (It should be noted that the PCA *does* dilate the airway during expiration at the same time as supraglottal articulators partially occlude it. There is no evidence however, that this dilation is subcortical or reflexive. It is simply part of the speech gesture for producing a supra-glottal sound). In response to this criticism, Schwartz acknowledged that the PCA is not active in expiration against a resistance, although he noted that in some of his work with children (which is not described) PCA dilation before utterance continues on into utterance. This weak defense, if not capitulation, against a criticism of the most essential aspect of Schwartz's theory seems to all but destroy it. It is difficult to understand how, having made this crucial concession, Schwartz can continue to defend other aspects of the theory.

Freeman, et al. make one other important criticism. They note that Schwartz's model predicts sudden, inappropriately high levels of PCA activity preceding stuttering blocks. Despite their own examination of this muscle's activity by hooked wire electrode EMG, no such evidence has appeared, although the PCA does show abnormal patterns during blocking, as do other muscles. Also, they noted that there is no sign that PCA activity *triggers* blocking. Schwartz answered this criticism by saying that PCA activity need not precede blocking. "Very often, voluntary, anticipatory abduction precedes the ADR as an attempt to cope with the response of the PCA" (Schwartz, 1975, p. 140). The source of this information is not clear, but if true, it suggests that the stutterer is able to inhibit the PCA as a coping behavior even though Schwartz's theory says that the stutterer is unable to inhibit the PCA at all.

Another prediction is derivable from Schwartz's theory and his elaborations on it. The theory predicts that when stuttering is not present, as in the utterance of simple sounds or nonsense syllables, stutterers' sound productions that involve laryngeal opening, such as /s/, should be faster than those of nonstutterers. At least they should not be slower. This prediction has been deconfirmed by at least one experiment (Adler, 1977) that will be described later. A number of other predictions derivable from Schwartz's theory have also been deconfirmed, and these too will be described later.

Adams' Position

Adams (1974) has described a different kind of position from that of Schwartz. More cautiously stated and more empirically based, Adams' has integrated a description of the facts related to stuttering and vocalization from which some predictions can be made. Adams based his position on the contributions that respiration, phonation, and articulation make to normally fluent speech, noting that not only articulation, but the timing and smoothness of vocal initiation, and the duration of airflow are necessary aspects of fluency. Furthermore, there must be "harmonious integrations of subglottal pressure, glottal resistance, and supraglottal pressure" (p. 13). By contrast, respiratory, laryngeal, and articulatory behavior during moments of stuttering shows a variety of anomalies. In respiration, shallow breathing, gasping, fluttering of the diaphragm, and uncoordinated movements have been identified during stuttering, and Adams noted that these anamalous behaviors "could generate stuttering." Also, stutterers have been shown to have less disfluency with deeper respirations than with more shallow ones (Starbuck & Steer, 1954).

As far as laryngeal behavior is concerned, arrhythmic vocal fold vibrations under certain conditions and isolated and "unpredictable" glottal openings, partial or complete absence of voicing during persistent rapid glottal activity, and defects in pitch modulation have all been observed by Chevrie-Müller (1963). Stromsta (1956) observed that there were phonatory stoppages during stuttering, and Adams and Reis (1971; 1973)[6] found less stuttering on the reading of a passage that was constructed to have no voiceless sounds. Finally, Adams recalls the conclusion reached by Wingate (1976) that a number of conditions sharing the characteristic of a change in the manner of vocalization tend to promote fluency in stutterers.

Without explicitly reviewing all the well-known articulatory anomalies seen in stutterers, Adams noted that some supraglottal stutterings may inhibit voicing by increasing supraglottal pressure to the point where the pressure gradient across the glottis is insufficient for vocal fold vibration. In substantiation of this point, he notes that intraoral air pressure is abnormally high in stutterers, even in single simple syllables (Agnello & Wingate, Note 1; Hutchinson, 1973). Note the direction of the effects Adams is suggesting—articulatory abnormalities may inhibit phonation. However, it may also be the case that phonatory abnormalities result in articulatory disturbance. When articulatory disturbances affect phonation, the initiation, timing, and maintenance of airflow and phonation is difficult to achieve. "It seems that adequate levels of subglottic pressure cannot be mounted and sustained when needed" (Adams, 1974, p. 24). However, it may also be that glottal disturbances exist, and when this is the case, it is not surprising that articulatory behavior deteriorates. "Articulatory postures and gesture are prolonged and repeated as the individual strives to coordinate labial, mandibular, and lingual movements with a wavering airstream and vocal note that sometimes are not present when needed. Only when dependable airflow and phonation are restored does fluent articulation appear" (Adams, 1974, p. 24). The direction of effect may be either way, so the problem may most accurately be described as a lack of coordination among the respiratory, phonatory, and articulatory systems. This two-way discoordination refers to the sequence of stuttering behaviors in the already-developed stutterer. As far as the development of the disorder in children is concerned, Adams suggests the possibility that the vocal abnormalities may precede the supraglottal ones, although no sources are cited in substantiation of this point.

Adams' (1978) careful review sets the stage for a more refined statement: "... fluent utterances produced by normals and stutterers are consistently associated with the prompt, smooth initiation and maintenance of voicing, and the integration of respiration, phonation, and articulation ... [whereas] the stutterings produced by stutterers involved abnormal respiratory, laryngeal, and articulatory events [and] disruptions in the coordination of the three systems ... [Furthermore], the stutterers' losses of coordination invariably involve obvious deviations from the timing and sequencing of normals" (Adams, 1978, p. 142). These views might be called "systems discoordination theory."[7]

Several predictions are derivable from this position. First the discoordination aspect predicts that any condition that simplifies speech by reducing the number of vocal or articulatory gestures per unit of time will result in less stuttering, a prediction that is confirmed by the fact that stuttering is less frequent when rate is reduced. The "systems" aspect predicts that stuttering will be reduced when the contribution of one system can be removed or greatly reduced because this will also reduce the amount of required coordination. This prediction is confirmed by the fact that stuttering is reduced in whisper.

Despite these confirmed predictions, the systems discoordination position is unsatisfactory because it lacks specificity. Of course, Adams did not intend this position to explain all aspects of the disorder. But nonetheless, the position does not provide much guidance about the origin of the stutterer's lower level of coordination. Nor does it help us understand why this lower level of coordination manifests itself only in the speech mechanism (if in fact it does) or why stutterers, at least in the early stages, have long episodes of fluent speech, or why stuttering follows a course of spontaneous recovery in most children but a regressive course in a few, or why males

[6]These two studies by Adams and Reis will be reviewed in detail.

[7]Adams did not intend for these views to be considered a "theory."

stutter more than females, or why stuttering occurs more at certain linguistic locations than at others. Because these specifics are lacking, further predictions are difficult to derive, and the predictions that can be confirmed are equally confirmatory of other, quite different, positions.

The integrations of data and the theoretical ideas of Wingate, Schwartz, and Adams, which we have just reviewed, form the background for the recent research on the relation between stuttering and vocalization that is the topic of this monograph. The section that follows provides a review of that research.

RECENT RESEARCH

The Physiology of Stuttering

In 1975, Freeman and Ushijima examined the fluent and stuttered utterances of a single stutterer by recording the muscle action potentials, via hooked wire electrodes, of the following muscles:

Muscle	*Function*
Posterior cricoarytenoid	opens the glottis
lateral cricoarytenoid	closes the glottis
vocalis	closes the glottis
cricothyroid	lenthens the glottis
inferior longitudinal	lowers tonguetip, shortens tongue
superior longitudinal	elevates tonguetip, shortens tongue
genioglossus	protrudes tongue
orbicularis oris	purses lips

During production of the word *less*, which was stuttered, there was activity in the tongue muscles well before onset of any acoustical signal, and there was a gradual increase in activity of the posterior cricoarytenoid (PCA) and vocalis (VOC) simultaneously. The first observation might be described as an error of timing or coordination. The second observation, which was of simultaneous contraction of an adductor and abductor, also suggested mistiming or uncoordination but of a specific type—an absence of the usual reciprocity that characterizes antagonistic muscles. In the subject's fluent utterance of the same word, reciprocity of antagonistic muscles is evident, and muscle tensions are appropriately timed with each other. The comparison of fluent and stuttered utterances of the word *effect* showed a similar phenomenon: excessively prolonged and high levels of lateral cricoarytenoid (adductor) tension throughout the word during stuttering, but brief spikes of activity for devoicing at /f/ and /kt/ in the fluent production. The same phenomena were seen in comparisons of fluent and stuttered utterances of the word *ancient*. Two conclusions seem appropriate; stuttering consists of or is accompanied by inappropriately timed and excessively high levels of muscular tension in laryngeal and supralaryngeal muscles, while fluent utterances are normally timed and have appropriate levels of tension.

In 1978, Freeman and Ushijima published a series of similar observations of the stuttered and fluent utterances of four stutterers. The subjects spoke under a variety of conditions—reading a prose passage, pacing their speech with a metronome, talking with masking noise, speaking in chorus, and speaking with delayed auditory feedback. One subject, who did not stutter when reading, spoke spontaneously under similar conditions. Muscle action potentials were recorded for all subjects via hooked wire electrodes.

The results were divided for convenience into those pertaining to levels of muscle activity and those pertaining to coordination. It was clear by inspection that there were higher levels of activity of the laryngeal muscles during stuttering, including stuttering on consonants, than during fluent utterances. In the fluency-enhancing conditions, the less frequent stuttering naturally increased the number of syllables produced per minute, which increased "artificially" the levels of muscle activity. This artifact was counteracted by using the average muscle activity level per syllable. With this adjustment it was evident that all muscles, oral and laryngeal, showed less activity during fluency than during stuttering. For one subject there was a greater difference between stuttered and fluent utterances in the laryngeal than in the oral muscles. This difference was not tested statistically, but was consistent. For the other subjects, no tendency suggestive of a difference between oral and laryngeal muscles was evident.

A comparison of 23 stuttered utterances of the word *syllable* with 26 fluent productions of the same word in one subject made it clear that there was less muscle activity for all muscles, oral and laryngeal, during fluent productions.

The findings related to coordination were equally interesting. Normally, adductors and abductors act reciprocally, that is, they do not contract simultaneously. During stuttering, however, Freeman and Ushijima observed significant positive correlations for antagonistic muscles during stuttering and significant negative correlations for antagonistic muscles during fluency. In other words, a high activity level in a particular muscle was predictive of a high activity level in that muscle's antagonists during stuttering but predictive of low levels of activity in the same muscles during fluency. However, there were exceptions to this observation. Correlations were negative in some of the stuttered utterances and positive in some fluent utterances. The exceptions seemed to constitute a significant minority—13% of the stuttered utterances showed negative correlations, 27% of the fluent utterances showed positive correlations. These minority correlations, although significantly greater than zero, tended to be smaller in degree than the majority. So, some fluent utterances show nonreciprocity and some stuttered utterances show reciprocity, although it is clear that stuttered speech is characterized by an absence of reciprocity in the laryngeal muscles, and in the case of

one subject, this was also true for a consonant prolongation. Considering the extent of the "minority report" in these observations, it would not be appropriate to say that stuttering is the absence of reciprocity or that fluency is definable by reciprocally functioning muscles. It is worth noting that in the one subject for whom a consonant prolongation was observed, there was an absence of reciprocity between the oral and the laryngeal muscles, confirming Adams' position on systems discoordination, at least in this one subject's consonant prolongation.

Because there is no measurement of antagonistic oral muscle activity, we do not know if the laryngeal muscles are the only ones that show nonreciprocity during stuttering. Possibly, the oral muscles also show this characteristic, and considering the high activity levels that were found in the oral muscles in this study, one would guess that nonreciprocity also characterizes stuttering in the oral muscles.

In their interpretation of these results, Freeman and Ushijima made a distinction between "distal" and "proximal" "causes" of stuttering, the former referring to etiology while the latter refers perhaps more to what have been called "precipitators" of stuttering or "triggers." The authors imply that their data bear more on proximal than on distal causes of stuttering. They noted that in normal speech and in the fluent speech of stutterers, reciprocity is evident. This suggests, that the stutterer's larynx is capable of normal function, at least insofar as reciprocity signifies normal function. However, because they observed an absence of reciprocity in laryngeal muscles during stuttering, the authors conclude that "a laryngeal component may be sufficient to account for the critical behaviors of stuttering" (p. 558). They hasten to add, however, that the "laryngeal component may be sufficient without being primary or necessary." Perhaps another way of saying this is that the laryngeal component is no more sufficient to account for the critical behaviors of stuttering than an oral articulatory component. Although acknowledging that more subjects are necessary before firm conclusions can be reached about laryngeal stuttering, the authors noted that in other studies with other subjects, "laryngeal involvement" has been found, and that therefore "laryngeal involvement is not a phenomenon idiosyncractic of these subjects (p. 558). This conclusion, although accurate, may tend to mislead. Probably most stutterers *do* stutter with their larynges as well as with their mouths, but there may be some stutterers who do not. Quite clearly a sampling study of laryngeal stuttering is needed before any firm conclusions can be drawn. The N's have been small.

The authors noted that "the data also support Wingate's hypothesis that the fluency-evoking conditions effect changes in the manner of vocalization. When the experimental subjects spoke under the four selected fluency-evoking conditions the levels of laryngeal muscle activity were generally lower, and fewer instances of abductor-adductor co-contraction occurred" (p. 559). Although the conclusion is correct, the interpretation is not. Wingate's vocalization hypothesis was that vocalization was the *only* change held in common by all fluency-evoking conditions, otherwise his hypothesis has little theoretical significance. In this study the activity level of the oral articulators also was reduced. Therefore these results do not support Wingate's hypothesis except in the limited sense that vocalization is among the things changed during fluency-changing conditions.

Ford and Luper (Note 7) investigated several physiological parameters of speech production. They gave three adult stutterers a word list containing a variety of sounds (/p/, /b/, /m/, /h/, /i/, and /a/) and asked them to repeat the words. Fluent and stuttered productions of the same words were compared. Five different measures were made: (a) vocalization, monitored by a contact microphone on the thyroid lamina, (b) intraoral air pressure, following Hardy's (1965) procedure, (c) subglottic air pressure, by a hypodermic needle inserted between the fifth and sixth crichoid rings, and (d) muscle action potential (MAP) at orbicularis oris inferior, by hooked wire electrode. The last two measures were for lip closure and lip opening respectively.

One subject showed a silent prolongation of /p/ on several productions, which could be compared to his/her fluent productions. The fluent productions showed a closing of the lips, followed by elevations of first subglottal, then intraoral air pressure, then lip opening and the onset of phonation. When /p/ was stuttered, however, both air pressure measures rose with lip closure, but then steadily fell as the lips tremored, reaching levels lower than in fluent productions before tremoring stopped and the lips opened. The abnormality was predominantly supraglottal.

Another subject also showed silent prolongations of /p/. For this subject, subglottal and intraoral air pressure increased during lip closure and exceeded the levels observed during fluency at the moment of release. This pressure elevation was preceded and accompanied by antagonistic labial activity.

A third subject showed silent prolongations characterized by a failure to achieve subglottic air pressure at a level equal to that which accompanied fluent productions of the same sound. Once sufficient pressure was achieved, the lips were opened. In other words, the site of difficulty appeared to be suboral, and the oral gestures were appropriately delayed until suboral conditions were adequate for production.

These and other similarly diverse observations led the authors to conclude that "apparent incoordination of [supraglottal] motor activity may precede, accompany, or follow onset of phonation and . . . the most obvious disruption of speech production processes may appear to originate in different parts of the mechanism" (p. 8). They also felt that ". . . single variable analysis of physiological activity accompanying disfluency may result in inappropriate emphasis on the contribution of that single activity" (Ford & Luper, p. 8).

Shapiro (1980) followed up the work of Freeman and colleagues with a similar investigation that examined

four stutterers and one nonstutterer electromyographically. Hooked wire electrodes were inserted with verification in the following muscles:

1. Orbicularis oris (N = 4)
2. Superior longitudinal (N = 3)
3. Lateral cricoarytenoid (N = 1)
4. Interarytenoid (N = 2)
5. Posterior cricoarytneoid (N = 1)
6. Vocalis (N = 2) (plus one insertion that was verified but provided weak signals)
7. Cricothyroid (N = 2)

Target words were embedded in a carrier phrase, and multiple tokens of the same type were obtained for comparison. Productions of the stutterers were judged by a reliable procedure to be fluent or nonfluent. Consequently, it was possible to compare the muscle activity of tokens judged to be fluent with those judged to be nonfluent. Three types of abnormal muscle function were observed in the stuttered tokens: (a) excessive muscle activity during production, (b) nonreciprocity, and (c) inappropriately timed activity before and after utterance. There was a clear tendency for these abnormalities to be present in the stuttered tokens more often than in the nonstuttered tokens. The same three types of abnormality were observed in oral articulatory as well as laryngeal muscles. These results clearly corroborated the observations of Freeman and colleagues that during stuttering, laryngeal muscles are abnormally active, the activity is poorly timed with regard to the segment being produced, and the activity is poorly coordinated in that antagonistic muscles tend to contract simultaneously. However, Shapiro also observed that all three types of abnormality were present in the oral articulatory as well as the laryngeal muscles. One further observation by Shapiro has very serious implications for many types of stuttering research. The same three types of muscle abnormality were also found in a number of tokens produced by the stutterers that were judged to be acoustically and behaviorally normal. No such behaviors occurred in the one normal subject. Many experiments on stuttering have been based on the acoustic analysis of speech that is judged to be fluent. Now it must be questioned whether the tokens observed in those investigations were muscularly as well as acoustically normal, and it appears to be necessary to repeat some of those experiments with a more vigorous operational definition of normal utterance. It is regrettable that so far the only technique for verifying the muscular normality of an utterance is by hooked wire electrode. A strong priority should be given to research into less invasive methods of verifying the normality of stutterers' speech. Also, there have been very few observations of laryngeal muscle activity in nonstutterers. It could be that nonreciprocity is more common in their speech than previously thought.

Another investigation into the physiology of stuttering was carried out by Conture, McCall, and Brewer (1977). From Schwartz's theory the PCA is disinhibited during stuttering, the authors predicted that laryngeal behaviors during the production of different types of stuttering should be similar in form. They examined 10 adult stutterers during a variety of different speech production tasks, but reported the results of only two of these tasks—conversation and the oral reading of a prose passage. During these tasks, the glottis was directly observed by a fiberoptic technique, and the presence or absence of glottal opening was noted during stuttered utterances. Typically, the glottis was either open or closed during a stuttered production, although occasionally there were changes in glottal status during stuttering. Three types of stuttering, part-word repetitions, prolongations, and broken words[8] were observed. A significant relation between the type of laryngeal behavior and the type of stuttering was found. Specifically, there was a significant tendency for part-word repetitions to be accompanied by abduction of the glottis (this occurred 60% of the time) and for prolongations to be accompanied by adduction of the glottis (this occurred 72% of the time). All of the broken words were accompanied by glottal abduction, but the sample of this stuttering type was too small to be the basis for generalization. The authors also noted that laryngeal behavior during prolongations was always appropriate for the sound being prolonged, that is, open for voiceless consonants, closed for vowels and voiced consonants. During part-word repetitions, however, the laryngeal behavior showed no relation to the normal voicing characteristic of the sound being repeated. It is important to note that despite the significance of the relation between stuttering type and glottal state, a substantial minority of part-word repetitions were produced with laryngeal adduction and that a substantial minority of prolongations were produced with glottal abduction. Consequently, the relation between oral and laryngeal stuttering behaviors is probabilistic.

Three conclusions seem justified by these observations. (a) Laryngeal stuttering behavior and oral stuttering behavior are related. (b) The relationship between oral and laryngeal behavior, although clear in its trend and statistically significant, is not completely consistent. (c) The larynx seems to function more normally during prolongations than during repetitions, tending to be open during part-word repetitions regardless of the voicing characteristics of the sound being repeated.

These studies on the physiology of stuttering clearly support what we have called the weak vocalization hypothesis—there are laryngeal stuttering behaviors. Certainly the work of Freeman and Ushijima (1974; 1978) of Ford and Luper (Note 7) and of Shapiro (1980) make it evident that during stuttering there is or can be excessive and poorly coordinated laryngeal muscle activity. But there is nothing in any of these observations to suggest that laryngeal stuttering behavior differs in any theoretically important way from oral articulatory stuttering behavior, although there have been few direct com-

[8]The results of this study should be qualified in consideration of the marginal interjudge reliability ratings that were reported for the identification of stuttering (.88, .90, .93) and particularly for the classifications of laryngeal behavior (.81, .86, .91).

parisons. Furthermore, these behaviors may occur in nonstutterers as well. Shapiro's (1980) results at least suggest that the characteristics of laryngeal stuttering behavior—excessive tension and simultaneous co-contraction of antagonistic muscle groups—are the same as those of oral stuttering behaviors.

There is evidence, as Adams' (1978) systems discoordination position predicted, that laryngeal and oral stuttering behaviors are related in that disturbances in one system may be accompanied by and possibly cause disturbances in the other. The work of Conture, McCall, and Brewer (1977) and some of Ford and Luper's observations make this clear. But nothing in any of these physiological observations supports the idea that laryngeal stuttering is any more likely to cause oral stuttering than the other way around, nor does anything preclude the possibility that oral and laryngeal stuttering are caused by some other variable. Thus, the weak but not the strong vocalization hypothesis has been supported by these experiments.

Future research in the area of stuttering physiology should continue present work. Our current understanding of stuttering physiology is based on a sample of stutterers that is too small. A second goal should be an increased understanding of the relation between oral and laryngeal systems. Simultaneous measurement of different oral and laryngeal variables, as in Ford and Luper's, and Shapiro's studies, but with enough subjects to permit the computation of correlations among different variables, will enable researchers to develop an understanding of the relations among airflow, muscle activity, and glottal and oral movement. With the alignment of each physiological variable to a constant timeline and the recording of the audio signal, it may be possible to determine in a more mathematically precise way if laryngeal behaviors precede oral ones more often than the other way around.

Experiments Based on the Adaptation Effect

Several experimenters have used the adaptation or practice effect to test the vocalization hypotheses. Brenner, Perkins, and Soderberg (1972) compared different types of practice conditions to see if some were more effective than others in reducing the frequency of stuttering. Twelve stutterers were asked to read four lists of 30 sentences. Embedded in these sentences were 10 test sentences that contained all the sounds of English. The subjects were asked to rehearse the four reading lists in four different ways—silently, silently with lip movement, whispered, and aloud. Three rehearsals were followed by an aloud (memorized) recitation on the fourth trial. Twenty minutes elapsed between conditions. A "no rehearsal" control observation was made by counting the frequency of stuttering on the first reading of the aloud recitation. The four conditions were counterbalanced to control for order effects, but since the no rehearsal condition was taken as the first reading of the aloud rehearsal condition, the no rehearsal condition was not counterbalanced with regard to the aloud rehearsal condition, since it always preceded it. That is, no rehearsal was often preceded by silent, lipped, and whispered rehearsal, but never by aloud rehearsal. A systematic ordering of conditions would tend to raise the frequency of no rehearsal relative to aloud rehearsal. If there had been aloud reading at the beginning of every condition, the confounding influence of this particular order effect could have been avoided. The dependent variable was the total number of stutterings on the fourth trial.

