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Slavoljub D. Djurdjic

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PSYCHOPHYSIOLOGICAL CORRELATES OF ATTENTION AND ANXIETY IN STUTTERERS AND NONSTUTTERERS DURING A SIMPLE REACTION TIME TASK

By

Slavoljub D. Djurdjić

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ABSTRACT

PSYCHOPHYSIOLOGICAL CORRELATES OF ATTENTION AND ANXIETY IN STUTTERERS AND NONSTUTTERERS DURING A SIMPLE REACTION TIME TASK

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Slavoljub D. Djurdjić

This study was designed to investigate the psychophysiological correlates of attention and anxiety in stutterers and nonstutterers during a simple fixed preparatory interval reaction time task. Using a within subjects design, research participants were exposed to two conditions in counterbalanced orders. In one condition subjects were required to read a sentence immediately after the warning signal was presented, while in the other condition subjects were not required to read. It was predicted that speaking during the preparatory interval would interfere with attentional processes and raise anxiety-arousal levels more in stutterers than in nonstutterers and that stutterers would differ in their reactions to the speaking and nonspeaking conditions.

The results indicated that speaking did not interfere with reaction time performance in either group. However, differences in physiological activity suggested that speaking was associated with attentional disorganization in stutterers. During the speaking condition, nonstutterers showed a gradual HR deceleration through the attend portion of the preparatory interval and an anticipatory

acceleration prior to the onset of the respond signal. Stutterers showed HR acceleration during the initial portion of the attend portion of the preparatory interval followed by HR deceleration and no anticipatory acceleration prior to the onset of the warning signal. Stutterers also exhibited greater heart rate variability across trials, greater respiration amplitude changes, and greater differences in respiration frequency during the speaking condition, although in some instances these differences were a function of the order in which the speaking and nonspeaking conditions were presented. While there were no differences between groups in tonic log skin conductance level, electrodermal frequency, or phasic skin conductance responses, there were differences in the correlations between base level log skin conductance level and scores on a measure of anxiety. Stutterers showed an inverse relationship between anxiety and base level skin conductance level for the speaking-nonspeaking order and a positive relationship during the nonspeaking-speaking order. Base level skin conductance and anxiety were not correlated in nonstutterers.

The following factors were suggested as important aspects to be pursued in future research: (1) the length of the preparatory interval, (2) the nature of the preparatory interval, (3) the nature of the speech task, (4) the nature of the performance task, (5) the use of extended trials, (6) the use of magnetic tape for computer analysis of data in order to generate coherence measures of autonomic balance, and (7) the degree of stuttering severity.

To my wife Natasha and son Alexandar.

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CHAPTER I

INTRODUCTION

Stuttering refers to a dysfunction of speech communication characterized by: (a) disruptions in the fluency of verbal expression, (b) mild to intense motor involvement (usually of the speech apparatus) that gives the appearance of a speech-related struggle, and (c) emotional arousal usually taking the form of excitement, tension, fear, embarrassment, irritation, or anxiety (Wingate, 1966, 1976). Speech dysfluency associated with stuttering most often takes the form of involuntary, audible or silent repetitions or prolongations in the utterance of speech elements, sounds, syllables, or words.

Stuttering symptoms vary from individual to individual. In some stutterers all of the characteristics mentioned above are present, while in others only repetitions or prolongations occur. The frequency and intensity of stuttering symptoms also vary. In some individuals stuttering may occur whenever they speak, while in others stuttering occurs only in specific situations (for example, when speaking with strangers, before large audiences, or to a member of the opposite sex). Sometimes stuttering is so severe that the individual cannot communicate effectively verbally, while at other times stuttering may be so mild that it is nearly undetectable.

Between these two extremes there is a wide variety of stuttering behaviors with different degrees of intensity, frequency, and noticeable characteristics.

Stuttering occurs more frequently in males than in females with reported ratios ranging from 2:1 to 5:1. While this sex difference is consistent with the general trend in developmental dysfunctions, and probably relates to the poorer biological viability of the human male (cf. Fitzgerald, 1977), no etiological theory has been advanced to account successfully for this specific difference. Moreover, there are no current theories nor empirical data which suggest that the dynamics of etiology or of therapeutic intervention are different for males and females.

Age of onset varies from individual to individual. Sometimes stuttering first appears in early childhood, while in other instances it does not make its first appearance until adolescence. Some stutterers show a biphasic developmental pattern--stuttering in early childhood followed by spontaneous remission with subsequent recurrence in adolescence. Stutterers sometimes associate their dysfluency with an experience of strong emotional stress, such as the loss of a parent or some frightening experience. However, more often than not, there is no obvious causal factor that can be identified whether it be genetic in origin or experiential. Despite the fact that the etiology of stuttering remains a mystery, many theories have been advanced to explain its development.