Significantly less stuttering occurred in the aloud rehearsal condition than in any other condition. Because a comparison of the aloud with no rehearsal is the only one that is confounded by order effects, the other comparisons are meaningful. Aloud rehearsal decreased stuttering more than whispered or lipped rehearsal. The authors concluded that "the one feature that distinguished [lipped rehearsal] (LR) and [whispered rehearsal] (WR) is vocalization." But that is not correct. All feedback cues vary in the different types of rehearsal, and one could argue that the degree to which rehearsal is effective depends on the number of feedback cues present. Certainly, feedback cues are increased from silent to whispered and probably also from whispered to aloud rehearsal, but the frequency of stuttering decreases. In addition to the difference in feedback cues, there is a strong possibility that the speech rate and the duration of individual sounds of speech vary in the different rehearsal conditions. Parnell, Amerman, and Wells (1977) showed that the duration of sounds is longer in whispered than in vocalized speech, which probably reflects a slower rate designed to increase the intelligibility of speech. Considering this finding, it is possible that rate-durational characteristics are also different in lipped speech. Consequently, a third explanation is that rehearsal at slower rates, or with altered rate or duration of sounds, is less fluency-enhancing than rehearsal at ordinary rates and with normal duration of sounds.

The experiment that is perhaps most often cited in support of the vocalization hypotheses was conducted by Adams and Reis in 1971 and replicated by them in 1974. In both the original and the replication, 14 adult stutterers were used as subjects. The replication was identical, except different subjects were used. The task was to read a specially constructed passage five times in succession. In the first condition, the passage contained only voiced sounds, the passage having been written for the purpose of excluding voiceless sounds. The second condition was identical except that the subjects read a different passage that contained both voiced and unvoiced sounds. Overall stuttering frequency was significantly less for the all voiced passage in the 1971 study, but there was no significant difference in overall stuttering frequency in the 1974 replication. Further comparison of the original with the replication suggests that the Trial 1 stuttering fre-

quency was higher and the rate of adaptation faster in 1971 than in 1974. There were however, two findings common to the original and the replication: (a) "more than 35% of stutterings . . . occurred at points . . . where vocalization was required" (Adams & Reis, 1974, p. 753), that is, at sentence and clause boundaries, and at unvoiced-to-voiced transitions, and (b) adaptation was more rapid on the all voiced than on the combined passage in both studies.

Several questions may be raised concerning these two common findings. The fact that 35% of stutterings occurred at places where voicing was required is of little value without further information about how many of these places there were. It is impossible to tell if the 35% figure is more than could be expected to occur by chance. However, even assuming that there would be a higher proportion of stuttering at these locations, an assumption that seems partially warranted by the observations of Wall (1977) of a high proportion of stuttering at clause boundaries in children. The results could be attributed to the fact that transitional probabilities and speech rate (both of which covary with stuttering frequency) change at clause boundaries, rather than to voicing adjustments. These objections do not of course pertain to stutterings that occurred at unvoiced-to-voiced transitions within the clause. It would be interesting to see if the percentage of stuttering at unvoiced-to-voiced transitions within the clause was higher than would be expected by chance. Wall (1977), in her study of the distribution of stutterings in 4- and 5-year-old children, did not observe a significantly higher proportion of stuttering at unvoiced-to-voiced transitions within the clause, although this finding may not apply to the stutterings of adults. Recent evidence, to be presented later in detail, suggests that in children at least, stuttered words are less likely to begin with unvoiced sounds (Wall & Pfeuffer, 1978).

The second common finding of the two Adams and Reis studies, the faster rate of adaptation on the all voiced passage, may also be explained in several different ways. Two explanations other than the vocalization hypothesis may be considered: (a) In their attempt to make the passage sound normal despite the fact that it contained no voiceless sounds, the authors may have made the passage syntactically or semantically more simple than the combined voiced and voiceless passage. (b) In the all voiced passage, there are fewer gestures per unit of time, making the reading of it more simple physiologically. Of course, the gestures that have been deleted are all vocal gestures, and it is the fewer number of vocal gestures to which the authors attribute the results. But nothing in the study controls for laryngeal as compared to other types of gestures. It may be that a passage constructed to reduce the number of oral articulatory gestures would have had the same effect. Considering this possibility, an explanation on the basis of fewer gestures (laryngeal or otherwise) seems more appropriate. The finding, when seen in this light, has more relevance for theories of stuttering based on timing or miscoordination than for theories based on explanations of stuttering as a laryngeal disturbance.

A third explanation may be interpreted as supporting either a vocalization or a timing hypothesis. Vowels surrounded by voiceless consonants are shorter than those surrounded by voiced consonants (House & Fairbanks, 1953), so the all-voiced passage, in addition to containing fewer vocal gestures also contains longer vowels. Perhaps, the increased vowel length, rather than the number of adjustments, promotes fluency in the all-voiced passage. Either of these two explanations may be taken to confirm the vocalization hypothesis, but increased vowel length also supports a timing hypothesis. For example, perhaps the extra time in the lengthened vowel makes subsequent or prior consonants easier for the stutterer to produce. In any event, increased vowel length is typically associated with less stuttering, as Wingate's (1976) review has made clear.

Another adaptation study was designed to investigate the effect of deleting oral as well as laryngeal gestures. Adams, Riemenschneider, Metz, and Conture (1974) asked seven stutterers to read three different specially constructed passages. The passages were matched for number of syllables, number of sentences, and for "overall meaning," although the operational definition of this last variable is unclear. The first passage contained all types of sounds. The second passage contained only voiced sounds, both stops and continuants. And the third passage contained only voiced consonants. The dependent variable was the rate of adaptation in successive readings of the same passage.

A portion of this study, the comparison of the first and second passages, may be seen as another replication of Adams and Reis (1971; 1974). In Adams, Riemenschneider, Metz, and Conture (1974), however, there was no concern for overall frequency of stuttering as in Adams and Reis (1971; 1974), only for the rate of adaptation. Although it was not evaluated statistically, it is evident from the results that there was a consistent trend for more stuttering on the all-voiced continuants passage than on either of the other two passages. If the first trial is examined, where one might suppose the special passages to have their greatest influence, the all-voiced, all-continuants passage produced more stuttering than the all-voiced, stops-plus-continuants passage, which in turn produced more stuttering than the voiced-unvoiced, stops-plus-continuants passage. If these differences had been assessed statistically and found to be significant, they would have deconfirmed the hypothesis with regard to gestural simplicity. They are the reverse of the predicted findings. If this result were significant, it would also reverse the finding of Adams and Reis (1971) in which significantly more overall stuttering was found on the combined passage than on the all-voiced passage.

Except for the number of sentences and the "overall meaning," grammatical factors were not controlled in this study. Sentence length and complexity, and transitional probabilities—variables known to influence the frequency of stuttering—may have varied from passage

to passage. In addition, there is reason to believe that these confounding variables varied systematically in the three passages. Both sentence complexity and transitional probabilities would be expected to increase as phonemic constraints become more rigorous. One must select less common words if one cannot select words beginning with stops. Less common words would tend, in turn, to lead to more complex sentences. This bias would cause more stuttering on the third than on the second passage and more on the second than on the first passage, which is what the authors found.

The results with regard to adaptation rate are also interesting. Adaptation rate was faster on the third than on the second passage, but this difference was associated with a probability of .25. The adaptation rate was faster on the second than on the first passage, but this difference was associated with a probability of .08. Neither of these are really acceptable levels of significance. However, the difference between the first and third passages was associated with a probability of .034, which is an acceptable level, and the overall difference for all three passages was associated with a probability less than .001. The hypothesis being tested is that gestural simplicity will affect adaptation rate, and the expected direction of the effect is for the gesturally simpler passages to promote more fluency and consequently to adapt at a faster rate. A close inspection of the data shows that there is a tendency for sharp differences between the first and second trials, followed by a recovery on Trial 3, an unusual adaptation curve. It is possible that the differences the authors observed in the adaptation rate are mostly caused by the large differences between the first two trials, since it is evident that adaptation rate was much slower, negative in some cases, following the second trial. Consequently, it is possible to conclude that gesturally simpler passages result in a faster adaptation rate but not because general simplicity promotes fluency. Quite the contrary, if the study had been controlled, we would have concluded that gestural simplicity promotes *stuttering*, since the tendency is for more stuttering on the passages constructed for simplicity. For some unknown reason this additional stuttering is concentrated on the first trial, which elevates artificially the rate of adaptation for the same passages.

Although it is uncertain what variables might have produced the odd adaptation curve observed in this study, it is worth noting that the specially constructed passages differed in several respects, particularly with regard to aspects of rate and duration. The duration of consonants in initial position increases as word frequency increases (Umeda, 1977), and the word frequencies, as previously noted, would be expected to be different in the three passages. The duration of vowels decreases in longer words (Klatt, 1973), and the length of words in the three passages might have been different. Because less common words could have been chosen to create passages with greater phonemic constraints, differences in timing may have been introduced—longer consonants, and insofar as less common words are also greater in length, shorter vowels. As in the Adams and Reis (1971) study, the fact that vowels surrounded by voiceless consonants are shorter than those surrounded by voiced consonants (House & Fairbanks, 1953), causes vowels in the second and third passages to be lengthened. But vowels are also shorter when surrounded by stops than when surrounded by continuants (House & Fairbanks, 1953), so vowels in the third passage have both a lengthening and a shortening effect. If stuttering is a disorder of timing control, one would expect such unusual timing relations to promote more stuttering. These bizarre timing relations (or some other artifact of the specially constructed passages) may have created the sharp differences between the Trial 1 and Trial 2 frequencies that were largely responsible for the differences in adaptation rate.

The Adams and Reis articles (and by implication the more recent Adams et al. [1974] paper) were criticized in a letter to the Editor of the *Journal of Speech and Hearing Research* by Young (1975). Young took issue primarily with the statistical treatment of the data used by Adams and Reis. He cited the use of medians as measures of central tendency, and noted that some information is lost by using these measures rather than means. Young also criticized the measure of adaptation rate that Adams and Reis had used. Adams and Reis computed adaptation rate by comparing the differences between adjacent or subsequent readings of the same material, so that any difference between two adjacent trials would contribute to the total rate of adaptation. Young suggested that a more appropriate measure would have been the difference between the first and last trials, divided by the total frequency of stuttering on all five trials. Young's measure ignores differences between adjacent trials. Using his own measure of adaptation rate, Young found no differences in the rate of adaptation between the two passages.

Young took issue with several other aspects of Adams and Reis' study. He noted that experimenter bias was not controlled since the frequency of stuttering was computed with knowledge of how the passages were constructed.

Finally, Young recomputed the overall frequency of stuttering using the mean frequencies instead of the medians across all five readings of the two passages and found no difference.

Adams replied to these criticisms by noting that the medians are more appropriate measures of central tendency than the means because medians are not influenced by extreme values. The means, which do use information from extreme scores, would have been misleading as measures of central tendency, in Adams' view.

This argument over means versus medians depends on the a priori assumptions one has about extreme stuttering frequency scores. Are extremely high or extremely low stuttering frequency scores in an experiment a result of error? Or are these scores a part of the phenomenon? If they are part of the phenomenon, should they be allowed to exert heavy or light influence on results? Ex-

treme scores in stuttering are widely seen, and it is clear that they are part of the phenomenon. The distribution of stuttering is skewed, with a few very severe stutterers and many mild ones (Van Riper, 1973a). The question is, should these few severe stutterers carry more weight in determining experimental results than the many mild ones? It seems to me that the question is resolved by supplying information about individual subjects, at least in cases where the significance of results depends on which measure of central tendency is being used. We don't know in this case if Young's failure to reach significance with Adams' data resulted from the use of means, because he made other changes as well. If it did, it suggests that the effect Adams and Reis observed is more typical of mild stutterers than of severe ones, an interesting possibility that could be determined by information about the performance of individual subjects.

Adams also suggested that Young's formula for adaptation rate over-emphasized the frequencies on early trials by subtracting the last trial from the first and by using the total of all trials in the denominator. It was evident to Adams that the difference between the two passages appeared only in the last few readings, and that any measure that placed a heavy emphasis on the early readings introduced a bias that obscured the essential observation. Adams and Reis did, however, see merit in some of Young's objections and based on these they reanalyzed the data using as a formula for adaptation rate the difference between the first and last trials, divided by the sum of the first, fourth, and fifth trials, thus removing the contribution of the second and third trials and giving greater weight to trials in which the difference appeared. A Wilcoxon test between the two passages yielded a result that was significant ($p = .007$). However, this last maneuver seems most questionable. It is not appropriate to remove systematically that portion of the data that diminishes the effect. How do we know that the tendency for adaptation to be slow in early trials in the all-voiced passage was not also due to the number of vocal adjustments? Adaptation rate is the change in stuttering frequency over successive readings of the same material. In their reanalysis, Adams and Reis defined it as changes in frequency in those trials where the changes were most influenced in the expected direction by the experimental variable.

In their reply to Young, Adams, and Reis also expanded on their original statement that 35% of stutterings were related to voice onset requirements. They looked at individual subjects and found that a range of 14-85% of stutterings were related to voice onset requirements, with a median percentage of 39.5. Although the additional information is welcome, it still means very little unless (a) it is also known what percentage of nonstuttered words were also "related to voice onset requirements" in the same way, and (b) there is a control for variables associated with clause and sentence initiation.

Another adaptation study was designed and executed as a challenge to the findings published by Adams and Reis. Hutchinson and Brown (1978) carried out a replication of Adams and Reis's experiment. It was not really a direct replication but a variation in procedures designed to answer the same question. Hutchinson and Brown used a longer passage, a somewhat different definition of stuttering, excluded words of more than three syllables, and had just one reading, rather than an adaptation sequence.

They found significantly more stuttering for the all-voiced passage than for the combined passage, which confirms the same but statistically unevaluated comparison in the experiment by Adams, Riemenschneider, Metz, and Conture (1974). In their discussion, Hutchinson and Brown suggested that the hypothesis is answered better by the frequency of stuttering on the first trial than by the rate of adaptation, since adaptation involves practice, repeated sensory stimulation, and other variables that interfere with testing the hypothesis. It is difficult to argue with this position. In their conclusion, Hutchinson and Brown noted that the all-voiced passage:

> Contained unusual wording necessary to retain voicing and semantic requirements, which occasioned some uncertainty in the stutterers and therefore prompted more dysfluency . . . Therefore citation of this evidence as support for a laryngeal-phonatory substrate to stuttering is extremely hazardous at this time. (p. 153)

In other words, the additional uncertainty caused by the introduction of more bizarre words and phrases to rid the passage of voiceless sounds elevated the Trial 1 frequency.

Consequently, although they say that their design is a better test of the hypothesis than Adams and Reis' because it examines a more appropriate dependent variable, they also say that their design is confounded by an uncontrolled independent variable. To complicate the picture further, it is not possible to attribute, at least not directly, Adams and Reis' results to the effects of word frequency differences, because these differences could only elevate stuttering on the all-voiced passage. Adams and Reis found no Trial 1 difference between the two passages. They did find an overall difference of *less* stuttering on the all-voiced passage in the 1971 version, but that is attributable to the faster rate of adaptation. Since Hutchinson and Brown (1975) and Adams, Riemenschneider, Metz, and Conture (1974) have both found *more* stuttering on the all-voiced passage on Trial 1, one wonders why Adams and Reis, in both of their experiments, failed to observe the same thing. One possibility is that they succeeded in constructing two passages that were matched for word-frequency effects. Or, as evidence to be presented later suggests, if words beginning with voiced sounds are more likely to be stuttered (Wall & Pfeuffer, Note 18), they may have oversimplified the all-voiced passage in their attempt to match it to the combined passage, and by oversimplifying it they may have reduced stuttering enough to cancel the tendency of voiced sounds to be stuttered more.

Hutchinson, McGee, and Deputy (Note 10) repeated the Adams and Reis experiment with children. Using the same passages Adams and Reis used, they asked children in grades 3–7 to read each one five times. Although significant adaptation was observed, the two passages did not differ in the extent to which they promoted adaptation nor in the frequency with which stuttering occurred on them. The authors concluded that the children did not show the same effect as adults because they were more likely to pause to begin with, and as a result, the all-voiced passage did not effectively reduce the number of off-on vocal adjustments.

Although nonsignificant results cannot really be used to support or refute any theoretical position, the results of this study are quite the opposite of what one would expect if Adams' and Reis' results are attributed to syntactic differences between the two passages, and they argue against such an explanation. If the adult stutterers in Adams' and Reis' study were less dysfluent on the all-voiced passage because it was syntactically more simple than the control passage, one would expect the same difference to occur with even greater strength when the readers are children. Since this did not occur when children read the passage, the argument is weakened.

In summary, it seems that despite substantial effort we still do not know if stuttering frequency is influenced by the number of vocal adjustments. At least the question has not been answered by using artificial passages.

One other adaptation study should be described. Bruce and Adams (1978) investigated the relationship between adaptation and vocalization in quite a different way to test a suggestion made by Wingate. He postulated that one of the reasons for adaptation was that repeated production of (practice on) the same syllables makes the stutterer more adept at oral articulation and at coordinating articulation with phonation and respiration. From this theoretical idea, Bruce and Adams predicted that silent or whispered reading would not provide the same amount of practice as aloud reading and would consequently not adapt as much. They cited Peins (1961) as having already shown that silent rehearsal neither facilitates nor impedes adaptation, and they cited Besozzi and Adams (1969) as having shown that there was less stuttering following three silent readings interposed in an adaptation sequence. The study by Brenner, Perkins, and Soderberg (1972) was felt not to be a genuine adaptation study because there was no aloud reading on the first trial of all conditions.

From this background, Bruce and Adams performed the following experiment. They asked eight adult stutterers to read two prose passages that were equated for number of syllables, reading difficulty, and parts of speech. Only 5 seconds elapsed between successive readings of the same material. In the first or control condition, the subjects read aloud for all five trials. In the second or experimental condition, they read aloud on Trials 1 and 5, but whispered during Trials 2, 3, and 4. Twenty-four hours were allowed to elapse between the two conditions. The frequency of stuttering on each trial was assessed.

In the control condition, a typical adaptation effect occurred in which the frequency of stuttering diminished from trial to trial. In the experimental condition, several observations may be made. First, there was significantly less stuttering on the fifth trial, after the three whispered trials, than on the first trial, before the whispered reading, so adaptation occurred even though whispered readings were interposed. Furthermore, the difference between Trials 1 and 5 in the experimental condition was comparable to the difference between Trials 1 and 2 in the control condition, suggesting that about the same amount of adaptation occurred between the two aloud readings, even though three whispered readings were interposed. So, they concluded, whispered readings neither impeded nor increased adaptation. The authors interpreted these findings as follows. Adaptation could not be caused by reduced propositionality (amount of exposure to the material is identical in the two conditions). Furthermore, adaptation could not be caused by reactive inhibition because there was more sensory stimulation and motor responding at the end of Trial 5, condition two, than at the end of Trial 2, condition one, but there was the same amount of stuttering.

The authors also concluded that whispering cannot be used to generate stuttering adaptation, but this conclusion seems unwarranted from the results they obtained. First, the results show a small but observable decrease in the amount of stuttering within the three whispered readings. Although this was probably not a significant rate of adaptation, the fact that decreases from trial to trial were small could be attributable to a "floor effect," that is the changes may have been small not because whispering has no effect on adaptation but because whispering had reduced the frequency of stuttering to a level so low that little change was possible.

Finally, in their discussion the authors said:

> There is considerable direct and indirect evidence that stutterers mismanage speech-related laryngeal adjustments and as a consequence have difficulty in quickly initiating and then maintaining voicing or airflow for speech. This transient problem in starting and sustaining phonation *then leads to breakdowns* in the fluency of oral articulatory movements (Adams & Reis, 1971; 1974; Conture, McCall, & Brewer, 1977) (p. 427). (italics mine)

This quote contains an important error in that the works cited do not document the assertion. Adams' and Reis' study has been questioned on several grounds. Conture, McCall, and Brewer (1977), in an experiment described earlier, found only that there was a tendency for types of oral articulatory stutterings to be accompanied by types of laryngeal activities. They did not demonstrate cause and effect, as the italicized phrase above suggests. Stutterers do demonstrate difficulty in initiating voicing (Adams & Hayden, 1976), but it is not known if this difficulty leads to oral articulatory breakdowns.

A cause and effect relation between laryngeal and oral stuttering may exist for certain stutterings and certain

stutterers. Indeed it would be surprising if laryngeal stuttering did not often precipitate oral stutterings. But there is no reason to believe that laryngeal stuttering precipitates oral stuttering any more than oral stuttering precipitates laryngeal ones. Equally important is the third possibility, that both oral and laryngeal stutterings are caused by a third unidentified variable.

Another quotation from Bruce and Adams' conclusion requires examination:

> The [decrease] in stuttering observed during whispered practice did not carry over to a subsequent reading done aloud. Presumably this was because the physiology of whispering denied subjects the opportunity to practice certain behaviors and coordinations that are requisite to fluent reading aloud, that is, laryngeal valving for voicing and the integration of this activity with respiration and oral articulation. The foregoing interpretation is quite compatible with the increasingly common view that inappropriate laryngeal behaviors are *the immediate cause* of the repetitions and prolongations of articulatory postures and gestures . . . (p. 428). (italics mine)

Although it may be true that the view is becoming increasingly popular, there is still no evidence that inappropriate laryngeal behaviors are the "immediate cause" of stuttering, and of course, the popularity of the belief is not necessarily related to its truth. We still do not know whether a reduction in the complexity of *oral articulation* would also reduce stuttering or whether an interposed condition of laryngeal without *oral practice* (humming prosodic melody?) would also fail to carry over to full reading. Stuttering may be precipitated by laryngeal tensions, and practice in laryngeal valving may assist in adaptation, as shown here, but there is no evidence that stuttering cannot also be precipitated by oral tensions nor that practice in oral gestures does not also assist in adaptation. Note the decreases from Trial 2 to Trial 4 in the whispered condition that take place despite very low absolute frequencies. If these changes are meaningful, practice in oral gestures without voicing can reduce stuttering. Of course, whispering, lipping, etc., are not as effective adaptation conditions as full speech in which all gestures are practiced simultaneously. Neither, I suspect, would be the humming of prosodic melody.

Some other interpretations of these results may also be made. During whispering, consonant and vowel durations are increased. Perhaps adaptation provides practice in syllable timing, and the whispering condition calls for a different set of timing controls. If stuttering is a disorder of slower reaction time or of timing control, these same results would have been obtained. During whisper, the durations and therefore the timing patterns of speech are different because of the changes in laryngeal activity and airflow; therefore, less stuttering would be expected when whispered readings are introduced. Practicing these timing patterns reduces stuttering on them. Returning to a new pattern of aloud speech as on the fifth trial, would change timing again and this pattern would have been practiced only once, so adaptation would be less.

Bruce and Adams' study shows that the adaptation effect is not only specific to the words that are spoken but also to the speech gestures that are used. It is dependent on the phonetic context as well as the semantic context. Changing from voiced to whispered reading alters the gesture much like changing the reading material does. Oral articulatory gestures in whisper are probably not the same as oral articulatory gestures in voiced speech, at least with regard to timing and airflow characteristics. Certainly, we cannot assume that they are. As a result, the differential effects of whispered adaptation readings can be attributed to something other than vocal gesture.

It seems fair to conclude that little has been learned about the relation between stuttering and vocalization by way of the adaptation effect. The major problem has been a poor understanding of the adaptation effect itself. We are not certain how to measure it, or what variables affect it and need to be controlled. Furthermore, adaptation rate is not as directly affected by manipulations on vocalization as is stuttering frequency in a single reading. They are different variables, and this is clear from the different effects the same variable can have on them. In Adams, Riemenschneider, Metz, and Conture (1974), for example, frequency was elevated and adaptation rate reduced by the same procedure.

Experiments with Reaction Time

A number of experimenters have explored the relation between stuttering and vocalization by comparing the vocal reaction times of stutterers and nonstutterers. The chief advantage of a reaction time technique is the simplicity of the response, which experimenters have used to decrease the possibility of stuttering. The study of reaction time has a long history in psychology, and a few studies comparing the reaction times of stutterers and nonstutterers were conducted in the early years of speech pathology. The first investigation of reaction time that sought information about the vocalization hypothesis was done by Adams and Hayden in 1976. Their experiment was prompted by Adams and Reis' (1971) finding that stuttering frequency was lower and the rate of adaptation faster on a passage containing no voiceless sounds. This study was partially replicated in 1973 and has been questioned in a number of different ways, but if the finding is assumed to be correct, several possible explanations may be given for it. Adams and Hayden considered two explanations: (a) tension and constriction in the oral structures during stuttering may make it difficult for the stutterer to achieve phonation, or (b) difficulty in initiating voice may cause the stutterer to repeat and prolong oral articulatory gestures until voicing is achieved. In other words, oral stuttering may precipitate laryngeal stuttering or laryngeal stuttering may precipitate oral stuttering. To test these two alternatives, they measured the vocal reaction times of stutterers and nonstutterers. They reasoned that if stutterers were

slower than nonstutterers, it would be the vocal difficulty that was "primary" and that precipitated oral stutterings, rather than the other way around. There are two major flaws in this design. First, demonstrating that the phonatory mechanism is slower in stutterers does not exclude the possibility that oral stutterings may also precipitate laryngeal ones. Perhaps the oral mechanism is also slower in stutterers. There is no a priori reason why only one of these alternatives can be true. In fact, considering the way the oral and laryngeal mechanisms work together in normal speech, it would be surprising if they did not reciprocally influence each other. The other major flaw in the design was overlooking another possibility. Some third variable, such as a general deficit in timing or poorly lateralized motor control of speech, could cause *both* oral and laryngeal stutterings. There is no necessity for either the oral or the laryngeal mechanism to be primary. Both may be secondary to some other wholly different event.

In any event, to test their two alternatives, the authors asked 10 stutterers and 10 controls to say "ah" immediately on hearing a brief 1000 Hz tone and to stop saying "ah" as soon as the tone stopped. The intervals of time between tones varied, and the tone durations were varied. Thus, two reaction times were obtained for each tone presentation, one for voice initiation and one for voice termination. Both the stutterers and the nonstutterers improved with practice, producing faster voice initiation times (VIT) with repeated practice. The stutterers, but not the nonstutterers, also improved with practice in voice termination time (VTT). But the most important result was that the stutterers were slower than the nonstutterers at VIT on the first and last trials and at VTT on all trials.

The authors were able to discount, to a large extent, three explanations of this result by citing the results of other experimenters. Specifically, they felt that the slower VIT and VTT times did not "evolve out of the [stutterers] long histories of stuttering", that the slower reaction times were not restricted to simple voice initiation tasks, and that they were probably not attributable to a deficit of the auditory mechanism. The authors noted that this slowness may also disrupt the fluency of stutterers' oral articulation, citing Conture, McCall, and Brewer (1977) and Freeman and Ushijima (1975), described earlier.

Another experiment using reaction time, and in part a replication of Adams and Hayden's study, was carried out by Starkweather, Hirschman, and Tannenbaum (1976). In this experiment, 11 stutterers and their controls were asked to respond to a flash of light by saying a syllable as quickly as possible. A wide variety of syllables was used. Syllables that were not produced fluently were recycled and presented later, so none of the reaction times obtained were from stuttered utterances. The reaction time was measured from the onset of the stimulus to the onset of voicing, regardless of interposing orally produced sounds. Thus, if a syllable began with a voiceless consonant, the occurrence of the consonant did not stop the clock. Three trials with each syllable were given.

As in the Adams and Hayden experiment, both stutterers and their controls improved with practice, but the stutterers improved faster. The nonstutterers seemed to reach their fastest speed by Trial 3, but the stutterers were still improving at the third trial. The differences between the stutterers and the nonstutterers was significant for all syllables except CV syllables beginning with an unvoiced consonant and VC syllables containing a voiced consonant. Significant differences between syllable types were found for CV versus VC (the VC's being faster) C_UV versus C_VV (the voiced CV's faster), and meaningful versus nonsense syllables (the nonsense syllables faster). These differences between syllable types were all significant both for stutterers and nonstutterers, but they were caused by using a procedure in which the time from stimulus to voice onset was measured regardless of interposing voiceless consonants. VC's were naturally faster than CV's because some of the CV's were initiated by voiceless consonants, which occupied time. Meaningful syllables were faster than nonsense syllables because more of the meaningful syllables were CV syllables. The exception was that there was no difference between groups for C_UV syllables, but there was a difference for C_VV syllables. To the authors this meant that the extra time "allowed" for voice initiation because of the C_U gave the stutterers, whose voice initiation time was slower, time to catch up. But this interpretation is inferential since the time from the stimulus to the consonants was not measured. The authors gave two explanations for the results: (a) a vocal dysfunction in stutterers that makes them react more slowly than nonstutterers, or (b) a central dysfunction, possibly related to poorly established cerebral dominance (the nondominant hemisphere being known to react more slowly than the dominant one). As in Adams and Hayden's experiment, the explanation based on vocal dysfunction was an overinterpretation of the available evidence because there were no measurements of oral reaction time with which to compare vocal reaction time.

An experiment by Cross, Shadden, and Luper (1979) should also be mentioned. They compared 10 stutterers and matched controls in vocal reaction time to pure tones presented to the left and right ears independently. The response was a simple schwa vowel. They found the stutterers slower than the nonstutterers, confirming the results of other reaction time studies, and this was true regardless of the ear of presentation. The average difference between groups was approximately 80 msec, similar to the difference of 60 msec found by Starkweather, Hirschman, and Tannenbaum (1976) with a visual stimulus, but smaller than the difference of 200 msec found by Adams and Hayden (1976) with a similar auditory stimulus. The absence of a difference between the two ears is not surprising since each ear sends fibers to both hemispheres. Although contralateral representation is greater than ipsilateral representation, this should not affect reaction time the way it affects perceptual and

judgmental, and particularly dichotic tasks. Reaction time depends on the presence or absence of information; perception and judgment depend on the *amount* of information.

Another reaction time experiment, using both visual and auditory stimuli, was carried out by MacFarlane (Note 14). He asked 12 adult stutterers and matched controls to respond in one condition to a green panel light and in another condition to a 1000 Hz tone. Both the tone and the light were matched for durations, which varied randomly. The auditory stimulus was presented independently to the left and right ears. The subjects responded in three different ways by saying /pæ/, by saying /bæ/, or by simple lip closure. The dependent variable was the time elapsed between the onset of the stimulus and the onset of EMG activity in orbicularis oris superior.

The stutterers were slower than the nonstutterers in the auditory task, but in the visual task the difference between the two groups did not reach significance. There were no significant differences among the three different responses. However, there was a significant difference between the two stimuli with the visual stimuli yielding longer RT's than the auditory stimuli for both groups. There was also a significant difference between groups for left ear presentation with the stutterers being slower. In connection with the vocalization hypotheses it should be noted that all of the tasks were oral. One at least (simple lip closure) was purely oral. This means that the slower reaction times of stutterers are not restricted to the vocal mechanism.

Adler (1977) did an experiment that was a follow-up to Starkweather, Hirschman, and Tannenbaum's (1976). Her purpose was to see if slower vocal reaction times in stutterers were (1) attributable to differences in CNS organization, specifically laterality of language function, and (2) characteristic of the oral articulators as well as the vocal apparatus. Twelve adult stutterers and their matched controls were given two tasks. In the first "nonlinguistic" task, the subjects said either "ah" or "sh" when they saw a small area illuminated on a screen. They were told which sound to say in advance, and this information served as a ready signal. Then they responded as rapidly as possible when the stimulus was presented. In this nonlinguistic task the stimulus (area of light) was presented either to the left or right of the screen while the subjects looked at the center of the screen where the ready signal (word) was. Gaze was monitored surreptitiously throughout the experiment, and stimuli on which there were deviations from central gaze were recycled. The stimulus was presented far enough away from the center of the screen to ensure that only the contralateral portion of the retina, and consequently the contralateral hemisphere of the brain, were stimulated. In the "linguistic" task, an identical area of light was presented to the center of the screen and served as the ready signal, while the words "ah" and "sh" were presented to the left and right and served as the stimulus. In both tasks, the stimuli were presented only to one hemisphere, but in the linguistic task, the two stimuli differed linguistically, which required the language hemisphere to be activated before the subject could respond. All subjects adopted a relaxed open-mouth position "as if for 'ah' " and maintained it throughout the experiment. The purpose of this posture was to make sure that the movement used to produce "sh" included mandibular and lingual movement but no voicing, while the movement for "ah" consisted of vocal activity but no movement of the oral articulators. It was understood, however, that the gesture involved in the production of "sh" would probably include glottal opening. The order of conditions was counterbalanced. Mouth-to-microphone distance was held constant throughout the experiment. The time between the onset of the stimulus and the onset of any subject-produced acoustic signal was measured and constituted the dependent variable. A 4-way analysis of variance yielded three significant results:

1. a difference between the tasks with the nonlinguistic task requiring less time,
2. a difference between responses with "sh" requiring less time, and
3. an interaction for groups × response, such that the nonstutterers produced "sh" faster than "ah" but the stutterers did not.

There was no significant difference between the two groups or between the two hemispheres, and no other interaction was significant. The stutterers were consistently slower, but the group difference did not reach significance. That there was no significant differences between the two hemispheres did not mean to the author that the reaction time differences of stutterers must be attributed to some variable other than differences in CNS organization. The interhemispheric reaction time differences in normals were only 10-35 msec in duration, and there was too much individual variation for such a small difference to be detected in this study with an N of 12. The possibility that reaction time differences are a result of poor laterality remains open.

The significant response × group interaction was the only interesting finding of this experiment. "Ah" involves glottal adduction and the initiation of airflow. "Sh" involves glottal abduction and the initiation of airflow, plus mandibular and lingual movement. Although the stutterers were not slower than the nonstutterers in an absolute sense, their speed in producing "sh" relative to their speed in producing "ah" was slower than that of the nonstutterers. Schwartz's theory predicts that the disinhibited PCA should open the glottis more rapidly in stutterers (or prevent the glottis from closing as rapidly), providing they are not stuttering, and in this experiment the opposite was found. So the finding deconfirms Schwartz's theory. The stutterers could also have been slower on "sh" because in this task "sh" was a more complex movement requiring coordination of respiratory, laryngeal, and oral mechanisms. "Ah" involved no articulation. The result consequently confirms Adams' position on systems discoordination in stutterers.

The data then were reanalyzed to remove a possibly contaminating variable, fatigue. In the experiment, there were 96 trials in each task, the number necessary to counterbalance for all possible orders of presentation. It seemed to Adler that the subjects became tired or bored during the experiment and that this slowed their performance. Consequently, in the second analysis of the data she used only the second through the sixth fastest trials of the 24 trials that were repeated in each condition. By this analysis the stutterers were slower than the nonstutterers in the nonlinguistic task but not in the linguistic task. The previous findings of response differences and response × groups interaction were retained in the reanalysis. This reanalysis result meant that in a simple motor task with no perceptual aspect, no judgment, and no linguistic processing, the difference between stutterers and nonstutterers is more evident, which suggests that the reaction time differences are more a motor than a linguistic phenomenon.

Cross and Luper (1979) wanted to test Adams and Hayden's interpretation of the fact that stutterers had slower voice reaction times than nonstutterers. Although they later abandoned the idea, Adams and Hayden originally explained their finding as being produced by faulty or noxious learning experiences. Cross and Luper reasoned that if learning were responsible, the difference between stutterers and nonstutterers should increase with age. On the other hand, Starkweather, Hirschman, and Tannenbaum (1976) attributed their similar finding to a central nervous system deficit, possibly related to hemispheric lateralization. If this were true, Cross and Luper reasoned, age should *decrease* the difference between stutterers and nonstutterers.

Accordingly, they asked 5-year-olds, 9-year-olds, and adults (older than 15) to participate in a voice reaction time experiment. There were nine stutterers and nine nonstutterers at each of the three age groups, and the groups were stratified for sex. The test stimuli were 1000 Hz tones of 1 sec duration, and the interstimulus interval varied randomly from 3 to 6 sec. The dependent variable was voice onset time to produce vowel /ʌ/. No stuttering was judged to have occurred (but see the discussion on p. 10). The results are shown in Table 1.

TABLE 1. Voice reaction times of stutterers and nonstutterers.

	5-year-olds	*9-year-olds*	*Adults*	*Mean*
Stutterers	562	351	300	404
Nonstutterers	483	292	268	348
Differences	79	59	32	56

The difference between stutterers and nonstutterers is smaller for the older groups. The effect of age on both groups was significant at ($F = 80.65$; $df = 2$; $p \leq .0001$), and the age × group interaction was not significant ($F = .67$; $df = 2$; $p \leq .52$). The difference between the two groups was significant ($F = 11.88$; $df = 1$; $p \leq .01$). The authors concluded that the improvement shown by both groups with increasing age was attributable to maturation. Various additional data were cited in support of the contention that this maturation effect on reaction time was neurological. They concluded that:

> since voicing initiation difficulty is exhibited at an early period in the development of motor speech control, the slower voice initiation ability appears to *contribute to* the child's difficulty in establishing fluent speech production rather than *result from* the stuttering problem itself. (Cross & Luper, 1979, p. 74)

Cullinan and Springer (1980) were also interested in investigating the effect of age on vocal reaction time in stutterers. Like Cross and Luper, they wanted to test some of the ideas raised in Adams and Hayden's experiment. They were interested also in the possible effects of frustrative nonreward on reaction time. Their experiment is consequently divided into two parts, one on age, reaction time, and some other variables, and one on the effects of frustrating nonreward on stuttering. The latter portion of the study is not directly related to the vocalization hypotheses, and will not be described here.

Cullinan and Springer observed 20 stuttering children, stratified for sex and ranging in age from 5:8-11:7. Eleven of these children had, in addition to stuttering, articulation-language disorders and/or learning disability. A series of 1000 Hz tones, varying in length from 1.5-4.0 sec were presented with interstimulus intervals varying randomly at 2, 3, or 4 sec. The subjects were asked to sustain /a/ during the tone and to respond as quickly as they could to both the onset and the offset of the tone. Thus, both voice initiation and voice termination times were obtained. The 20 stutterers had significantly slower VIT and VTT than the nonstutterers. However, when the subgroup of stutterers who did not have any other speech-language disorders (the "stuttering only" subgroup) were compared with the nonstutterers, no significant difference was found. When subgroups of different ages were examined differentially, it was found that stutterers younger than eight were not significantly slower than the nonstutterers, while those older than eight were. That suggests that the difference in reaction time between stutterers and nonstutterers actually increased with age, the opposite of the finding by Cross and Luper (1979). A close examination of the subjects used by Cullinan and Springer, however, makes it evident that the presence of differences in severity in the subgroups, both the subgroups divided by age and the subgroups divided by the presence or absence of disorders other than stuttering, confounded the comparison. The authors recognized that the differences observed in reaction time could be attributed to severity, and, to test the hypothesis, they performed an analysis of variance on the response times with severity as the independent variable. A nonsignificant main effect for severity was found in spite of the fact that 12 of 18 comparisons showed that milder stutterers had "higher"

mean response times (by "higher" one must assume the authors meant "faster"). They concluded that, although interaction may be present:

> the . . . analyses suggest that differences between stutterers and nonstutterers in VIT's and VTT's are related to the presence or absence of problems other than stuttering and to age or length of experience with stuttering but not to differences in severity of stuttering (p. 353).

This, however, is an erroneous conclusion. The nonsignificant ANOVA result does not establish that severity did not affect VIT and VTT. Nonsignificant differences can never be the basis for rejection of a null hypothesis (Roscoe, 1975). In fact, with two-thirds of the comparisons in the expected direction, one would expect that with an appropriate statistical test (ANOVA's assumptions of heterogeneity of variance and symmetry of the distribution of the parameter are seriously violated in the case both of severity and reaction time measures) a difference attributable to severity would be found.

Luper and Cross (1978) conducted another experiment to see if the slower reaction times of stutterers, which other researchers had found so consistently, were restricted to the vocal mechanism. They asked nine stutterers and nine matched nonstutterers at each of three age groups—5-year-olds, 9-year-olds, and adults—to press a telegraph key with the finger of their dominant hand in response to an unexpected 1-sec 1000 Hz stimulus tone. A second condition was virtually identical except that the subjects responded by saying /a/.

They found that finger reaction time, like voice reaction time, is slower in stutterers than in nonstutterers. Table 2 displays these results. The difference between stutterers and nonstutterers, and the difference between age groups, were both significant with age accounting for considerably more of the variance ($F = 75.94$; $df = 2$; $p \leq .0001$) than whether the subject was a stutterer or nonstutterer ($F = 6.92$; $df = 1$; $p \leq .01$). A trend is evident in Table 1 for the difference between the two groups to decrease with age, although the group × age interaction was not significant. Perhaps the most important finding was the correlation between finger reaction time and voice reaction time in this group of subjects. For the nonstutterers ($N = 27$) this correlation was +.96; for the stutterers ($N = 27$) it was +.88; for both groups combined it was +.92. Stutterers are slower not only in voice reaction time but in finger reaction time too, and the high correlation between these two measures means that both are probably a function of some common variable, which leads to the prediction that *all* reaction times in stutterers will be slower than those of nonstutterers. This prediction recalls a study performed by Dinnan, McGuiness, and Perrin (1970) in which slower galvanic skin responses (GSR) were found in stutterers than in nonstutterers. Since GSR does not require movement, the reaction time differences between stutterers and nonstutterers result from some very general difference. Luper and Cross concluded that "slower reaction times found among stutterers must be a reflection of a more general timing problem rather than . . . anything abnormal in the phonatory apparatus or in the speech mechanism" (p. 7).

TABLE 2. Finger reaction times of stutterers and nonstutterers, in msec.

	Stutterers	*Nonstutterers*	*Difference*
5-yr-olds	469.41	406.11	63.30
9-yr-olds	288.64	249.52	39.12
Adults	207.51	179.12	28.39
Mean	321.85	278.21	43.64

In another study of reaction time, Prosek, Montgomery, Walden, and Schwartz (1979), observed 10 adult male stutterers and nonstutterers with a mean age of 25:9 years. Two stimuli, a light flash and a pure tone, were presented monaurally. Sixteen different VC words, e.g., *ape, abe, ice, eyes,* were used as responses. In addition, a button-pressing reaction time task was also included. Different combinations of stimuli and responses were compared. Surface electrodes in the laryngeal region were used to assess neural response time in the verbal tasks. The authors found no significant differences between the two groups in manual reaction time, although there was a trend for the stutterers to be slower. In the verbal tasks, there was no significant difference between the two groups when reaction time was measured by EMG surface electrode in the laryngeal region, but there was a significant difference when RT was measured acoustically. The authors concluded that the laryngeal muscles of stutterers are innervated on time, but that poor coordination in the laryngeal region prevents them from responding on time. The finding of significant differences between stutterers and nonstutterers in verbal reaction time confirms the results of a number of different investigators. The two other nonsignificant findings, however, fail to confirm the results of two other experiments. First, they failed to confirm MacFarlane's (Note 14) observation of significant differences between adults in neural response times at the orbicularis oris. Two differences in method could have been responsible for the different results. MacFarlane used nonsense syllables beginning with bilabial sounds as the response, whereas Prosek et al. used actual words beginning with vowels. Recall that Shapiro observed abnormal incoordinations of the laryngeal muscles that failed to produce acoustically observable stuttering behaviors. Since Prosek et al. used real words and MacFarlane did not, it would seem most likely that the stutterers in Prosek et al.'s study should have been slowed more by subacoustic incoordinations than those in MacFarlane's study. It is, however, wise to bear in mind the extraordinary individual variation of stuttering behavior. It could be that the particular stutterers used in MacFarlane's study were more severely impaired than those in Prosek's study, or they happened to be more prone to stutter on nonsense words, or, most likely, they were more likely to stutter

on syllables (even nonsense syllables) that began with consonants. It should be clear that once the possibility exists that these "fluent" productions are less than fluent, a variety of different contaminating variables arise. A replication of these studies, using a procedure to verify the fluency of tokens, would help in solving this question.

It is also possible that the divergence of these two findings occurred because of the location at which the electrodes were placed. MacFarlane placed electrodes on the orbicularis oris, whereas Prosek et al. placed his on the surface in the laryngeal region. It is, of course, questionable what muscle or group of muscles laryngeal region surface electrodes respond to. Most probably, activity in the strap muscles of the neck produce the signals. This raises the possibility that the EMG activity observed by Prosek et al., was partly or wholly associated with respiratory functions or laryngeal descent (Conture, Gould, & Caruso, Note 5) rather than with phonation. Of course, it may also be simply that the difference between stutterers and nonstutterers in reaction time is more pronounced at the lips than at the glottis. This idea was partly confirmed by the significant response × group interaction that Adler observed, in which the difference between stutterers and nonstutterers was greater for a predominantly oral task than it was for a laryngeal one.

Prosek et al.'s failure to confirm the results of Luper and Cross (1978) should also be considered. Luper and Cross found significant differences in finger reaction time between stutterers and nonstutterers. Although the difference was more pronounced for the children than for the adults, the difference for the adults was significant. In absolute terms, Prosek found a difference of 5 msec, on the average, between the two groups, whereas Luper and Cross found an average difference of 28 msec. Because the sample was small in both studies, it is possible that the difference was simply due to sample selection. This is particularly so since there is no way to compare the severity levels of the stutterers in the two studies, and, in any event, the correlation of reaction time with severity is not known. It is, however, unwise to conclude from Prosek et al.'s failure to observe a difference between the two groups in manual reaction time that there is no such difference. Such a difference has been demonstrated.

In another experiment (Hayden & Jordahl, Note 9), an attempt was made to see if stutterer's reaction times were improved under two of the conditions that are known to improve fluency—masking and pacing. Ten stutterers were asked to say /a/ as quickly as possible in response to the onset of a stimulus tone. Only voice initiation time was measured, and there was no ready signal. The tones were presented with a randomly varying interstimulus interval, except in the pacing condition where they were presented at a regular interstimulus interval. VIT was found to be shorter in the two experimental conditions when compared to the control. As far as the pacing condition is concerned, it is hardly surprising that VIT was faster. The fact that the interstimulus interval was regular means that the subjects could anticipate the onset of the stimulus and time their initiation of /a/ to coincide with it. This anticipation characterizes any pacing condition and is presumably responsible for the fluency-enhancement of rhythmic stimulation. It is, however, misleading to think of this as a reaction-time experiment, at least this part of it. The result only demonstrates that stutterers can pace the onset of /a/ the way they (or anyone else) can pace the onset of any response.

The masking condition in this study, however, is comparable to other reaction-time experiments. Here too the authors found more rapid initiation of voicing than in the control condition. Once again, however, it is necessary to recall the observations made by Shapiro (1980) that abnormalities of the laryngeal and oral musculature can and do occur even in the absence of stuttering that is acoustically observable. In Hayden and Jordahl's experiment, where there was no ready signal, and where voice termination time was not measured, contamination by unobservable, stuttering-like abnormalities is particularly likely. The use of a ready signal permits the stutterer to preposition the speech mechanism and thus simplifies the gesture. Presumably, this reduces the likelihood of stuttering. The use of voice termination time has the same effect since the onset of the tone acts like a ready signal, and producing the vocal response positions the apparatus. The offset gesture should consequently be simpler than the onset gesture. Recall that in Adams and Hayden's (1976) original study, VTT was faster than VIT for both groups.

Several other reaction-time studies should be reviewed briefly. Lewis, Ingham, and Gervens (Note 12) replicated the Adams and Hayden (1976) and Starkweather, Hirschman, and Tannenbaum (1976) experiments with 10 stutterers and matched controls. They modified the procedures of these two experiments only by extending the number of trials, and by choosing the response used by Adams and Hayden /a/ rather than the series of syllables used by Starkweather, Hirschman, and Tannenbaum. Both sets of results were confirmed. The performance of subjects tended to level after 12 trials.

Cross and Cooke (Note 6) also replicated the same two experiments with eight stutterers and matched controls, but included a manual reaction time task as a replication of Luper and Cross (Note 13). All three studies were confirmed.

Reich, Till, Goldsmith, and Prins (Note 16) assessed the reaction times of 10 stutterers and their matched controls using a tone as a stimulus and a variety of response modes—button-pressing with left and right forefingers, inspiratory phonation, and expiratory throat-clearing, the vowel /ʌ/, and the word "upper." The stutterers were slower than the nonstutterers for all response modes except left forefinger. There were larger differences for the laryngeal tasks, particularly the word "upper."

Webster and Clark (Note 19) measured speech latencies of 10 stutterers and their matched controls under white noise masking and no noise conditions. In this

study, the syllable to be spoken was presented visually and constituted the stimulus to which the subjects reacted. The results were a significantly slower reaction time for the stutterers under both conditions and a significant difference within the stuttering group between noise and no noise conditions. In this last study, it was not reported that stuttered syllables were discarded, but even if they were, contamination by stuttering-like abnormal muscle activity that did not result in acoustically observable stuttering cannot be ruled out. In these last two studies, the pattern of results—shorter latencies in stutterers under masking and larger between-group differences for vowels and words than for nonspeech stimuli—is the pattern that would be expected if the slower reaction times of stutterers were attributable to abnormal muscle activity of the type described by Shapiro (1980). However, if abnormal muscle activity is responsible for these results, it is probably present in oral articulatory as well as vocal muscles and in nonspeech muscles as well, since significant differences between stutterers and nonstutterers have been found in bilabial, jaw, and finger movements as well as in vocal adjustments.

If reaction times in stutterers are slowed because antagonistic muscles are simultaneously contracting, because muscles are overactive, or because muscles are contracting too soon, perhaps stutterers have a generalized difficulty inhibiting muscular contraction. A child acquiring language with such a disability would find it hard to initiate long strings of syllables and might repeat gestures when simultaneous co-contraction of antagonistic muscles was sufficiently strong to produce a temporary oscillation. Stressful conditions might be expected to raise muscle tonus and to exacerbate the tendency for incoordination. Speech that is artificially slowed or paced might be expected to alleviate the condition. This explanation of reaction-time differences fails to account for the results of Dinnan, McGuinness, and Perrin (1978) in which stutterers' GSR's were found to be slower than those of nonstutterers and no movement was involved. It does, however, explain another curious experiment. Barrett and Stoeckel (1979) asked stutterers and nonstutterers to wink while they were filmed. The authors were able to measure the amount of movement in the nonwinking eye. Stutterers showed more movement in the nonwinking eye than nonstutterers, which suggests that they had difficulty inhibiting contraction of the muscles responsible for closing the eye. A replication of this study and similar studies of other muscle systems seem warranted.

Summarizing the reaction-time studies, some important information about stuttering has been discovered by this technique, although only a small portion of it bears directly on the vocalization hypotheses. Certainly, stutterers are slower than nonstutterers in their ability to initiate phonation. In only two reported attempts Adler (1977) and part of MacFarlane (Note 14) did this comparison fail to reach significance. Furthermore, the effect can be obtained with a wide variety of syllables and with visual as well as auditory stimuli. It is important to note, however, that auditory stimuli seem to generate a greater difference between stutterers and nonstutterers since two of three published attempts with a visual stimulus failed to reach significance. But these findings have little or no bearing on the vocalization hypotheses. Similar differences have been found for oral, manual, and even autonomic responses. In the one direct comparison between laryngeal and oral responses (Adler, 1977), the difference between stutterers and nonstutterers was greater for the oral response. Only this last finding bears directly on the issue of whether stuttering develops as a result of a faulty vocal mechanism, and it deconfirms the hypothesis. Nevertheless, the fact that stutterers' reaction times are typically slower, even in nonspeech activities, and the high correlation between speech and nonspeech reaction times found by Luper and Cross (Note 13) suggest that stuttering may be caused by a delay in the maturation of some aspect of central nervous system (CNS) functioning. Although the possibility of poorly lateralized language functioning has been raised, and even shown to characterize stutterers by other techniques, the relation of slower reaction times to cerebral lateralization has yet to be demonstrated.

Several questions about reaction time seem important. Where physiologically is this difference located? Is it part of neural or mechanical reaction time? The one study designed to answer this question (Prosek, et al., 1979) found a mechanical but not a neural difference. MacFarlane, however, observed a neural difference in a nonlaryngeal muscle. If neural, is the difference sensory or motor or both? Is it caused by slower neural conduction time, slower synaptic transmission time, or to a larger number of synapses? Can the difference between stutterers and nonstutterers be associated with cerebral lateralization? Is the slower reaction-time of stutterers caused by poorly coordinated muscle activity which prevents the structure from moving quickly, as Prosek, et al. concluded?

Finally, all the reaction time research is called into question by the possible presence of abnormal muscle activity that does not result in acoustically observable stuttering. When a noninvasive method of monitoring laryngeal muscle activity is developed, the vocal reaction-time studies will have to be redone. This question can be partially answered by further reaction-time experiments of nonlaryngeal and nonspeech responses in which abnormal muscle activity can be more easily monitored. If the slower reaction-times of stutterers can be shown to be independent of such abnormalities in nonspeech or nonlaryngeal responses, it will be reasonable to conclude, in the absence of direct observation, that the larynx functions similarly.

There is one experiment, however, that does address the question of "subacoustic" stutterings, although with less than satisfying results. The experiment was reported by Ciambrone, Adams, and Berkowitz (1980) and was not designed to answer the question of "subacoustic" stuttering. The authors were interested in determining if the

reduction in stuttering that occurred in an adaptation series could be attributed to motor practice. They reasoned that if adaptation was attributable to motor practice, there should be a positive correlation between the reduction in stuttering during adaptation and the improvement stutterers had been observed to show during repeated reaction-time trials. Consequently, they asked 13 stutterers to read a passage five times in succession and also to participate in 20 trials of a voice reaction-time task. An auditory tone was used as a stimulus, and the production of the vowel /a/ was the response in the reaction-time portion of the study. The extent of improvement in the adaptation task was measured by the difference between the number of words stuttered on the first reading and the number of words stuttered on the fifth and final reading. In the reaction-time tasks, the difference between the first and 20th trials was the measure of improvement. A correlation of +.45 was obtained between the two measures. This approached but did not reach significance (.46 is required with $N = 14$, .51 with $N = 12$, in a one-tailed test).

Studies of Voice Onset and Voice Termination Times

Stutterers' inability to react as quickly as nonstutterers should affect their speech. Timing is important to speech in several ways: Changes in articulatory rate occur at clause and word boundaries, the duration of vowels is an important part of syllabic stress, and the rate of speech varies with the speaker's autonomic arousal. Each of these—clause and word boundaries, stress, and the speaker's autonomic arousal—is also correlated with stuttering. But timing is particularly important as a signal of the voicing feature. In voiced plosive consonants, the plosive burst and the onset of voicing are simultaneous or approximately so, but in voiceless plosives the onset of voicing is delayed by about 50 msec or more. It is an obvious place to look for a difference between stutterers and nonstutterers, either from a timing or a vocalization viewpoint.

Unlike vocal reaction time, voice onset and termination times are measured during continuous speech. Typically, voice onset time is the time between articulatory release of a plosive consonant and the beginning of vocal fold vibration (Lisker & Abramson, 1967). Voice termination time is the time between the end of vocal fold vibration and the beginning of the subsequent consonant (Agnello, 1974). Together, voice termination and voice onset times, along with the duration of the consonant, make up the intervocalic interval. In all of the studies on voice timing, an attempt has been made to eliminate stuttered utterances from the sample, so that the differences observed between stutterers and nonstutterers could not be attributed to instances of stuttering. These studies were consequently tests of the strong vocalization hypothesis.

The earliest of these studies was done by Agnello, Wingate, and Wendell (Note 2). They asked 12 children and 12 adults all of whom stuttered, as well as nonstuttering controls for both age groups, to produce a set of nonsense syllables—/pa/, /ba/, /ap/, /ab/, /apa/, and /aba/. Both voice onset and voice termination times were observed. They found that the VOT's and VTT's for children were longer than for the adults, that VOT's for the stuttering children were longer than for the nonstuttering children, and that both VOT and VTT were longer in the adult stutterers than in the nonstutterers.

But before discussing the implications of Agnello, Wingate, and Wendell's (1974) results it will be helpful to look at another experiment. De Simoni (1974) wanted to see if the influence of a sound on the duration of a sound adjacent to it was similar for stutterers and nonstutterers. Several effects of phonetic environment on duration are known—(a) vowels surrounded by voiced consonants are longer in duration than those surrounded by voiceless ones, (b) vowels surrounded by continuants are longer than those surrounded by stops (House & Fairbanks, 1953). Similarly, the duration of continuant consonants is influenced by the vowels surrounding it. To see if these effects were true for stutterers, De Simoni asked six adult stutterers to produce a series of nonsense syllables—/pip/, /sis/, /bib/, /ziz/, /pap/, /bab/, /sas/, /zaz/, /isi/, /isa/, /asa/, and /asi/. The duration of the vowel in the first eight syllables and the duration of the /s/ in the last four syllables were measured. Tokens judged to have been stuttered were discarded. De Simoni found that the effects of phonetic environment are the same in stutterers, but they are greater in degree. The tendency for vowels followed by fricatives to be longer was twice as great in the stutterers as in the nonstutterers. Similarly the tendency for vowels followed by voiced consonants to be longer was twice as great for the stutterers as for the nonstutterers. For /s/ the environmental effects were the same between the two groups on Trial 1 but different between the two groups on Trial 2, although in all cases, the stutterers' /s/'s were longer.

Klatt (1974) has suggested that sounds tend to lengthen or shorten as a percentage of their inherent duration, and, although this suggestion was intended to explain the effects of neighboring sounds rather than durational differences attributable to characteristics of the speaker (such as his being a stutterer), the explanation may hold in such a case too. It seems evident that stutterers' consonants at least are longer than those of nonstutterers, since voice onset time is an aspect of the consonant. Maybe stutterers' vowels are also longer. Being "inherently" longer, stutterers' sounds may be more affected by subsequent sounds than those of nonstutterers. This interpretation is by no means certain, but the regularity of the differences in vowel length and the fact that the stutterers' sounds were affected in exactly the same way as the nonstutterers' suggests it. One fact is evident, the "fluent" vowels and consonants of stutterers in nonsense syllables are longer than those of nonstutterers. If vocalization were harder to initiate

and terminate, as the vocalization hypothesis and the reaction-time experiments suggest, the stutterers' vowels should be as long as the nonstutterers' but delayed, lagging in VOT and VTT. Instead they are lengthened in CVC syllables. Furthermore, the consonants are also lengthened. These results suggest that stutterers move their oral and laryngeal mechanisms more slowly during "fluent" speech than nonstutterers do, so that articulatory rate in general is decreased. It is, however, necessary to challenge these results. It is not certain that the acoustically fluent tokens were free from muscular abnormality. Evidence bearing on the question might come from an assessment of the *variability* of stutterers' "fluent" tokens in comparison with the variability of duration in nonstutterers' speech.

The most important question, however, is do stutterers move their speech and voice mechanisms more slowly because they are stutterers, or are they stutterers because they move their speech and voice mechanisms more slowly? The former alternative implies that the stutterers might have learned that they must talk slowly in order to keep from stuttering. Although possible, this explanation is unlikely in a nonsense syllable task. The latter alternative implies that slowness of movement is a cause of stuttering. If repetitions of word parts and other stuttering behaviors can be demonstrated to develop as a result of the difficulty of learning language with a mechanism that cannot move quickly, such a relation would be acceptable. Of course, it may also be that stuttering and slow movement both arise from some common variable.

Another experiment (Hillman & Gilbert, 1977) was designed to see if the VOT and VTT results found by Agnello, Wingate, and Wendell (Note 2) with nonsense syllables would also be found with continuous speech. Hillman and Gilbert asked 10 adult stutterers and their matched controls to read the Rainbow Passage aloud following one rehearsal. Intervocalic intervals associated with the consonants /p,t,k/ in unstressed environments were measured to determine the duration of VOT. In addition, syllabic rate was also measured with pauses greater than 250 msec excluded.

The syllabic rate was 4.26 syllables per second for the nonstutterers and 4.06 syllables per second for the stutterers. The mean difference in VOT between the stutterers and the nonstutterers was 9 msec; the stutterers had the longer VOT's. Furthermore, for both groups, VOT increased as the place of articulation moved back in the oral cavity, and for both groups the differences between consonants were the same. These results are shown in Table 3.

TABLE 3. Average VOT for stutterers and nonstutterers on three consonants, in msec.

	/p/	/t/	/k/
Stutterers	38	46	54
Nonstutterers	28	37	45

The authors suggested that the differences between the two groups might be attributed to reduced subglottic air pressure and weak airflow. However, an earlier study, which is described in the next section, found that stutterers had higher rates of airflow (Adams, Runyan, & Mallard, 1975). A simpler explanation is that the stutterers moved their oral and laryngeal mechanisms more slowly, and this accounted for the slower rate and the longer average VOT.

An experiment conducted by Kerr and Cooper (Note 11) combined elements of the VOT and reaction-time techniques. They asked 15 adult stutterers and their controls to sustain the production of a continuant until signalled, and then to produce a different sound. Three trials were given, but only the third was measured. Three different types of transitions were examined, and there were four sounds in each transition type. The actual stimuli used were as follows:

1. Voiced-to-voiceless transition: a-s, a-f, a-θ, a-ʃ
2. Voiced-to-voiced: a-z, a-v, a-ð, a-ʒ
3. Voiceless-to-voiced: s-a, f-a, θ-a, ʃ-a

The duration of the first sound was varied randomly within a range of .5-3 sec. The time from the signal to the change in the response was the dependent variable. The stutterers were slower, and the difference was significant. However, differences between the three tasks were not significant. The most obvious conclusion is that the stutterers were slower than the nonstutterers at moving the oral articulators, since all three conditions involved movements of these structures. Furthermore, the group by task interaction was significant, with the group difference being greater for the VL-V than for the V-VL condition. The stutterers, although slower at both tasks, showed a greater deficit in initiating vocalization than in terminating it. This asymmetry between the VOT and VTT differences has been seen before. Agnello, Wingate, and Wendell (Note 2) failed to find a significant difference between child stutterers and nonstutterers in VTT although the difference for VIT was significant. Adams and Hayden (1976), however, found no significant difference on one VIT trial, but all VTT trials were significantly different in a reaction-time experiment. Perhaps the CV nonsense syllable, because it is more like a stressed syllable of actual speech, evokes greater delays. Perhaps in the subacoustic sense Shapiro (1980) described, these subjects are stuttering. Freeman and Ushijima (1978) also observed events typically associated with stuttering, such as nonreciprocity of antagonistic muscles, during speech in which no stuttering occurred. Although this may mean that some fluent speech shows nonreciprocity, it may also mean that stutterings can occur in a form too mild to be observed easily. All of the results of experiments on the fluent speech of stutterers, as with the simple reaction-time experiments, may be called into question by subacoustic stutterings. The reaction-time experiments escape this criticism only partially because the same group differences

have been found in manual reaction times and these have been correlated with speech reaction times. But the "fluent" speech of stutterers is questionable. One solution to the problem is a "correlation control" in which reaction time measures on nonspeech tasks, such as finger reaction time, are correlated with voice onset time or whatever aspect of speech timing is being compared. If, in addition to a significant difference between stutterers and nonstutterers in VOT, there is a significant positive correlation of VOT with finger reaction, then it would be clear that VOT was related to a nonspeech or general physiological trait and not simply a by-product of "subacoustic stuttering." That would not be the case of course if abnormal muscle activity that is stuttering-like is also found to occur in nonspeech muscle systems. Experimentation to confirm or deconfirm this idea is needed.

In the case of Kerr and Cooper's experiment, the slowest condition (VL-V) is the only one that calls for a transition from a consonant into a stressed vowel, that is, where a "beat" in continuous speech would fall and stuttering would be most likely to occur. It is regrettable that the authors did not include a V-V condition in which the first sound was a voiced continuant (e.g., z-a) so that a comparison between voiced and voiceless transitions into stressed vowels could have been made.

Another study of VOT was carried out by Metz, Conture, and Caruso (1979). They asked five adult stutterers to produce a series of English monosyllables, all of which were composed of stop consonants or stop consonant clusters joined with various vowels. Only fluent productions were examined. The dependent variable was VOT. Separate durations for the burst of noise associated with plosive release (frication) and for the noise of aspiration were also measured in the syllables that began with /t/ and /k/.

They found the stutterers to be slower on six of the test items (/p, br, pr, dr, tw, and b/) but not on the other 12 (/b, d, g, t, k, tr, kr, gr, pl, kl, gl, and kw/). Using an index of determination, they found that variation in VOT was predictable from knowledge of group membership (stuttering versus nonstuttering) only for /p/ and /tw/. For the other syllables, it was not possible to predict VOT from this knowledge. They also found that the aspiration duration for /tw/ was significantly slower in stutterers, but otherwise there were no significant differences for aspiration and frication between the two groups. There was also no difference between voiced and voiceless transitions. The authors concluded that the "off-on" hypothesis, first described by Adams and Reis (1971), was probably too simplistic an explanation of what occurs during intervocalic intervals and that laryngeal adjustments are made for all types of transitions, voiced to voiced, as well as voiced to voiceless and voiceless to voiced. The last point is well taken. However, the number of subjects in Metz, Conture, and Caruso's study is so small that it is difficult to assess its generality. It is perhaps surprising that any significant differences at all were obtained. On the other hand, when multiple comparisons are made, one expects some of them to be significant by chance.

Starkweather and Myers (1979) did an experiment to find out where within the intervocalic intervals (IVI) this additional time was located, given that stutterers' IVI were longer than those of nonstutterers. They asked 14 adult stutterers and controls matched for age and sex to read the first paragraph of the Rainbow Passage. The IVI /əsʌ/ in "the sunlight" was chosen for analysis, and stuttered tokens of this phrase were discarded. Playing the tape at one-quarter the original speed, they made a 160-4K spectrogram and an amplitude display aligned with it for time. On the spectrogram, they located eight landmarks that identified the beginnings and endings of subsegments within the IVI. The eight landmarks were:

1. The beginning of the decline in vocal intensity.
2. The end of voicing.
3. The beginning of frication.
4. The end of the rise in frication frequency.
5. The beginning of the decline in frication frequency.
6. The end of frication.
7. The beginning of voicing.
8. The end of the rise in vocal intensity.

The duration of intervals between these landmarks were then measured. Significant differences were found for three of the subsegments and for the total IVI. The three subsegments that were longer in the stutterers' speech shared the characteristic of a change in frequency or intensity or both, but the subsegments that were not different between the two groups (with one exception) were steady states in which neither frequency nor intensity changed. The authors noted that changes in frequency and intensity can be produced only by movements of the oral and laryngeal mechanisms, whereas the duration of steady-state subsegments can be changed only by differences in timing (coarticulation). Consequently, they attributed the stutterers' longer IVIs to slower movements of the speech and voice mechanisms, not to differences in timing.

As with other studies of the fluent speech of stutterers, these differences in subsegments of the IVI may be the result of "stuttering" events (poorly timed or excessively tense muscular activity) that are neither visible nor audible but that nonetheless slow the stutterers' rate of movement. It is not immediately clear why subacoustic stutterings would affect rate of articulatory movement without disturbing coarticulation. In fact, two types of stuttering, silent and audible prolongations, are clearly lengthened steady-states, but the possibility of subacoustic stuttering remains. A "control" condition, in which speed of movement of a nonspeech activity is measured would get at this question. If this measure were found to be correlated with IVI duration, it would mean that IVI duration was related to general physiology, not just speech, and by implication it would not result in stuttering. Of course, the possibility exists that stuttering-like abnormalities could slow nonspeech as well as speech movements.

The most serious objection to the study by

Starkweather and Myers (1979) is that the information it provided was derived from only one IVI, and it is not clear that other IVIs would show the same trends. Further examination of the subsegmental durations of other IVIs should be made before implications for stuttering theory are drawn.

One experiment related to the VOT studies should be mentioned briefly. Healey (1980) examined fundamental frequency of eight male stutterers and matched controls as they produced test words in a sentence frame. Words judged to have been stuttered were discarded. The stutterers changed their fundamental frequency more slowly than the nonstutterers and had lower average fundamental frequency than the nonstutterers. The first result would be predicted from the many other results indicating that stutterers move their speech mechanisms more slowly than nonstutterers, but the second result is contrary to prediction. One would expect heightened tonus in the laryngeal muscles to raise fundamental frequency. Perhaps, male stutterers learn to compensate for this tendency and lower their voices. This study, like all the studies on the fluent speech of stutterers, is confounded by the possibility that the "fluent" speech that was examined may have contained words that were produced with abnormal muscle activity. The presence of this activity could certainly explain the slower changes in fundamental frequency, but it is not clear how these abnormalities could explain lower fundamental frequency.

Zimmermann (1980) examined productions of the words *mom, pop,* and *bob* that were judged to have been fluently produced by six stutterers and seven nonstutterers. The two groups were not matched for age or sex, and in fact they differed substantially on both of these variables. The mean age of the stutterers was 39, that of the nonstutterers 26. The nonstuttering group consisted of four males and three females, but all of the stutterers were males. The speakers were asked to repeat the three words 10 times at a "comfortable rate." Tokens judged to have been stuttered were discarded, as were tokens that contained "descriptively aberrant movement patterns, such as repetitive movements or prolonged posturing at a consonantal position" (p. 97). This procedure is more likely to reject tokens that contain abnormal muscle activity, but it cannot be certain that the tokens were entirely free of abnormal muscle activity as a result of the procedure.

Lateral view cinefluorographic films of the subjects were made during the utterance, and movements of the lower lip and jaw were monitored. Several measurements were made: (a) the time from the beginning to the end of a movement (the transition time), (b) the time between the end of the downward movement into the vowel and the beginning of the upward movement out of the vowel, (the steady state), and (c) the time between the beginning of the downward movement and the onset of voicing (voice onset time).

The stutterers were found to be slower than the nonstutterers in all three of these measures. The mean differences between stutterers and nonstutterers on these three measures for movements of lip and jaw in producing the three tokens are shown in Table 4.

TABLE 4. Mean differences between stutterers and nonstutterers in measures of articulatory movement.*

	Transitions		*Steady States*		*Voice Onset Time*	
	Jaw	*Lip*	*Jaw*	*Lip*	*Jaw*	*Lip*
Bob	20	25	52	48	30	34
Mom	58	31	39	12	−2	7
Pop	45	52	8	−10	11	31
Sum	123	108	99	50	39	72
Mean	41	39	33	17	13	24

*Derived from data in Zimmermann (1980).

It is apparent that the slower movements Zimmermann observed in stutterers were present in the oral articulators (jaw and lip transition times) as well as in the laryngeal movements participating in voice onset time. If anything, the differences were smaller for laryngeal than for oral movements. Of course, any comparison between laryngeal and oral movements is of questionable validity because the two systems are closely related physiologically and they function synergistically.

However, the differences between stutterers and nonstutterers were possibly a result of uncontrolled differences between the stuttering and nonstuttering groups reflective to age and sex differences, or the presence of undetected abnormal muscle activity in the stutterers. The first of these two possibilities is extremely likely. With increased age beyond maturity, speech rate declines (Starkweather, 1980), so it is entirely possible that the stutterers in this experiment simply spoke more slowly than the nonstutterers because they were older. Sex too may have influenced the results. Although not clearly identified as faster or slower speakers, females and males may differ on some variables related to speech rate (Starkweather, 1980). Perhaps more important, however, females and males respond differently in any experimental situation according to the sex of the experimenter, females tending to try harder than males to produce results which they believe the experimenter anticipates (Littig & Waddell, 1967; Rosenthal, 1966). The experimenter's anticipation of results may be communicated in many ways before or during the experiment, even in prerecorded instructions (Adair, 1973). Regardless of the means by which the contamination may have occurred, it is evident that the possible differential performance of males and females was not controlled in this study. When asked to produce syllables at a "most comfortable rate," females may have tended to speak faster. Since there were more females in the nonstuttering group, where the faster speech was observed, the failure to control sex confounds the results.

In another related experiment, Hand and Luper (Note 8) examined the intervocalic intervals of stutterers and nonstutterers. Ten male stutterers and controls matched

for age and sex repeated a carrier phrase containing one of eight different nonsense VCV syllables consisting of the sounds /b/ and /g/ combined in all possible ways with the vowels /i/ and /u/. Tokens judged to have been stuttered were discarded. The durations of the steady-state portions of the two vowels and the transition times into and out of the consonants were measured spectrographically. Tokens judged to have been stuttered were discarded. The authors found that the stutterers had longer vowel steady states but *shorter* transition times than the stutterers. At first glance, this experiment seems to contradict both Zimmermann's (1980) and Starkweather and Myers' (1979) results. Zimmermann (1980) found longer steady states and longer transition times. Starkweather and Myers (1979) found longer transition times but no significant difference in steady-states. All three of these experiments are potentially contaminated by two sources of error, sampling error and the intrusion of subacoustic abnormal muscle activity. The presence of unusually high muscle tension or poorly timed muscle action potentials may have been responsible for any of the observations of lengthened subsegments in the stutterers. This kind of contamination may even explain Hand and Luper's (1980) finding of faster transition times in the stutterers, since as the authors interpret their results, the faster transition times are an attempt to compensate for the slower steady-states.

Sampling error is also a factor to consider. The sample size has been extremely small, Zimmermann, 6; Hand and Luper, 10; Starkweather and Myers, 14. Furthermore, since stuttering is known to take highly individualized patterns with regard to which words or sounds are "difficult," it may be that these three studies drew samples that tended to show subacoustic muscle abnormality in different ways or on different sounds or words. In this way, sampling error may account for the different results.

If we suppose, however, that the results of these three studies are not contaminated by sampling error or subacoustic muscle activity (replications may prove this to be the case), it is worthwhile to see if their apparent discrepancies can be resolved. To make this comparison, it will also be necessary to set aside temporarily the fact, more serious in my view, that Zimmermann's study was confounded by differences in sex and age between the experimental and control groups. In comparing the methods of the three studies, it is clear that they dealt with different speech tasks, and the differences among their results may be attributable to the differences in task. Zimmermann (1980) looked at CVC syllables for which the consonants were a voiced and voiceless plosive and a nasal. Hand and Luper (1980) looked at a VCV sequence, in which the consonant was a voiced plosive. Starkweather and Myers (1979) looked at a VCV sequence in which the consonant was a voiceless fricative.

Considering first the findings on steady states, Zimmermann (1980) and Hand and Luper (1980) both looked at vowels, whereas Starkweather and Myers (1975) looked at a fricative. The discrepancy with regard to steady states, then, is not very real. It may be that the duration of steady state portions of vowels is longer in stutterers than in nonstutterers while at the same time the duration of steady states in frication is not. Or, it may be that future experimenters will observe a difference in the steady-state portions of fricatives as well as those of vowels. After all, Starkweather and Myers' (1979) failure to observe a difference in steady-state portions of a fricative should not be given the weight of an observation. The absence of evidence for a phenomenon is not the same as evidence for the phenomenon's nonexistence.

With regard to transitions, Zimmermann (1980) and Starkweather and Myers (1979) found stutterers slower than nonstutterers, whereas Hand and Luper (1980) found stutterers' transition times to be faster than nonstutterers'. The discrepancy between results is much more real for the transition times than for the steady states. This difference, however, may be attributable to the different sounds used in the experimental tasks. Starkweather and Myers (1979) examined transition into and out of a fricative, but Zimmermann (1980) and Hand and Luper (1980) both looked at transitions into and out of plosives. The voicing feature seems not to have mattered because Zimmermann's subjects had slower transition times for both voiced and voiceless sounds. It should be remembered, however, that these results may be attributable to sex or age differences; consequently, the difference between voiced and voiceless transitions in stutterers and nonstutterers has not yet been effectively assessed. But the distinction between plosives and fricatives may be important. To achieve a plosive that is within phonemic limits, a rapid release of the articulatory position is required. No comparable restraint exists for fricatives. Maybe stutterers move slower into and out of fricatives, but speed up in producing plosives as they approach the required rapid release. That is, the tendency to speed up for the required rapid movement away from the consonant may spread to the movement approaching the consonant. This explains the discrepancy between Hand and Luper's (Note 8) and Starkweather and Myers' (1979) results, but does not explain Zimmermann's (1980) observation of slower transition movements into and out of plosives. To explain these results, and still retain the preceding explanation, it is necessary to fall back on the possibility that Zimmermann's (1980) results were confounded by age and sex differences.

In summary, the experiments on VOT and VTT, like those on reaction time, have not provided much information about the vocalization hypotheses but they have nonetheless been fruitful. The fact that the IVI's of stutterers are longer than those of nonstutterers is well established, but it is equally clear that this additional length is not attributable solely to the vocal apparatus. VOT and VTT in continuous speech are determined not only by vocal but by articulatory and respiratory movements. There is no evidence yet that the stutterers' slower IVI's result from a dysfunction of the vocal system any more than any other system, and in fact there is

some evidence (Kerr & Cooper, Note 11; Starkweather & Myers, 1979; Zimmermann, 1980) that the oral articulatory movements are also slower and contribute to the difference. None of these experiments have been specific enough about physiology to reach any conclusion about the relation between stuttering and vocalization. Furthermore, the duration of VOT and VTT reflects two different aspects of speech rate, the speed of articulatory movement and the extent of coarticulatory overlapping. In three experiments (Hand & Luper, Note 8; Starkweather & Myers, 1979; Zimmermann, 1980) a distinction was made between these two variables, but discrepancies between the results and the presence of design errors require further experimentation before conclusions can be drawn. In short, the VOT and VTT experiments, like the reaction time experiments, have failed to answer the question about vocalization, but they have led beyond vocalization to ask more general questions about timing and duration in the speech of stutterers.

As with reaction time, future research in VOT/VTT will depend largely on the development of a noninvasive technique for monitoring abnormal muscle activity. In addition, the discrepancies between experiments on the steady states and transition times of syllables in stutterers will need to be resolved empirically. An experiment comparing transition times for voiced and voiceless fricatives with those of voiced and voiceless plosives would be a first step. Once it is clear, how the fluent syllables of stutterers differ from those of nonstutterers, assuming "fluency" can be established independently of acoustic judgments, a next step will be the observation of the same effects in children and the comparison of the effects between stuttering children and stuttering adults.

Another area of future research is the correlations between speech and nonspeech reaction times on the one hand and the timing variables of syllables on the other, particularly in the speech of both stuttering and nonstuttering children. Speech involves rapid, continuous movement, and many movements must be initiated at a point in time that depends on the occurrence of some other movement. Under such circumstances the ability to react quickly should affect speech production, at least in children. In adult speech, reaction time may be less important because the speaker places more reliance on preprogrammed sequences of movement (Borden, 1979; Borden & Starkweather, Note 4). Even in the adult, however, reaction time should affect speech in some way. Future experimenters should focus on the correlations between reaction time and variables such as syllabic rate, coarticulation, consonant and vowel durations, pause time, and steady state and transition times, in the speech of children and adults. Another important line of investigation will be the correlations between reaction time and aspects of stuttering. An obvious question is the correlation between reaction time and stuttering severity. So many variables contribute to stuttering severity that a complete answer to the question will likely come only from a complex multivariate analysis, but partial and valuable answers can be found in more limited experiments. For example, two of the most obvious variables of stuttering severity, frequency and duration of stuttering events, could be correlated with reaction time. Further investigation of the neurological basis for stutterers' slower reaction times is also in order.

Finally, two important contaminating variables to the relation between stuttering and reaction time need to be ruled out. One is the presence of subacoustic muscle abnormalities. The other is anxiety or arousal level. Autonomic arousal slows reaction time, and it has also been related to stuttering (Brutten & Shoemaker, 1967) although only theoretically. Nevertheless, the fact that many stutterers say that they stutter more when anxious is reason enough to control this variable. A first step would be the replication of one reaction time experiment with an independent measure of autonomic arousal. A second step would be the comparison of stutterers' and nonstutterers' reaction times in high and low arousal conditions. Even if the reaction time measures are found to depend on autonomic arousal, the relation between reaction time and stuttering is no less interesting. It may be that stuttering is more likely to occur in high arousal conditions because arousal slows down reaction time. This is an unlikely possibility, but it should be explored.

A Study Using Feature Analysis

Wall and Pfeuffer (Note 18) sought to specify the relation between stuttering and vocalization in an entirely different way. They reasoned that if the vocal mechanisms of stutterers were impaired in some way, as Schwartz (1974) maintained, and if this impairment manifested itself at moments in the utterance where voice onset or termination was required, as Adams and Reis (1971; 1974) had suggested, then the voicing feature, by its presence or by its absence, should predict stuttering. Furthermore, since adult stutterers may have learned to avoid words containing these transitions, the question is answered with less possibility of contamination by examining the speech of very young stutterers. Accordingly, 11 4- and 5-year-old children who had been identified by certified speech-language pathologists as stutterers were engaged in a free-play situation while their speech was recorded. The experimenters determined the words on which stuttering occurred and then counted the frequency of each distinctive feature of the first and also of the second sounds of those words. A sample of nonstuttered words, distributed evenly throughout the same speech samples, was obtained by a random procedure and the frequency of each distinctive feature on the first and second sounds of these words was also counted. Finally, the two sets of frequencies of each feature were compared between the stuttered and the fluent words, and this was done both for the sounds in first and those in second position. There was no significant difference in the frequency of the voicing feature in stuttered and nonstuttered words. The first sounds of stuttered words

contained significantly higher frequencies of tense, low, syllabic, and continuant features than the first sounds of nonstuttered words. The second sounds of stuttered words contained significantly lower frequencies of the round and continuant features and a significantly higher frequency of the consonantal feature than the second sounds of nonstuttered words. On further analysis, this distribution of features on the first and second sounds seemed to be caused by the fact that consonants and vowels often reciprocate, and by the fact that vowels were more often than consonants the first sounds of stuttered words. Two specific vowels (/æ/ and /aɪ/ were stuttered more often than any others, and most often these vowels initiated words, typically "I" and "and," that occurred at clause boundaries. Since it had already been determined independently, using these same transcripts, that stuttering was more likely to occur at clause boundaries (Wall, 1977), the authors concluded that syntax or semantics, or both, determined the location of stuttering more than distinctive features.

Since vowels are all voiced and there were clearly word effects increasing the frequency of vowels, the authors looked at the frequency of the voicing feature in words beginning with consonants. Here it was found that stuttered words began with voiceless consonants (/h/ was excluded because it has no voiced cognate) significantly less often than nonstuttered words. This was the opposite of the prediction that is most easily derived from the vocalization hypotheses. If stutterers have difficulty initiating voicing, one would expect stuttered words to begin with voiceless consonants, which require both voice termination and initiation, more often than they begin with voiced ones, which require neither termination nor initiation of voicing except at the beginnings and ends of utterance. But instead, the authors found a significant difference in the opposite direction.

The authors felt their results meant that the frequency of stuttering in young children is more predictable by grammatical than by phonetic characteristics, but when grammatical effects are excluded, voiced rather than unvoiced sounds are more likely to initiate stuttered words. This may be taken either to confirm or deconfirm the vocalization hypothesis, depending on which of the two predictions is made. The first prediction is that made by Adams and Reis (1971) that stuttering should occur more often on voiceless sounds where rapid laryngeal movements are made. This was the prediction the experiment was designed to test, and it was deconfirmed. However, one could also argue that, if voicing is difficult to achieve or maintain, words beginning with voiced sounds should be stuttered more often, and this might be true even though in continuous utterance voicing for most words is already underway. I think it can be taken as a weakness of Adams' vocalization hypothesis that it lacks the specificity to generate an unequivocal prediction on such a simple matter. Schwartz's theory is more specific. Glottal closure should be more difficult than glottal opening for stutterers. However, despite this extra specificity, Schwartz's theory also fails to make an unequivocal prediction. Words beginning with voiced sounds require neither opening nor closure of the glottis in continuous utterance, whereas words beginning with voiceless sounds require both opening and closure. So, no really clear test is possible.

Experiments on Fluency Enhancement

Wingate, in his review of the conditions that enhance fluency, suggests that these conditions change the stutterer's manner of speaking, specifically his vocalization, and they change it by emphasizing vowels and subordinating consonants. A number of investigators have tested this idea by looking at the effects of fluency enhancement on stutterers' speech. The earliest of these studies was carried out by Adams and Moore in 1972. They asked 12 adult stutterers to read a prose passage aloud under two conditions. In the first, 90 dB SL white noise was presented bilaterally during the reading. In the other no noise was presented. Four different measures were taken:

1. palmar sweat index (the amount of sweat present in the finger tips during an interval of a few seconds),
2. the number of stutterings,
3. the subject's vocal intensity and
4. the subject's total reading time.

Under noise, as expected, fewer stutterings occurred and vocal intensity was raised, but there was no significant difference between the two conditions for total reading time or for palmar sweat. The authors felt these results meant that masking does not reduce stuttering by lowering anxiety or arousal, but they were uncertain whether rate-reduction or raised vocal intensity was responsible. The rate of articulation could still have been slowed (as part of the Lombard Effect). Because there were fewer stutterings in the noise condition, the overall reading time was shortened, and this could have happened even though rate was slower, leaving overall reading time unchanged. It was consequently not possible to tell whether increased vocal intensity or slower articulatory rate was responsible for the effect of masking noise.

A more direct test of the effect of vocal intensity on fluency in masking noise was made by Garber and Martin (1977). Eight adult stutterers, trained in monitoring their own vocal intensity, talked under two different noise conditions, quiet and 100 dB SPL bilateral white noise. They also talked at two levels of vocal intensity, normal and loud. "Loud" was arbitrarily defined as 12 dB above normal.

There was no significant difference in the frequency of stuttering between normal and loud vocal levels, but there was significantly less stuttering in noise than in quiet. Furthermore, the fewest stutterings occurred when the subjects read at normal vocal loudness but in noisy conditions. Apparently, the extent of the difference between masking noise and vocal intensity determined how much stuttering would be reduced. That is, the less

able the stutterers were to hear themselves the less they stuttered. This is certainly an explanation that is derivable from the results. Another explanation, however, is that articulatory rate, which ordinarily slowed in the Lombard Effect, would be slowed most in the condition with the least amount of auditory feedback, since the slowed rate is probably an attempt to increase intelligibility. The number of words per minute (WPM) increased in the noise conditions and they increased more when vocal intensity was normal than when it was loud, but this could have been caused by the decreased stuttering. Articulatory rate may still have been reduced in the noise conditions and further reduced when vocal intensity was normal than when it was loud. The results are as attributable to rate reduction as they are to reduced auditory feedback.

The effect of masking noise still cannot be explained unequivocally, but all that is needed is another factorial experiment, like Garber and Martin's, but in which the actual rate of articulation is measured, independent of stuttering effects. A simple technique is to cut stuttering out of the tape and measure the length of what's left. However, more information is obtained when the duration of consonants, vowels, and pauses are measured spectrographically and compared with their durations in unmasked speech.

Lechner (1979) examined several aspects of voicing in stutterers and nonstutterers during masking and delayed auditory feedback. She asked 15 adult male stutterers and their matched controls to read the Rainbow Passage six times under conditions of normal, masked, and delayed auditory feedback. One sentence was extracted from the second, fourth, and sixth readings, and played back at one-eighth the original speed. Measurements were made of the average fundamental frequency, the total number of inflections (pitch changes without pause), the number of upward and downward inflections, and the number of total, upward, and downward pitch shifts (pitch changes following a pause in vocalization). She found that the stutterers had more downward inflections and that they read more slowly. Both groups read with higher fundamental frequency under masking and under DAF. DAF, in addition, tended to produce more pitch shifts and inflections. The two groups were compared on the percentage of vocalization time under the three conditions and no between-group differences were found. However, some of the group × condition interactions were interesting. For the stutterers, the proportion of time spent producing voice was the same in the different feedback conditions, but the nonstutterers read with a smaller proportion of vocalization in DAF than in the other two conditions. The author interpreted this interaction as suggesting that DAF caused nonstutterers to prolong silence more than vocalization but caused the stutterers to prolong both vocalization and silence. Although interesting, these results do little to explain why stutterers speak more fluently under conditions of altered auditory feedback. Some changes in manner of vocalization were noted, such as the greater number of downward inflections and the higher fundamental frequency, but the relation of these changes to the enhancement of fluency was not demonstrated, nor were the changes in vocalization distinguished from changes in rate. It would be a simple matter to reanalyze the data, computing the correlation between rate change and fluency change, and between vocalization changes and fluency change, to see which of the two effects were more predictive of fluency.

Several recent studies have focused on the effect of whispering on stuttering. Whispering is particularly important because it is clearly a change in the manner of vocalization and is undeniably effective in reducing stuttering in most stutterers, but it is unlike the other fluency-enhancing conditions in that there is a reduction of vocalization in whisper while in the other fluency-enhancing conditions vocalization is enhanced. It is a major challenge to discover what whispering has in common with other fluency-enhancing conditions. Although it is true enough to say that whispering and the other fluency-enhancing conditions have in common a change in the manner of vocalization, this is an unsatisfactory explanation of how they have the same effect on stuttering. How can changes of opposite direction affect stuttering in the same way? This objection would be removed if a common characteristic of whispering and other fluency-enhancers could be found that was also related to voicing. One possibility is a change in some aspect of airflow. Airflow is interesting because it is an aspect of speech that is related to all three systems—respiration, vocalization, and articulation—and may consequently reveal discoordination among these systems.

Adams, Runyan, and Mallard (1975) examined several measures of airflow during whispered and voiced speech in stutterers and in nonstutterers. Six adult stutterers and matched controls read a list of 12 CVC words, voicing the words in one condition and whispering them in the other. Four measures of airflow just outside the mouth were taken: (a) the initial rate associated with plosive release at the beginning of the syllables, (b) the final rate associated with articulatory closure at the end of the syllable, (c) the peak-to-peak volume, which was the volume of air expended between initial and final airflow peaks, and (d) the amount of time that passed between one airflow peak and the other.

In both voiced and whispered speech the stutterers had higher airflow rate and volume and slower articulatory rates than the nonstutterers. It is possible that the higher airflow rate could have resulted from the slower articulatory rate, given a constant subglottic air pressure. Also, in the whispered condition, the nonstutterers produced the CVC's faster than in the voiced condition, but the stutterers did not show this difference. This faster rate in whispered nonsense syllables is unlike what occurs in whispered communication where the rate of articulation is typically slower (Parnell, Amerman, & Wells, 1977). Finally, airflow rates for both groups were greater in whispering than in voiced production, but the peak-to-peak volume was much greater for the stutterers.

An interpretation of these results not made by the authors is that stutterers have slower articulatory rates than nonstutterers, even when not stuttering. When the conditions call for whispered CVC's, which the nonstutterers produce faster than voiced CVC's, the stutterers are unable to increase the rate of their production further. Since more time elapses while air flows, the volume of airflow is greater for the stutterers. Articulatory rate seems to be different between the two groups to begin with and is changed differentially by whispering. This suggests that the fluency stutterers achieve in whispered conversational speech may be due to the decreased articulatory rate that is usually used in whisper.

Perkins, Rudas, Johnson, and Bell (1976) sought to determine if the fluency-enhancing effects of whispering were attributable to rate reduction or to simplification of glottal gesture. Simplification of glottal gesture is another candidate for the characteristic shared by all the fluency-enhancing conditions. It is clear without argument that glottal gesture is simplified in whisper, but to demonstrate that it was this variable that was responsible for its fluency-enhancing effects, the authors tried to simplify glottal gesture even more than in whisper by asking their subjects to "talk" without airflow. They asked 30 stutterers, all of whom were known to stutter during whispering, to read a 130 syllable excerpt from the Rainbow Passage, a different excerpt for each of the three conditions. In one condition, the subjects whispered, in a second they read normally with full voicing, and in a third they made the oral movements of the words in the passage without airflow, in other words, they "mouthed" or "lipped" the passage. Order effects were partially counterbalanced, with seven subjects reading the passages in voiced, lipped, whispered order, 11 in lipped, whispered, voiced order, and 12 in whispered, voiced, lipped order. As a result, more subjects had the voiced condition later in the experiment than had it earlier, so any order effect should have had the effect of producing less stuttering in the voiced condition. A post hoc test for order effect was carried out, and no significant difference attributable to order was observed. Of course the possibility remains that an order effect not potent enough to reach significance contaminated the results. However, the predicted direction that such an effect would have was the opposite of the results that were observed.

An attempt was made to measure the rate of articulation, independent of the effect of reduced stuttering on the number of WPM. To do this, someone listened to a tape recording of the subjects and pressed a switch that activated a timer. This switch was held down during speech. When the subjects stuttered, the key was held down for what was estimated to be the "equivalent" of a syllable as it would have been produced without stuttering. By this technique, the authors hoped to subtract the time spent stuttering from time spent speaking. This is not really a measure of articulatory rate but a measure of syllables per minute with the estimated durations of stutterings (partially) subtracted. As measured, the rate of speech increased significantly from voiced to whispered to lipped speech. Stuttering occurred more frequently in whispered than in lipped speech. The increases in rate in these conditions are less impressive. Although articulatory rate was not really measured, the authors probably eliminated most of the time taken up by stuttering. Some doubt must remain, however. Since in normal speakers rate is decreased in whisper, it is puzzling why it was not decreased here. Do stutterers actually speed up their articulatory rate in whisper? Did the authors really eliminate stuttering from the rate measure? Could they reliably measure rate and the frequency of stuttering in lipped and whispered speech?

The authors concluded that the differences among the three conditions were caused by changes in the complexity of phonatory adjustments, these being simpler in whispered than in voiced and simpler yet in lipped speech. However, a number of other potential independent variables were not controlled. Auditory cues were different in the three conditions. The durations of consonants and vowels may have been changed in the whispered and the lipped condition. Even if stuttering was eliminated from the rate measure, it is possible that the vowel durations were shortened and the consonant durations lengthened, or vice versa.

Another type of rate change could also be responsible for the results. The rate of speech in syllables per minute results from the combination of two factors, the speed with which the articulators move and the extent to which adjoining gestures are overlapped (coarticulation) (Gay, 1978). If only one of these two factors is slowed in a fluency-enhancing condition, the other can speed up, within certain limits, to adjust for it.

Finally, the amount of information transmitted is diminished to zero in the lipping condition and may have been diminished slightly in the whisper condition, particularly if rate was increased.

Despite these alternative explanations, it seems quite likely that simplifying the activities of one speech system can reduce the amount of stuttering. Such a simplification, as in this case for the vocal apparatus, reduces the number of gestures per unit of time, and this may be true even when rate in syllables per minute has increased. However, simplifications in the oral articulatory mechanism could have the same effect. As the authors said, "The fact that oral articulatory and respiratory discoordination disappeared when phonatory complexity was simplified . . . only demonstrates the possible causal role of phonation. It does not eliminate the alternative that articulatory and respiratory processes could serve the same role" (p. 518). Unfortunately, the authors go on to cite the study by Adams and Hayden (1976), which was described earlier and in which the authors observed that the vocal reaction times of stutterers were slower than those of nonstutterers, as evidence that *only* phonation could be causative because the stutterers in Adams and Hayden's study showed slower reaction times of voicing even when not stuttering. However, Adams and Hayden's study demonstrated only that stutterers' vocal

mechanisms reacted slowly during fluent, simple productions. They did not eliminate the possibility that the oral mechanism might also be slower in stutterers during fluent, simple productions. In fact, other studies described earlier (Adler, 1977; MacFarlane, Note 14) have shown this to be the case, and Luper and Cross (Note 13) have shown that nonspeech reaction times are also slower in stutterers than in nonstutterers. Apparently, a more general deficit in the capacity to react quickly slows both vocal and articulatory reaction times as well as nonspeech reaction times in stutterers. The high correlations between finger reaction times and vocal reaction times observed by Luper and Cross (Note 13) suggest that it is indeed the same process that slows both finger and vocal reaction times, and it is not unreasonable to assume that this process also slows articulatory, respiratory, and other systems.

If stutterers are unable to move their speech and voice mechanisms as quickly as nonstutterers, and if this deficit causes stuttering behaviors to occur, it follows that a reduction in the number of gestures per unit of time, whether in the vocal, articulatory, or respiratory mechanisms, will enhance fluency.

Another fluency enhancing condition that has been tested directly is choral speaking; Adams and Ramig (1980) were interested in seeing if, during choral speech, stutterers actually showed the longer vowels that Wingate had suggested were responsible for the fluency-enhancing effects of speaking this way. They asked 10 stutterers and matched controls to read a short passage in unison with a prerecorded tape and, in a counterbalanced control condition, to read the same passage alone. Acoustically observable stutterings were discarded, and the remaining segments were analysed spectrographically. Three dependent variables were measured—vowel duration, vocal sound pressure level, and sustained vocalization across word boundaries—as three ways vocalization might be "emphasized," to use Wingate's term.

The results neither confirmed nor deconfirmed Wingate's explanation. The stutterers' vowel durations were significantly longer than the normals in both conditions. That is, they were longer to begin with, which suggests that the stutterers had a slower rate of articulation. The stutterers also had significantly lower vocal SPL than the nonstutterers, in both conditions. The only change that occurred in choral speech was that the stutterers shortened their vowel durations significantly in order to match the model's rate. There were no differences between conditions in vocal SPL or in continuity of vocalization.

Although Wingate's explanation of fluency-enhancement in choral speech was not confirmed in this experiment, neither can the effect be attributed to rate-reduction because the model the stutterers spoke with was prerecorded and because their vowel durations were shorter in the more fluent condition. The explanation lies elsewhere, possibly in the fact that choral speech, like shadowing, may be a simplified speech task because the speaker is not responsible for the rhythm and timing. However, before completely abandoning rate change as an explanation of these results, it would be wise to measure the durations of consonants and pauses and the amount of coarticulation in the two conditions. Considerably more understanding of fluency-enhancement might have been achieved had the authors obtained correlations between fluency changes and the vocal parameters that they measured.

Another fluency-enhancing condition with obvious connections to the vocalization hypothesis is pitch change. Many stutterers report that they can speak without stuttering by talking in a high- or low-pitched voice. In a previous study, Healey (1977) had found that stutterers and normal speakers both reduced speech rate when asked by increasing the duration of phonetic elements and by increasing pause time. Ramig and Adams (1980) asked four groups of speakers, two groups of nine stutterers and two groups of nine nonstutterers matched for age and sex, to read a passage that was specially constructed to contain a few CVC words in which the vowel was located between two voiceless fricatives, e.g., *fish, sauce*. There were three conditions: Habitual speech, reading in a high-pitched voice, and reading in a low-pitched voice. Sentences judged to be fluent on an acoustic basis were selected and measured for rate spectrographically. Vowel durations and intersentential pause times were specifically measured. All four groups showed fewer nonfluencies in low and high pitched speech than in the habitual reading condition, as shown in Table 5. Furthermore, all four groups spoke slower (only the "fluent" utterances were counted) in the changed-pitch conditions, as shown in Table 6. Both vowels and pause times were lengthened in most subjects, and no particular strategy was discerned for either group. Unfortunately the duration of consonants was not measured. As designed, this experiment provides little evidence on the question of why stuttering is reduced during speech that is produced with abnormally high or low pitch. The effect may be due to the changes in vocalization or the changes in rate. A replication of this experiment or a reexamination of the data including an assessment of the correlation between the extent of pitch change and the extent of fluency change, along with a similar assessment of the correlation between rate reduc-

TABLE 5. Mean number of nonfluencies for different levels of vocal pitch.*

	Habitual	*Low*	*High*
Children			
Normals	1.4	.22	.66
Stutterers	3.0	.87	1.4
Adults			
Normals	.55	.0	.44
Stutterers	5.2	.77	1.6

*From Ramig and Adams (1980).

TABLE 6. Rate of speech for different levels of vocal pitch, in syllables per second.*

	Habitual	*Low*	*High*
Children			
Normals	4.5	3.7	3.8
Stutterers	4.1	3.9	3.8
Adults			
Normals	4.4	4.0	4.1
Stutterers	3.9	3.1	3.5

*From Ramig and Adams (1980).

tion and stuttering reduction, would provide helpful additional information.

Singing is one of the most powerful of the fluency enhancers, and Wingate suggested that it was the emphasized vocalization in singing that was responsible for the effect. Colcord and Adams (1979) were interested in testing this hypothesis, and they saw two ways vocalization could be emphasized—by increasing vocal sound pressure level and by lengthening the vowel durations compared with unsung speech. They asked eight stutterers and their matched controls to read aloud a set of altered lyrics to the song "Home on the Range," and then to sing the lyrics using the familiar melody. The experimenters listened to the subjects' productions and extracted fluently produced words for analysis. Pairs of sung and spoken words were compared, but in no case was the same pair fluently produced by all subjects. Five different word-pairs were used, and at least four pairs of subjects produced each word-pair fluently. The duration of voicing was measured by observing the onset and offset of the vertical striations that characterize a periodic signal on a spectrogram. In a reliability check of the duration measures, 88% of the measurements were within 20 msec of each other. This measurement procedure and the reliability with which it was executed seem less than totally adequate. Although measuring the duration of voicing by observing the onset and offset of the vertical striations in a spectrogram is the usual method, it should be questioned whether this is precisely the right way to measure it when, as in this case, it is the vocal gesture rather than the acoustic product that is of primary interest. The beginning of the gesture can typically be discerned as a change in the frequency/intensity characteristics of the preceding segment. For example, in the sequence "the fair," (*fair* was one of the words analyzed by the authors), the frication associated with /f/ begins to decrease in frequency and intensity, then stops altogether for a brief period of aspiration before voicing actually begins. This movement away from the consonantal articulation, it can be argued, begins the vocal gesture. The gesture is completed when full voicing is achieved. After a period of full voicing, a second gesture is made to perform the transition to the next segment. The authors are, of course, entitled to measure whatever they want to, but it seems reasonable that experimenters with an interest in stuttering will concern themselves with the movements into and out of voicing as well as the duration of the vowel itself.

The second point in connection with the measurement of vowel duration concerns the reliability. It is questionable that the measures were indeed reliably made when only 88% of the measurements were within 20 msec of each other. Although the steady-state portion of the vowel is considerably longer than this (100-150 msec), differences have been observed in the transition period alone (Starkweather & Myers, 1979; Zimmermann, 1980), which is typically not much longer than 10-20 msec. It is consequently the case that as many as 20% of the measurements differed between the two judges by an amount that was equal to or greater than the entire transition time.

The results were that the control group showed longer voicing duration, higher peak SPL, and higher average vocal SPL in singing than in speech. For stutterers, the results are shown in Table 7. The stutterers showed the same amount of change in the duration of voicing as the nonstutterers did. The authors rightly describe the stutterers' concomitant changes in voicing duration and frequency of dysfluency as possibly coincidental. After all, it is difficult to sing without increasing vocal duration. However, they go on to interpret them as something more than coincidence: "... These ... results ... could be interpreted to mean that the stutterers extension of voicing duration was partially or solely responsible for their reduced dysfluency during singing" (p. 476).

TABLE 7. Dysfluencies and vocal parameters in singing and reading, by stutterers.*

	Frequency of Dysfluencies	*Voicing Duration*	*Peak Voc. SPL*	*Average Vocal SPL*
Reading				
M	11.50	226.40	73.85	70.43
SD	13.94	9.85	.40	.37
Singing				
M	1.88	308.35	73.69	70.97
SD	1.36	14.93	.38	.34
	Significant	Significant	Nonsignificant	Nonsignificant

*From Colcord and Adams (1979).

These data could have been made considerably more meaningful by two additional procedures. First, if the duration of nonvocal speech segments were also measured, both groups of subjects might have lengthened not just vowels but also continuant consonants during singing, as is typical. Although this might appear to weaken the case for lengthened vocalization as an explanation of fluency enhancement in singing, it might also have turned out that consonants were less lengthened in stutterers or not lengthened at all. The results would certainly have been more meaningful in either case. Second, the possibility that the results are merely coin-

cidental, which weakens the experiment severely, could be removed by simply computing the correlation between the duration of vowels and the frequency of stuttering. Although a high positive correlation between the two measures would not demonstrate causality, it would demonstrate that the two variables are related and not merely coincidentally concomitant. Finally, it must be noted again that it is not possible to be certain that the tokens were entirely free of stuttering-like abnormalities of muscle activity on the basis of judgments made from the acoustic product. In this experiment, the authors noted that the stutterers had longer voicing durations than the nonstutterers in the reading condition as well as in the singing condition. It may be that a few of the tokens were "stuttered" subacoustically and that these lengthened segments slowed the group average.

An interesting experiment by Martin and Haroldson (1979) explored relations among different fluency-enhancing conditions. They asked 20 stutterers to speak for 50 minutes on topics suggested by word cards. The first 20 minutes was a base-rate portion. During the second 30 minutes, one of five experimental conditions was administered: 5 seconds of time-out contingent on stuttering, presentation of the tape-recorded word "wrong" contingent on stuttering, continuously present 250 msec DAF, continuous bilateral 100 dB SPL white noise, and continuous 90 beats/sec metronome. Appropriate instructions accompanied each of these conditions. Changes in both the percentage and the duration of stutterings from the base rate to the experimental segments were the two observed variables. All of the conditions were effective in reducing stuttering percentage, and all but the noise condition was effective in reducing stuttering duration. For purposes of understanding the laryngeal behavior of stutterers, the most interesting results were in the correlations among the fluency-enhancing conditions. It will be helpful for our purposes to ignore the "wrong" and TO conditions. Those conditions certainly enhance fluency but probably not by changing the stutterer's manner of speaking as DAF, noise, and metronomic pacing probably do. The correlations among these three fluency enhancers for percent stuttering and duration of stuttering are shown in Table 8.

These results suggest that the reduction in a stutterer's frequency of stuttering caused by DAF is related to the change caused by noise and to the change caused by rhythmic pacing, but that rhythmic pacing and noise are not related. In the case of stuttering duration, only DAF and pacing are related. All three of these conditions change laryngeal behavior, but they do so somewhat differently. Noise and DAF raise vocal intensity, whereas pacing typically does not. Similarly, DAF and pacing alter timing and prosody dramatically, but noise has a relatively small effect on rate. In stutterers, of course, these generalizations may not all hold. I am tempted to argue that the stronger relation between DAF and pacing compared to the slightly weaker relation between DAF and noise and the lack of a relation between noise and pacing suggests an explanation of fluency enhancement based on general timing changes rather than a specifically laryngeal explanation, timing or otherwise. But this argument is weakened by the fact that there may be laryngeal changes in pacing independent of rate change. We simply do not know. The strong relation in the duration measures between pacing and DAF in the absence of such a relation between noise and DAF or between noise and pacing also suggests the possibility that a general timing explanation may be a better account than a specifically laryngeal one. This argument, however, is not compelling. In fact, there is not enough information in these results to support or refute the idea that laryngeal changes are responsible for fluency enhancement. What is needed to answer these questions is the correlations between fluency change and changes in different aspects of speech, such as vowel and consonant duration, pause time, coarticulation, vocal SPL, stress contrast reductions, and perhaps others, such as respiratory events, phrase lengths, and muscle activity levels and coordination.

TABLE 8. Intercorrelations among fluency enhancing conditions.

	DAF		*Metronome*	
	%	*Duration*	*%*	*Duration*
Noise	.60*	−.39	.35	−.22
DAF	—	—	.64*	.60**

*$p \leq .05$.
**$p \leq .01$.

In summary, the experiments on fluency enhancement have done little to explain why these conditions produce sometimes dramatic but temporary reductions in stuttering. Metronomic pacing, one of the most important fluency-enhancers as far as theoretical implications are concerned, has not been examined carefully for its effects on respiratory, vocal, and articulatory movements in stutterers and nonstutterers. Of the studies that have been done on masking and DAF, it seems clear only that the effect of masking is greatest when the subject is least able to hear himself but is not caused by anxiety reduction or by increased vocal loudness. Both DAF and masking raise fundamental frequency and increase the number of pitch changes, but the relation of these vocal changes to stuttering frequency has not been assessed. Rate changes have not been controlled or carefully observed. The experiments on whispering have not been conclusive. The effect could be attributable to changes in vocalization, particularly simplification of vocal gesture, but it could also be attributable to simplification of gesture (not exclusively vocal), possibly even to rate reduction, or to differences in the number of feedback cues.

Altering vocal pitch and singing both reduce speech rate by lengthening speech segments, but it is unclear whether vocalized segments are lengthened more than nonvocalized ones, and even if they are, it is not clear if

this is more true for stutterers than for nonstutterers or more than merely coincidental with changes in the frequency of dysfluency.

It seems evident that the effect of choral speaking is not attributable to rate reduction because stutterers are more fluent even when they speak more quickly in this condition. However, it is not clear either if vocalization is emphasized in choral speaking. The possibility remains that choral speaking, like metronomically paced speech (and perhaps singing), is physiologically simpler than speech in which the talker takes sole responsibility for the rhythm and timing of utterance.

One of the major contributions of Wingate's review was to point out that understanding why the fluency-enhancing conditions reduce stuttering would be an important key to our understanding of the disorder. Wingate was also right in focussing on the effects these conditions have on speech, rather than distraction, propositionality, and other esoteric explanations. He may have erred in specifying vocalization before enough evidence was available. That evidence may yet be obtained, but other explanations of fluency-enhancement should also be considered. Future research in this area should look closely at the effects these conditions have on the speech of nonstutterers as well as stutterers, particularly on the duration of consonants, vowels, pauses, and on coarticulatory overlap. Furthermore, at least one other major fluency-enhancer should be examined, and that is repeating the same material (adaptation). To date, experiments with adaptation have seen it as a dependent, not an independent variable. As these studies have become more advanced, physiological variables should be measured along with acoustic output.

Explanatory Inadequacies of the Vocalization Hypotheses

No review of the relation between stuttering and vocalization would be complete without an accounting of facts that are known about stuttering that the vocalization hypotheses fails to account for, or accounts for only weakly. Chief among these facts are those related to the localization of stuttering in continuous speech. A number of linguistic variables have been identified as predicting stuttering. The problem is that these linguistic variables seem unrelated to vocalization.[9] It will be useful in reviewing these facts to consider an alternative to the vocalization hypotheses specifically that stuttering in continuous speech occurs where there is a change in the rate of articulation. It should be recognized at the outset, however, that this is not a particularly powerful hypothesis because the rate of articulation changes continuously. Nevertheless, it changes more abruptly at certain locations, as indicated by variation in the duration of speech sounds. It is known, for example, that longer words are more often stuttered than shorter ones, even when embedded in continuous utterance (Brown & Moren, 1942; Soderberg, 1966), and it is difficult to understand how such a fact could be accounted for by any of the vocalization hypotheses. However, it is known that if a stressed vowel is in the first syllable of a two-syllable word (e.g., /ɪ/ in "bitten") its duration is 30% less than with the same syllable in a monosyllabic word (e.g., "bit") (Klatt, 1973). In order to shorten the vowel for the longer word or lengthen it for the shorter word, an adjustment in the rate or timing of articulation is required.

Similarly, content words are stuttered more often than function words (Brown, 1938; Hejna, 1955), at least in adults. Children may reverse this tendency (Bloodstein & Gantwerk, 1967), or their stuttering may be determined more by grammatical than by word factors (Wall, 1977). The rate of timing of articulation is also affected by a word's grammatical function. Stressed vowels and consonants that introduce stressed syllables are longer in content than in function words in adult speakers (Umeda, 1975). It is hard to see any relation between the content/function word distinction and vocalization.

Stuttering also occurs more on less frequently used words, provided the sentences are syntactically simple (Ronson, 1976). Timing is changed too with frequency of usage. Stressed vowels and consonants introducing stressed syllables are longer in words that occur less frequently in the language (Umeda, 1975). Of course, all of these effects—length, the content/function distinction, and word frequency effects—may be variations of the same variable, although it is by no means certain just what that variable is. In any event, changes in articulatory rate and/or timing, as well as in stutterings, are known to occur at these locations. It is difficult to see how the length of a word in continuous utterance, its categorization as a content or function word, or its frequency of usage can be related to vocalization.

Stuttering has long been observed to occur on stressed syllables (Froeschels, 1961), and although there are clearly vocal changes associated with stress—both intensity and frequency are altered—note that there are timing and durational changes associated with stress as well. Vowels are shortened considerably by unstressing (Umeda, 1975). In fact, unstressing is a phenomenon of increased rate of speaking (Gay, 1978). Consonants are also shorter when they introduce an unstressed syllable provided they are in word-initial or mid-word position (Umeda, 1977).

Stuttering occurs much more often at the beginnings of words than in mid-word position, and rarely occurs at the ends of words, even during continuous speech when the beginning of the word may not coincide with the initiation of voicing. There are, however, substantial duration effects related to word boundaries. Consonants are longer at the beginning of a word than at the end, and even shorter in the middle (Klatt, 1974; Oller, 1973; Umeda, 1977), but vowels are lengthened at the ends of words (Umeda, 1975). Because vowels contribute to syl-

[9]For a different point of view, see St. Louis (1979).

lable length more than consonants, syllabic duration is increased at the ends of words, but shortened at the beginning. Clearly, a complex adjustment in the rate of articulation is necessary at word boundaries, since vowels are relatively longer and consonants relatively shorter at the ends than at the beginnings of words.

Consonants are stuttered more than vowels in adult speech (Brown, 1938; Hahn, 1942; Quarrington, Conway, & Siegel, 1962; Taylor, 1966). In children, however, syntactic variables may be more important than the class of the speech sound (Wall & Pfeuffer, Note 18). This aspect of the distribution of stuttering can be clearly related to vocalization because only consonants may be voiceless, but it seems not to be the potential voicelessness of consonants that causes them to be stuttered more because stuttering is likely to occur more on words initiated by voiced consonants than on words initiated by voiceless ones (Wall & Pfeuffer, Note 18). Consonants are of course shorter than vowels, so that a timing hypothesis helps explain this phenomenon.

There is more stuttering on longer sentences than on shorter ones (Tomick & Bloodstein, 1976), a fact that may be explained by the vocalization hypotheses; stuttering results from some additional difficulty in establishing vocalization for a sustained utterance than for a brief one. The idea certainly has merit but it remains to be tested. It has been established, however, that syllables are produced faster in longer utterances than in shorter ones (Jones, 1948; Lindblom, 1968; Malécot, Johnston, & Kizziar, 1972). The relation between length of utterance and syllabic rate was also found in the speech of 6- and 9-year-olds, but not in that of 3-year-old children by De Simoni (1974a), although Menyuk and Klatt (1975) found faster VOT values in longer utterances of 3- and 4-year-olds, as well as adults.

Meaningful speech yields more stuttering than nonmeaningful speech (Bloodstein, 1950; Eisenson & Horowitz, 1945), and stuttering is more likely to occur at points of high uncertainty or high information load (Taylor, 1966), facts that are difficult to account for with the vocalization hypotheses. Several facts, however, implicate information or uncertainty as a variable affecting the rate of speech. Rate slows just before grammatical boundaries and speeds up afterwards where uncertainty rises then falls, at least insofar as rate can be inferred from changes in the duration of sounds (Umeda, 1975). Umeda (1975) also found that stressed vowels are shortened in words that occur repeatedly in the same material. Repeated words would be more predictable and consequently less uncertain.

Of the nine variables known to predict the location of stuttering in continuous speech—word length, grammatical function, word frequency, syllabic stress, position in the word, the consonant/vowel distinction, sentence length, information load, and clause boundary—only three (syllabic stress, sentence length, and the consonant/vowel distinction) can be related to vocalization. And of these three, one (the consonant/vowel distinction) seems not to predict stuttering the way the vocalization hypotheses suggest it should. All of these variables may be related to rate or timing changes. It seems fair to conclude that the vocalization hypotheses do not account for the distribution of stuttering in continuous speech as completely as rate-change or timing explanations.

Another fact about stuttering that is well established is its higher prevalence in males than in females. The sexes of course differ substantially in the length and mass of the vocal folds and in the fundamental frequency of voicing. Furthermore, the extent of sexual dimorphism in voicing is sufficient to account for the extent of the sex ratio of stuttering distribution, which has been said to range from 3:1 to 10:1 (Van Riper, 1973a). The difficulty, however, is that stuttering typically begins in childhood before this extensive vocal dimorphism has developed. Furthermore, as the difference in laryngeal size increases with age, the chances of stuttering onset decrease. Still, the child's vocal mechanism is potentially different according to sex, and some characteristic of it that has not yet been identified may be different enough in children to account for the sex ratio. Differences between the sexes with regard to rate and duration, however, are just as difficult to use as explanations of the sex ratio. A difference in the rate of speech between males and females in childhood has been observed (Dawson, 1929), but the observations are old and the observational techniques odd. In adults, there is a difference between the sexes in the length of utterance, a variable that is correlated with the rate of speech and duration of sounds, but the extent of this effect is small and the research has been limited to French adults (Malécot, Johnston, & Kizziar, 1972). It should be noted that there is a substantial difference between the sexes in reaction time, males being faster, and although the extent of this difference and its reliability are probably sufficient to account for the sex ratio in stuttering, the direction of the difference is the opposite of what one would predict. That is, the males are faster but more prone to stutter, whereas stutterers are slower. A somewhat similar difficulty is encountered in explaining the fact that stuttering is so prevalent in childhood and relatively rare in adulthood. Children, like stutterers, react more slowly and talk more slowly than adults, and reaction time or rate might consequently be used to explain the greater prevalence of stuttering in children. Unfortunately, reaction time and speech rate both decline again in middle age and older years. Stuttering, however, continues to become less prevalent with maturity and advancing age (Shames & Beams, 1956). Neither vocalization nor timing explain the sex ratio adequately.

Two areas of information have been identified in which the vocalization hypotheses fail to account for facts that are known about stuttering, the distribution of stutterings in continuous speech and the sex ratio. In the first of these areas, the vocalization hypothesis does not explain the facts as well as a timing hypothesis does. In the second area, neither hypothesis explains the facts adequately.

CONCLUSION

The purpose of this monograph was to review critically the literature on stuttering behavior and to sharpen an understanding of the disorder. What, then, do these experiments have to say about the relation between stuttering and vocalization? Two hypotheses, one weak and one strong, have been identified. The weak one is that stuttering behaviors are performed by the larynx. The strong one is that these laryngeal stuttering behaviors are in some sense "primary." Two senses of "primary" can be identified: in the first, laryngeal stuttering occurs first in the sequence of stuttering behaviors; in the second, laryngeal stuttering occurs first in the development of the disorder. A collateral idea is that in either of the two senses of "primary" above, the nonlaryngeal stuttering behaviors, as Schwartz (1974; 1975) suggested, are "precipitated" by larngeal stuttering behaviors, perhaps through vocal tract aerodynamics, as Adams (1974) has suggested.

It is clear that the weak vocalization hypothesis has been demonstrated. The laryngeal mechanism shows tension and co-contraction of antagonistic muscles that are similar in magnitude to the tensions and co-contractions seen in the oral mechanism during stuttering. These abnormalities are absent from most fluent productions. The exceptions are abnormalities that fail to produce acoustically observable dysfluency.[10] Some stutterings that are observable as oral articulatory tensions are also accompanied by inappropriate laryngeal tensions or timing, and these will not be visible and may also be inaudible.

These facts may seem trivial in the theoretical sense, and indeed they do not tell us much about what causes stuttering, but they have some important clinical implications that will be discussed shortly.

There have been no direct tests of the idea that laryngeal behaviors are "primary" in the developmental sense. Neither Van Riper's (1971) nor Bloodstein's (1960) review of stuttering development suggest that they are. Similarly, there have been no direct tests of the idea that laryngeal behaviors are primary in the physiological sense, although the observations of Ford and Luper (Note 7) and to a lesser extent those of Freeman and Ushijima (1975; 1978) might have been expected to uncover such a phenomenon. Although laryngeal behaviors were observed early in the stuttering sequence, and consistently so in some subjects, other subjects showed other sequences. The evidence to date seems to show that the sequential patterns are individual and do not favor the early occurrence of stuttering behavior in the laryngeal mechanism, or for that matter in the oral or respiratory mechanisms.

There have been some tests of the collateral idea that nonlaryngeal behaviors are a consequence of laryngeal ones, either as coping devices or as the result of aerodynamic forces. The correlations found by Conture, McCall, and Brewer (1977) mean that laryngeal and oral behaviors often co-vary and that stuttering in one system may precipitate stuttering in the other, but there is no evidence that stuttering in one system precedes stuttering in the other system in any systematic way that is true for stutterers in general.

Most tests of the strong vocalization hypothesis have looked for differences in the vocal functioning of stutterers and nonstutterers, differences that are independent of stuttering itself, by comparing stutterers and nonstutterers on vocal reaction time or by comparing VOT and VTT with stutterings excluded. When over-interpretations are removed and alternative explanations allowed, there is no evidence that, except for stuttering behaviors themselves, the vocal functioning of stutterers differs from that of nonstutterers in any way that is not also true of oral articulatory functioning. One observation has been made repeatedly: stutterers do not react with their speech systems as quickly as nonstutterers, but this is true not just of the vocal but of the oral mechanism too and indeed seems to be a general deficit probably of central origin. Even this observation, however, may result from the failure to identify subacoustic "stuttering-like" muscle abnormalities.

Other tests of the vocalization hypotheses have dealt with their predictions about adaptation, fluency enhancement, or the distribution of stuttering. But in these tests too, no clear evidence favoring vocalization theory has emerged.

Despite the failure of these tests to support the strong vocalization hypothesis, and despite the deconfirmation of some of its predictions, it is too soon to turn entirely to new directions of research and abandon this explanation of stuttering. Many of the experiments were poorly designed to permit the conclusion that the vocalization hypothesis is a complete failure. The most common design errors were: (a) insufficient sample size, (b) failure to control suspected independent variables, particularly rate/duration effects, (c) failure to demonstrate with correlation tests that concomitant variations are more than merely coincidental. New experiments designed to answer the same questions but with better designs need to be conducted before the vocalization hypothesis can be abandoned. The effects of fluency-enhancing conditions on the speech of stutterers and nonstutterers is a particularly important area in which more work needs to be done. At this point however, the outlook for the vocalization theory of stuttering seems bleak.

Clinical Implications

Although the theoretical implications of the vocalization data have been few and often unrelated to the hypothesis, the clinical implications are substantial. What we know about laryngeal stuttering is that it occurs, that it is difficult to observe, and that it may pre-

[10]It is important to remember that there is really very little evidence yet that these behaviors are really "abnormalities." They may occur in nonstutterers too.

cipitate or be precipitated by oral stutterings. Even these simple facts have important implications for treatment.

Consider diagnostics. Stutterers who stutter orally may also be stuttering laryngeally, but laryngeal stutterings will often be missed, and as a result, severity may be underestimated (Adams, 1975). Careful listening will go a long way toward solving this problem. Vocal tension makes itself known by rising pitch, aperiodic sounds, and changes in quality. Symptoms such as devoicing of voiced sounds (/p/ for /b/ substitution), sudden or violent vocal attack, or voice breaks, which have long been observed in stutterers, take on added meaning. Laryngeal tension has two other effects that can be observed. First, there is excessive movement or tension of the external laryngeal (strap) muscles accompanying tension of the internal muscles. Conture, Gould, and Caruso (Note 5) examined laryngeal movement in eight adult stutterers and found that in all subjects the larynx was lowered visibly during most stutterings, and in some stutterers during every instance of stuttering. External tension is a nearly certain sign of internal tension, but the absence of external tension does not mean that the intrinsic muscles are tension-free. Second, habitual vocal tension leads to vocal fatigue, usually changes in voice quality that occur late in the day or after more talking than usual. A particularly meaningful symptom is hoarseness or some other quality change, or frequent voice breaks, after a lengthy period of severe stuttering.

These observations of vocal tension can be quantified, recorded, and placed in the client's record. They can be used as a basal level of performance against which the clinician can measure the success of future attempts at intervention. In the case of externally visible laryngeal movement, clinicians can videotape clients during evaluations and later count the percentage of words on which the laryngeal movement was seen. If there are different types of laryngeal movement in the same client, they are counted separately. Examples include descent or ascent of the larynx or bulging or lateral movement of one or more of the strap muscles, both of which are common in many stutterers. At the same time, acoustically observable signs of laryngeal involvement should also be noted; quality changes, pitch breaks, pitch changes during words, voice breaks, vocal fading, etc. In addition to the proportion of words on which each of these acoustically and visually observable behaviors occurs, it is also helpful to measure their duration. Duration of an abnormal laryngeal behavior is an indirect measure of the degree of tension in the larynx. Finally, the clinician can note what proportion of acoustically stuttered words was accompanied by laryngeal movement, and if any unstuttered words were produced with laryngeal movement. The first of these two observations gives some indication of the extent to which the larynx is involved in a particular client's stuttering. To evaluate the extent of laryngeal involvement by comparison with other structures, each stuttering behavior has to be categorized according to the structural system—respiratory, laryngeal, oral—so that the proportion of stuttered words assignable to each category can be assessed. A profile is obtained for each client that shows the percentage of stutterings that are laryngeal, the percentage that are oral, and the percentage that are respiratory. Later, after time has been spent in remediation, a similar profile can be taken and compared with the one taken earlier. Emphasis in therapy on one system or another should be reflected in changes in the distribution of stuttering behaviors as well as in a reduction of the overall number (proportion of words stuttered). If an emphasis on laryngeal behavior is having the desired effect, the reevaluation profile should show a diminished proportion of laryngeal involvement. Reductions in other nonlaryngeal behavior will probably also occur, and this may be because of a placebo effect (the stutterer's very strong tendency to improve because he believes the clinician is helping him) but they may also occur because for the particular client laryngeal stutterings were precipitating oral or respiratory events that also interfered with the forward flow of speech.[11] It is not really possible to distinguish between these two effects. One word of caution is necessary about the interpretation of profiles. Speech tends to involve the entire vocal tract. The tract functions during speech very much like a single organ, so the fact that a particular client shows a predominantly laryngeal profile does not mean that respiratory or oral mechanisms are not involved in the stuttering behaviors. They almost certainly are. The reverse is also true, of course. The predominantly oral stutterer almost certainly has some laryngeal involvement. Profiles of this sort tell as much about how the stutterer is viewed by listeners as they tell about how the stuttering behaviors are produced. The important point is that the stutterer is one of his listeners, and if others hear him as predominantly laryngeal, then that is how he hears himself. Ultimately it is the stutterer's own ear that will have to be trained by therapy to distinguish between behaviors that hinder and behaviors that foster smooth and easy communication. It is in this sense that the behavioral profile will have an effect on therapy; it will determine what the client learns to listen for.

The second observation that can be made from externally visible laryngeal activity is the proportion of acoustically "fluent" words that show the same pattern of laryngeal movement the client typically shows on stuttered words. The clinician cannot be certain, but if the type, extent, and timing of movement is similar to the client's pattern of laryngeal movement on stuttered words, it is reasonable to suppose that the occurrence of the same pattern on fluent words is a "subacoustic" stuttering. Although this supposition is speculative and uncertain, the clinician may wish to go ahead and count the proportion of words on which these movements or signs of tension were seen. The proportion of unstuttered words accompanied by laryngeal tension or movement can then be compared with the proportion of stuttered

[11]The reverse is also true—working on oral behaviors tends to improve laryngeal ones and for the same reasons.

words accompanied by the same pattern of behavior. The differences between the two is an index of the extent to which the client is controlling his laryngeal stuttering. Clients who show a high proportion of unstuttered words accompanied by stuttering-like laryngeal movements or tensions are exerting effort in order to talk without stuttering. Such clients are not speaking fluently in the broad sense of the word. They are not speaking effortlessly. They are "controlling," "hiding," or "interiorizing" their stuttering, depending on one's point of view. It seems appropriate for clinicians to remove these behaviors by retraining the clients to speak with less tension and effort and without the use of extraneous laryngeal movements.

These suggestions for the observation of external laryngeal movements, and what to do on the basis of them, are all essentially stop-gap measures. What is needed is a diagnostic tool that will enable the clinician to identify tension or contraction in specific internal laryngeal muscles. No such instrument exists. However, the glottograph provides an external view of internal movement. Although it does not provide details about specific muscles, it seems nonetheless promising for clinical use. The glottograph works on the principle that changes in the position and thickness of tissue alter its capacity to conduct electricity. An imperceptible current is passed through the larynx. A sensitive instrument measures the current before and after its passage through the tissues. Movements within the larynx change the ability of the tissues to conduct electricity, and these changes in impedance over time are recorded on graph paper. Some training is required to learn how to interpret the output of the instrument, but once trained, a clinician can detect opening and closing of the laryngeal valve, rapid vibratory movements of the vocal folds, and the opening and closing of the arytenoid cartilages at voice onset and offset. To date, however, a complete description of glottographic measurement in stutterers is lacking. A comparison of the timing and extent of laryngeal movement in stuttered and unstuttered tokens produced by stutterers and nonstutterers and the relation of these movements to oral and respiratory events and to acoustic events is needed before clinical application of the instrument is feasible.

Of all the instruments with which laryngeal activity can be monitored, the glottograph is least invasive. It is the only instrument that could conceivably be used by the practicing clinician in order to monitor laryngeal activity in stutterers.

The facts about laryngeal stuttering also have implications about prognosis. Laryngeal stuttering is probably more difficult to treat than oral stuttering, if only because it is so difficult to observe. Prognosis will be poorer for clients whose stuttering is more laryngeal than oral. If oral stuttering behaviors resist modification, one reason (among many) may be that they are being precipitated by laryngeal behaviors that have not been observed or identified. That notion, however, has not been substantiated experimentally.

As far as therapy itself is concerned, careful and concerned clinicians have for many years sought to modify the laryngeal behaviors of stutterers when an evaluation of the client suggested that laryngeal gestures were excessively tense or effortful or were inappropriately timed or poorly coordinated with respiratory or oral articulatory movements. This concern is evident in the works of Van Riper (1971; 1973), Makuen (Note 15), Cooper (1979), and Peins, McGough, and Lee (1972). Concern can also be inferred from the therapies described by Williams (1958), Brutten and Shoemaker (1967), and probably many others. More recently, there has been a somewhat greater emphasis on laryngeal behavior. Therapies have been described in which a major component is directed at the modification of laryngeal behavior, often without regard to any specific indications from pretherapy evaluation that laryngeal behavior was abnormal. Webster (1974; 1979) has described a therapy in which one of the major goals is to achieve gentle phonatory onset. Since the program is administered with little individual modification, there is a presumption that each client presents abrupt vocal onset as a symptom. The research reviewed in this monograph does not warrant that presumption. Of course, an individual not presenting this symptom would be expected to achieve the gentle vocal onset goal more quickly than one who did not. Perkins (1973a; 1973b) has described a course of therapy aimed at modifications in speaking behavior, two of which are focussed on laryngeal behavior. The two laryngeally related goals are normal breath flow and normal prosody. Normal breath flow is achieved in three steps: shorter phrase lengths for the slower speech rates established in an earlier part of the therapy, continuous airflow throughout the phrase, and gentle phonatory onset. Normal prosody is achieved in one step as an adjustment to the already slowed rate. These two goals are then maintained as rate is gradually increased to normal levels. Perkins' therapy appears to be more sensitive to individual differences than Webster's, and clients whose symptoms do not include laryngeal abnormalities would presumably be checked through these subgoals with little delay. Nevertheless, the therapy places some emphasis on the modification of abnormal laryngeal behavior.

A therapy described by Wiener (1978) goes even further and places the greatest emphasis on vocal control and on the modification of behavior related to the production of voice and resonance. The basic goal of the program is for the clients to be able to initiate and sustain phonation with adequate timing, quality, and level without using excessive effort. To achieve this goal, training in the maintenance of adequate airflow is used as a starting point. Other subgoals are the elimination of vocal fry, "constricted phonation," and "reduced resonance." Harsh glottal attack is replaced by easy onset of phonation. In cases where oral stutterings remain part of the problem (it is anticipated that they will disappear following work on voice production), they are treated directly. Once clients have established new phonatory behavior and fluent speech, they are desensitized to feared

speech situations, using Wolpean techniques, so that they will be able to practice the newly learned behaviors in daily speaking situations without reverting to stuttering behaviors. Although Wiener's therapy is not preprogrammed and is consequently sensitive to individual client differences, its strong focus on voice and resonance suggests a presumption, or at least a bias, that laryngeal and/or respiratory disturbance is at the heart of the problem. Although research evidence does not warrant the presumption, or even the bias in my opinion, it should be noted that Wiener describes the program as "experimental." Thus, it may be seen as a valuable source of clinical data on the evaluation and treatment of laryngeal stuttering.

We still know less about the treatment of laryngeal stuttering than we do about the treatment of oral stuttering, and the development and refinement of these therapies should be encouraged. But the development and evaluation of new therapy techniques is difficult and time-consuming, and they are particularly susceptible to experimenter and subject biases. Consequently, a rigorous standard, such as that suggested by Bloodstein (1981), should be adopted. This standard includes the following criteria:

1. A large and representative sample of stutterers.
2. Objective measures such as stuttering frequency, speech rate, and judged severity. Judges must be reliable, and they must be other than the experimenters.
3. Speech must be evaluated often and in a variety of situations.
4. Speech must be evaluated covertly and outside the clinical situations.
5. Long-term follow-up of the results, at least two years, must be employed, and this must also be done covertly and outside the clinic.
6. Controls for the placebo effect, for spontaneous recovery, and for the tendency of stutterers to seek help when their stuttering is at its worst must be used.
7. The subjects' speech must be completely fluent, not just free of stuttering. That is, they must speak in a natural rhythm, at a normal rate of speech, and without conscious effort or concentration. They must speak spontaneously.
8. The subjects must not consider themselves stutterers at the end of treatment.
9. Case selection must be all-inclusive or randomized, and drop-outs must be considered failures of the method.
10. The therapy must not depend on unusual personality or on status of the clinician.
11. The method's effectiveness must continue after the first flush of success has worn off. This standard (Bloodstein, 1981, pp. 386-390) is rigorous and may discourage many investigators. But we are better off without data on the effectiveness of therapy than we are with "results" that fool us into believing a therapy is effective when it is not.

Reference Notes

1. Agnello, J., & Wingate, M. E. *Some acoustical and physiological aspects of stuttered speech.* Paper presented at the convention of the American Speech and Hearing Association, San Francisco, 1972.
2. Agnello, J., Wingate, M. E., & Wendell, M. V. *Voice onset and voice termination times of children and adult stutterers.* Paper presented at the convention of the American Speech and Hearing Association, Las Vegas, 1974.
3. Barrett, R. S., & Stoeckel, C. M. *Unilateral eyelid movement control in stutterers and nonstutterers.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.
4. Borden, G., & Starkweather, C. W. *Feedback and the acquisition of language in humans and song in birds.* Symposium presented at Temple University, Philadelphia, 1977.
5. Conture, E. G., Gould, L. W., & Caruso, A. *The descent of the larynx during stuttering.* Paper presented at the convention of the American Speech-Language-Hearing Association, Detroit, 1980.
6. Cross, J., & Cooke, P. A. *Vocal and manual reaction times of adult stutterers and nonstutterers.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.
7. Ford, S., & Luper, H. *Aerodynamic, phonatory, and labial EMG patterns during fluent and stuttered speech.* Paper presented at the convention of the American Speech and Hearing Association, Washington, D.C., 1975.
8. Hand, R., & Luper, H. *Durational characteristics of stutterers' and nonstutterers' fluent speech.* Paper presented at the convention of the American Speech-Language-Hearing Association, Detroit, 1980.
9. Hayden, P. A., & Jordahl, N. *Stutterers' voice initiation times during conditions of novel stimuli.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.
10. Hutchinson, J. M., McGee, S. R., & Deputy, P. N. *Phonatory onsets and stuttering frequency in children.* Paper presented at the convention of the American Speech-Language-Hearing Association, Detroit, 1980.
11. Kerr, S. H., & Cooper, E. B. *Phonatory adjustment times in stutterers and nonstutterers.* Paper presented at the convention of the American Speech and Hearing Association, Houston, 1976.
12. Lewis, J. I., Ingham, R. J., & Gervens, A. *Voice initiation and termination times in stutterers and normal speakers.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.
13. Luper, H. L., & Cross, D. E. *Finger reaction time of stuttering and nonstuttering children and adults.* Paper presented at the convention of the American Speech and Hearing Association, San Francisco, 1978.
14. MacFarlane, S. C. *Neural response time of stutterers and nonstutterers for certain oral motor tasks as a function of auditory stimulation.* Paper presented at the convention of the American Speech and Hearing Association, Houston, 1976.
15. Makuen, G. Quoted in M. Steel, *How Dr. G. Hudson Makuen treated stammering.* In R. West (Ed.), Symposium on Stuttering presented at the American Society of the study of Disorders of Speech, 1930/1931.
16. Reich, A., Till, J., Goldsmith, H., & Prins, D. *Reaction-time experiments in stuttering and nonstuttering adults.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.
17. Shapiro, A. *Studies of abnormal phonation.* Unpublished manuscript, 1978.
18. Wall, M. J., & Pfeuffer, S. *A distinctive feature analysis of stuttered phonemes in four- and five-year-olds.* Paper presented at the convention of the American Speech and Hearing Association, San Francisco, 1978.
19. Webster, R. L., & Clark, A. A. *Masking influences on speech tract latencies in stutterers.* Paper presented at the convention of the American Speech-Language-Hearing Association, Atlanta, 1979.

References

Adair, J. *The human subject: The social psychology of the psychological experiment.* Boston: Little, Brown and Company, 1973.

Adams, M. R. Vocal tract dynamics in fluency and stuttering: A review and interpretation of past research. In L. M. Webster & L. C. Furst (Eds.), *Vocal tract dynamics and dysfluency.* New York: Speech and Hearing Institute, 1974.

Adams, M. R. Comments on Schwartz's: "The core of stuttering block." *Journal of Speech and Hearing Disorders,* 1975, *40,* 136.

Adams, M. R. Stuttering theory, research, and therapy: The present and future. *Journal of Fluency Disorders,* 1978, *3,* 139-147.

Adams, M. R., & Hayden, P. The ability of stutterers and nonstutterers to initiate and terminate phonation during production of an isolated vowel. *Journal of Speech and Hearing Research,* 1976, *19,* 290-296.

Adams, M. R., & Moore, W. H., Jr. The effects of auditory masking on the anxiety level, frequency of dysfluency, and selected vocal characteristics of stutterers. *Journal of Speech and Hearing Research,* 1972, *15,* 572-578.

Adams, M. R., & Ramig, P. Vocal characteristics of normal speakers and stutterers during choral reading. *Journal of Speech and Hearing Research,* 1980, 23, 457-469.

Adams, M. R., Riemenschneider, S., Metz, D., & Conture, E. Voice onset and articulatory constriction requirements in a speech segment and their relation to the amount of stuttering adaptation. *Journal of Fluency Disorders,* 1974, *1,* 23-29.

Adams, M. R., & Reis, R. The influence of the onset of phonation on the frequency of stuttering. *Journal of Speech and Hearing Research,* 1971, *14,* 639-644.

Adams, M. R., & Reis, R. The influence of the onset of phonation on the frequency of stuttering: A replication and re-evaluation. *Journal of Speech and Hearing Research,* 1974, *17,* 752-754.

Adams, M. R., Runyan, C., & Mallard, A. R. Air flow characteristics of the speech of stutterers and nonstutterers. *Journal of Fluency Disorders,* 1975, *1,* 3-12.

Adler, J. *Hemispheric differences in vocal and articulatory reaction times to linguistic and nonlinguistic visual stimuli in stutterers and nonstutterers.* Master's thesis, Temple University, 1977.

Agnello, J. Durational differences in speech production under normal and delayed auditory feedback. *Speech Monograph,* 1970, 37, 195-198.

Agnello, J. Laryngeal and articulatory dynamics of dysfluency interpreted within a vocal tract model. In L. M. Webster & L. C. Furst (Eds.), *Vocal tract dynamics and dysfluency.* New York: Speech Rehabilitation Institute, 1974.

Allen, G. D. The location of rhythmic stress beats in English: An experimental study. *Language and Speech,* 1972, *15,* 72-100.

Azrin, N., Jones, R. J., & Flye, B. A synchronization effect and its application to stuttering by a portable apparatus. *Journal of Applied Behavioral Analysis,* 1968, *1,* 283-295.

Barber, V. Studies in the psychology of stuttering XVI: Rhythm as a distraction in stuttering. *Journal of Speech Disorders,* 1940, 5, 29-42.

Besozzi, T., & Adams, M. R. The influence of prosody on stuttering adaptation. *Journal of Speech and Hearing Research,* 1969, *12,* 818-824.

BLOODSTEIN, O. Hypothetic conditions under which stuttering is reduced or absent. *Journal of Speech and Hearing Disorders*, 1950, *15*, 142-153.

BLOODSTEIN, O. The development of stuttering I. Changes in nine basic features. *Journal of Speech and Hearing Disorders*, 1960, *25*, 219-237.

BLOODSTEIN, O. *A handbook on stuttering* (3rd ed.). Chicago: The Easter Seal Society, 1981.

BLOODSTEIN, O., & GANTWERK, B. F. Grammatical function in relation to stuttering in young children. *Journal of Speech and Hearing Research*, 1967, *10*, 786-789.

BOOMSLITER, P. C., & HASTINGS, G. S., Jr. The "instant" of the syllable. *Journal of the Acoustical Society of America*, 1971, *49*, 104.

BORDEN, G. An interpretation of research on feedback interruption in speech. *Brain and Language*, 1979, *7*, 307-319.

BRADY, J. P. Studies of the metronome effect on stuttering. *Behavioral Research and Therapy*, 1969, *7*, 197-204.

BRENNER, N. C., PERKINS, W. H., & SODERBERG, G. A. The effect of rehearsal on frequency of stuttering. *Journal of Speech and Hearing Research*, 1972, *15*, 483-486.

BROWN, S. F. Stuttering with relation to word accent and word position. *Journal of Abnormal and Social Psychology*, 1938, *32*, 112-120.

BROWN, S. F., & MOREN, A. The frequency of stuttering in relation to word length in oral reading. *Journal of Speech Disorders*, 1942, *7*, 153-159.

BRUCE, M. C., & ADAMS, M. R. Effects of two types of motor practice on stuttering. *Journal of Speech and Hearing Research*, 1978, *21*, 421-428.

BRUTTEN, G. J., & SHOEMAKER, D. J. *The modification of stuttering*. Englewood Cliffs, NJ: Prentice-Hall, 1967.

CHERRY, E. C. Some experiments on the recognition of speech, with one and two ears. *Journal of the Acoustical Society of America*, 1953, *25*, 975-979.

CHERRY, E. C., SAYERS, B. M., & MARLAND, P. Experiments upon the total inhibition of stammering by external control, and some clinical results. *Journal of Psychosomatic Research*, 1956, *1*, 233-246.

CHEVRIE-MÜLLER, C. A study of laryngeal function in stutterers by the glottographic method. *Proc. VII^e Congrès de la société francaise de médecine de la voix et de la parole*. Paris, 1963.

CIAMBRONE, S., ADAMS, M., & BERKOWITZ, M. *A correlational study of stutterers' adaptation and voice initiation times*. Paper presented at the American Speech-Language-Hearing Association Convention, Los Angeles, 1980.

COLCORD, R., & ADAMS, M. Voicing duration and vocal SPL changes associated with stuttering reduction during singing. *Journal of Speech and Hearing Research*, 1979, *22*, 468-479.

CONTURE, E. G., MCCALL, G., & BREWER, D. W. Laryngeal behavior during stuttering. *Journal of Speech and Hearing Research*, 1977, *20*, 661-668.

CONWAY, J. K., & QUARRINGTON, B. Positional effects in the stuttering of contextually organized verbal material. *Journal of Abnormal and Social Psychology*, 1963, *47*, 299-303.

COOPER, E. Intervention procedures for the young stutterer. In H. Gregory (Ed.), *Controversies about stuttering therapy*. Baltimore: University Park Press, 1979.

CROSS, D. E., & LUPER, H. L. Voice reaction time of stuttering and nonstuttering children and adults. *Journal of Fluency Disorders*, 1979, *4*, 59-77.

CROSS, D. E., SHADDEN, B. B., & LUPER, H. L. Effects of stimulus ear presentation on the voice reaction time of adult stutterers and nonstutterers. *Journal of Fluency Disorders*, 1979, *4*, 45-58.

CULLINAN, W., & SPRINGER, M. Voice initiation and termination times in stuttering and nonstuttering children. *Journal of Speech and Hearing Research*, 1980, *23*, 344-360.

DANILOFF, R. Normal articulation processes. In F. Minifie, T. Hixon, & F. Williams (Eds.), *Normal aspects of speech, hearing and language*. Englewood Cliffs, NJ: Prentice-Hall, 1973.

DAWSON, L. O. A study of the development of the rate of articulation. *Elementary School Journal*, 1929, *29*, 610-615.

DE SIMONI, F. G. Influence of utterance length upon bilabial closure duration for /p/ in three-, six-, and nine-year-old children. *Journal of the Acoustical Society of America*, 1974, *55*, 1353-1354. (a)

DE SIMONI, F. G. Preliminary study of certain timing relationships in the speech of stutterers. *Journal of the Acoustical Society of America*, 1974, *56*, 695-696. (b)

DINNAN, J. A., MCGUINESS, E., & PERRIN, L. Auditory feedback: Stutterers v. nonstutterers. *Journal of Learning Disabilities*, 1970, *3*, 209-213.

EISENSON, J., & HOROWITZ, E. The influence of propositionality on stuttering. *Journal of Speech Disorders*, 1945, *10*, 193-197.

FOLKINS, J. W., MILLER, C. J., & MINIFIE, F. D. Rhythm and syllable timing in phrase level stress patterning. *Journal of Speech and Hearing Research*, 1975, *18*, 739-753.

FRANSELLA, F. Rhythm as a distraction in the modification of stuttering. *Behavioral Research and Therapy*, 1967, *5*, 253-255.

FRANSELLA, F., & BEECH, H. R. An experimental analysis of the effect of rhythm on the speech of stutterers. *Behavioral Research and Therapy*, 1965, *3*, 195-201.

FREEMAN, F., & USHIJIMA, T. Laryngeal activity accompanying the moment of stuttering: A preliminary report of EMG investigation. *Journal of Fluency Disorders*, 1975, *1*, 36-45.

FREEMAN, F., & USHIJIMA, T. Laryngeal muscle activity during stuttering. *Journal of Speech and Hearing Research*, 1978, *21*, 538-562.

FREEMAN, F., USHIJIMA, T., & HIROSE, H. Reply to Schwartz's: "The core of the stuttering block." *Journal of Speech and Hearing Disorders*, 1975, *40*, 137-139.

FROESCHELS, E. New viewpoints on stuttering. *Folia Phoniatrica*, 1961, *13*, 187-201.

GARBER, S. F., & MARTIN, R. R. Effects of noise on increased vocal intensity and stuttering. *Journal of Speech and Hearing Research*, 1977, *20*, 233-240.

GAY, T. Effect of speaking rate on vowel formant movements. *Journal of the Acoustical Society of America*, 1978, *63*, 223-230.

HAHN, E. A study of the relationship between stuttering occurrence and grammatical factors in oral reading. *Journal of Speech Disorders*, 1942, *7*, 329-355.

HARDY, J. C. Air flow and air pressure studies. *ASHA Reports*, 1965, *No. 1*, 141-152.

HEALEY, C. E. *Temporal relationships and strategies used by normal and stuttering children and adults in the self-regulation of speaking rate*. Doctoral dissertation, Purdue University, 1977.

HEALEY, E. C. *Fundamental frequency contours of stutterers' vowels following fluent stop consonant productions*. Paper presented at the American Speech-Language-Hearing Association Convention, Los Angeles, 1980.

HEALEY, E. C., MALLARD, A. R., & ADAMS, M. R. Factors contributing to the reduction of stuttering during singing. *Journal of Speech and Hearing Research*, 1976, *19*, 475-480.

HEJNA, R. F. A study of the loci of stuttering in spontaneous speech. *Dissertation Abstracts*, 1955, *15*, 1674-1675.

HILLMAN, R. E., & GILBERT, H. R. Voice onset time for voiceless stop consonants in the fluent reading of stutterers and nonstutterers. *Journal of the Acoustical Society of America*, 1977, *61*, 610-611.

HOUSE, A. S., & FAIRBANKS, G. The influence of consonant environment upon the secondary acoustical characteristics of vowels. *Journal of the Acoustical Society of America*, 1953, *25*, 105-113.

HUTCHINSON, J. *The effect of oral sensory deprivation on stuttering behavior*. Unpublished doctoral dissertation, Purdue University, 1973.

HUTCHINSON, J. M., & BROWN, D. The Adams and Reis obser-

vations revisited. *Journal of Fluency Disorders*, 1978, *3*, 149-154.

JONES, D. Chronemes and tonemes. *Acta Linguistica*, 1948, *4*, 1-10.

KLATT, D. H. Interaction between two factors that influence vowel duration. *Journal of the Acoustical Society of America*, 1973, *54*, 1102-1104.

KLATT, D. The duration of /s/ in English words. *Journal of Speech and Hearing Research*, 1974, *17*, 51-63.

LASS, N. J., & SANDUSKY, J. C. A study of the relationship of diadochokinetic rate, speaking rate, and reading rate. *Today's Speech*, 1971, *19*, 49-54.

LECHNER, B. The effects of delayed auditory feedback and masking on the fundamental frequency of stutterers and nonstutterers. *Journal of Speech and Hearing Research*, 1979, *22*, 343-353.

LINDBLOM, B. Temporal organization of syllable production. Speech Transmission Laboratory. Royal Institute of Technology. *Quarterly Progress Status Report*, Stockholm, Sweden, 1968.

LISKER, L., & ABRAMSON, A. S. Some effects of context on voice onset time in English stops. *Language and Speech*, 1967, *10*, 1-28.

LITTIG, L., & WADDELL, C. Sex and experimenter interaction in serial learning. *Journal of Verbal Learning and Verbal Behavior*, 1967, *6*, 676-678.

MACKAY, D. Metamorphosis of a critical interval: Age-linked changes in the delay in auditory feedback that produces maximal disruption of speech. *Journal of the Acoustical Society of America*, 1968, *43*, 811-821.

MALÉCOT, A., JOHNSTON, R., & KIZZIAR, P. A. Syllabic rate and utterance length in French. *Phonetica*, 1972, *26*, 235-251.

MARTIN, R., & HAROLDSON, S. Effects of five experimental treatments on stuttering. *Journal of Speech and Hearing Research*, 1979, *22*, B2-46.

MENYUK, P., & KLATT, D. Voice onset time in consonant cluster production by children and adults. *Journal of Child Language*, 1975, *2*, 223-231.

METZ, D., CONTURE, E., & CARUSO, A. Voice onset time, frication and aspiration durations: A comparison of stutterers and nonstutterers. *Journal of Speech and Hearing Research*, 1979, *22*, 649-656.

MEYER, V., & MAIR, J. M. A new technique to control stammering: A preliminary report. *Behavioral Research and Therapy*, 1963, *1*, 251-254.

MILLER, G. A. Speech and language. In S. S. Stevens (Ed.), *Handbook of experimental psychology*. New York: Wiley, 1951.

NETSELL, R., & DANIELL, B. Neural and mechanical response time for speech production. *Journal of Speech and Hearing Research*, 1974, *17*, 608-618.

OLLER, D. K. The effect of position in utterance on speech segment duration in English. *Journal of the Acoustical Society of America*, 1973, *54*, 1235-1247.

PARNELL, M., AMERMAN, J. D., & WELLS, G. B. Closure and constrictions duration for alveolar consonants during voiced and whispered speaking conditions. *Journal of the Acoustical Society of America*, 1977, *61*, 612-613.

PEINS, M. Adaptation effect and spontaneous recovery in stuttering expectancy. *Journal of Speech and Hearing Research*, 1961, *4*, 91-99.

PEINS, M., MCGOUGH, W., & LEE, B. Evaluation of a tape-recorded method of stuttering therapy: Improvement in a speaking task. *Journal of Speech and Hearing Research*, 1972, *15*, 364-371.

PERKINS, W. Replacement of stuttering with normal speech: I. Rationale. *Journal of Speech and Hearing Research*, 1973, *38*, 283-294. (a)

PERKINS, W. Replacement of stuttering with normal speech: II. Clinical procedures. *Journal of Speech and Hearing Research*, 1973, *38*, 295-303. (b)

PERKINS, W., RUDAS, J., JOHNSON, L., & BELL, J. Stuttering: Discoordination of phonation with articulation and respiration. *Journal of Speech and Hearing Research*, 1976, *19*, 509-522.

PROSEK, R., MONTGOMERY, A., WALDEN, B., & SCHWARTZ, D. Reaction-time measures of stutterers and nonstutterers. *Journal of Fluency Disorders*, 1979, *4*, 269-278.

QUARRINGTON, B., CONWAY, J., & SIEGEL, N. An experimental study of some properties of stuttered words. *Journal of Speech and Hearing Research*, 1962, *5*, 387-394.

RAMIG, P., & ADAMS, M. Rate reduction strategies used by stutterers and nonstutterers during high- and low-pitched speech. *Journal of Fluency Disorders*, 1980, *5*, 27-41.

RINGEL, R., & STEER, M. Same effects of tactile and auditory alterations on speech output. *Journal of Speech and Hearing Research*, 1963, *6*, 369-378.

RONSON, I. Word frequency and stuttering: The relationship to sentence structure. *Journal of Speech and Hearing Research*, 1976, *19*, 813-819.

ROSCOE, J. *Fundamental research statistics for the behavioral sciences* (2nd ed.). New York: Holt, 1975.

ROSENTHAL, R. *Experimenter effects in behavioral research*. New York: Appleton, 1966.

SCHWARTZ, M. The core of the stuttering block. *Journal of Speech and Hearing Disorders*, 1974, *39*, 169-177.

SCHWARTZ, M. Author's Response to Zimmermann and Allen, and Adams. *Journal of Speech and Hearing Disorders*, 1975, *40*, 136-137.

SERGEANT, R. L. Concurrent repetition of a continuous flow of words. *Journal of Speech and Hearing Research*, 1961, *4*, 373-380.

SHAMES, G., & BEAMS, H. Incidence of stuttering in older age groups. *Journal of Speech and Hearing Disorders*, 1956, *21*, 313-316.

SHAPIRO, A. An electromyographic analysis of the fluent and dysfluent utterance of several types of stutterers. *Journal of Fluency Disorders*, 1980, *5*, 203-231.

SODERBERG, G. The relations of stuttering to word length and word frequency. *Journal of Speech and Hearing Research*, 1966, *9*, 584-589.

STARBUCK, H., & STEER, M. The adaptation effect in stuttering and its relation to thoracic and abdominal breathing. *Journal of Speech and Hearing Disorders*, 1954, *19*, 440-449.

STARKWEATHER, C. W. Speech fluency and its development in children. In N. Lass (Ed.), *Speech and language: Research and theory*. New York: Academic Press, 1980.

STARKWEATHER, C. W., HIRSCHMAN, P., & TANNENBAUM, R. S. Latency of vocalization: Stutterers v. nonstutterers. *Journal of Speech and Hearing Research*, 1976, *19*, 481-492.

STARKWEATHER, C. W., & MYERS, M. The duration of subsegments within the intervocalic intervals of stutterers and nonstutterers. *Journal of Fluency Disorders*, 1979, *4*, 205-214.

STEVENS, K. N., & HOUSE, A. S. Perturbation of vowel articulation by consonantal context: An acoustical study. *Journal of Speech and Hearing Research*, 1963, *6*, 111-128.

ST. LOUIS, N. Linguistic and motor aspects of stuttering. In N. Lass (Ed.), *Speech and language: Advances in basic research and practice*. New York: Academic Press, 1979.

STROMSTA, C. Experimental blockage of phonation by distorted sidetone. *Journal of Speech and Hearing Research*, 1959, *2*, 286-301.

TAYLOR, I. K. The properties of stuttered words. *Journal of Verbal Learning and Verbal Behavior*, 1966, *5*, 112-118.

TORNICK, G. B., & BLOODSTEIN, O. Stuttering and sentence length. *Journal of Speech and Hearing Research*, 1976, *19*, 651-654.

UMEDA, N. Vowel duration in American English, *Journal of the Acoustical Society of America*, 1975, *58*, 434-445.

UMEDA, N. Consonant duration in American English, *Journal of the Acoustical Society of America*, 1977, *61*, 846-858.

VAN RIPER, C. *The nature of stuttering*. Englewood Cliffs, NJ: Prentice-Hall, 1973. (a)

VAN RIPER, C. *The treatment of stuttering*. Englewood Cliffs, NJ: Prentice-Hall, 1973. (b)

WALL, M. J. *The location of stuttering in the spontaneous speech of young child stutterers*. Unpublished doctoral dissertation, SUNY, 1977.

WALKER, C., & BLACK, J. *The intrinsic intensity of oral phrases*. (Joint Project Report No. 2). U.S. Naval School of Aviation Medicine, Naval Air Station, Pensacola, FL, 1950.

WEBSTER, R. A behavioral analysis of stuttering: Treatment and theory. In K. Calhoun, H. Adams, & K. Mitchell (Eds.), *Innovative treatment methods in psychopathology*. New York: Wiley, 1974.

WEBSTER, R. Empirical consideration regarding stuttering therapy. In H. Gregory (Ed.), *Controversies about stuttering therapy*. Baltimore: University Park Press, 1979.

WEINER, A. Vocal control therapy for stutterers: A trial program. *Journal of Fluency Disorders*, 1978, 3, 115-126.

WINGATE, M. E. Sound and pattern in "artificial" fluency. *Journal of Speech and Hearing Research*, 1969, *12*, 677-686.

WINGATE, M. E. Effect on stuttering of changes in audition. *Journal of Speech and Hearing Research*, 1970, *13*, 861-873.

WINGATE, M. E. *Stuttering: Theory and treatment*. New York: Irvington, 1976.

YOUNG, M. A. Comment on stuttering frequency and the onset of phonation. *Journal of Speech and Hearing Research*, 1975, *18*, 822-832.

ZIMMERMANN, G. Articulatory dynamics of fluent utterances of stutterers and nonstutterers. *Journal of Speech and Hearing Research*, 1980, 23, 95-107.

ZIMMERMANN, G., & ALLEN, E. Questions concerning Schwartz's "The core of the stuttering block." *Journal of Speech and Hearing Disorders*, 1975, *40*, 135-136.