Despite differences among theoretical accounts of the development of stuttering, two psychological states, attention and

anxiety, are recurrently cited as important correlates of stuttering. According to one theoretical view (Brajovic & Brajovic, 1974, 1976), attentional disorganization is a key aspect both in the development of stuttering and in the therapeutic rehabilitation of the stutterer. Briefly, this theory attempts to integrate psychological and physiological aspects of behavior disorganization as it relates to stuttering. Anxiety, or to be more specific, speech-related anxiety, often is cited as a causal factor in stuttering (Fibirger, 1972; Johnson, 1948, 1956; Nystal & Muszynski, 1976; Wishner, 1947, 1950, 1952). However, other investigators deny that anxiety is related to stuttering (Wingate, 1976), or consider anxiety to be a secondary component of stuttering which arises as a result of stuttering rather than being causative of stuttering (Bluemel, 1935).

Despite the implications that attention and anxiety are important correlates of stuttering, to date there has been no published research which attempted to identify the explicit role of attention in stuttering, nor any attempt to simultaneously measure these processes during stuttered speech. Therefore, the present study was designed to investigate psychophysiological correlates of attention and anxiety in stutterers and nonstutterers. A simple reaction time task, during which subjects were required to either speak or to remain silent, was used as a measure of performance. As such, this study represents the first study designed to assess directly attentional processes in stutterers using psychophysiological

dependent variables while simultaneously recording similar measures of arousal level which are thought to reflect anxiety.

CHAPTER II

LITERATURE REVIEW

Over the years an extraordinary number of hypotheses have been advanced to account for the development of stuttering. Without too much danger of oversimplification, these hypotheses have been derived primarily from one of three theoretical traditions: psychodynamic (including psychoanalytic), neurophysiological, and learning. This chapter opens with a brief overview of each of these theoretical traditions as they relate to the etiology of stuttering. (For detailed discussions of the theories of stuttering the reader is referred to Van Riper, 1971, and Wingate, 1976).

Cutting across theoretical boundaries, two psychological states--anxiety and attention--are recurrently cited as important aspects of stuttering. Here I am using these terms in their broadest senses. Subsumed within anxiety are tension, stress, upset, and negative emotions, whereas subsumed within attention are aspects of stimulus processing, feedback from muscle systems, cognitive processing, and social contexts as they relate to stuttering. Throughout the literature review, emphasis is given to aspects of the literature which provide support for the hypotheses described at the conclusion of the chapter.

Theories of Stuttering: An Overview

Classifying explanations of the etiology of stuttering as "theories" in fact extends the concept of theory a bit beyond its generally accepted use in science. As Wingate (1976) has succinctly pointed out, explanations of stuttering qualify more as myths than as theories. According to his analysis, all extant theories of stuttering are marked by "inconsistency, assertions of dubious credibility, and assumptions that are not made explicit" (Wingate, 1977, p. 38). Moreover, current theories of stuttering tend to consider only a certain portion of the available evidence, distort empirical findings through re-interpretation, and/or selectively ignore evidence contrary to theoretical assumptions (Wingate, 1976, 1977).

Interestingly enough, Wingate's (1976) own theory of stuttering may be subject to many of the same criticisms that he has leveled against competing theorists. According to Wingate (1976), stuttering is a phonological dysfunction involving linguistic stress. Specifically, stuttering is defined as a dysfunction related to movement into a stressed vowel. Whereas, most speech pathologists assert that stuttering blocks involve consonants, Wingate argues that it is not the consonant per se that elicits stuttering, but rather it is the stressed vowel which immediately follows the consonant which causes stuttering. However, he does not offer an explanation of why stuttering occurs to stressed vowels. Nor does his observation that stuttering is highly associated with stressed vowels easily extend to all languages. For example, in Serbo-Croatian

"vrt" (garden) is a word that frequently elicits stuttering. It is difficult to see how Wingate's hypothesis applies to a word that contains no vowel. To be sure, the letter "r" often has syllabic value in Serbo-Croatian and is thus often used as a vowel. However, it is always pronounced as a consonant and viewed as a consonant by the speaker. Moreover, in Serbo-Croatian the pronunciation of vowels is not affected by their position in a word relative to the stressed syllable. Nevertheless, a more detailed analysis of Wingate's hypothesis as applied to languages other than English is required before one can dismiss his hypothesis as untenable. For example, Langova and Moravek (1966) have offered a phonological explanation for stuttering in Czechoslovakian speakers that is similar to Wingate's analysis. In any event, the important point is that Wingate's hypothesis, even if correct, only helps to explain what stuttering is, it does not help to understand how stuttering develops. Thus, it seems premature to dismiss so readily current theories of stuttering solely on the basis that they have not yet been able to account fully for the developmental process.

Psychodynamic Theory

Essentially, psychodynamic explanations of stuttering emphasize stuttering as a manifestation of personality disturbance. Thus, stuttering is viewed as an overt manifestation of some deeper and covert personality dysfunction. As a result, psychodynamic therapy tends to address the covert disturbance while doing little with the overt behavioral problem, namely, stuttering.

Within the psychoanalytic literature, stuttering has been ascribed variously to restriction of oral gratification during infancy (Alpert & Bernstein, 1964), sibling rivalry (Alpert & Bernstein, 1964; Kolansky, 1960), over-stimulating intimacy with mother in the bathroom (Alpert & Bernstein, 1964), father absence in combination with a variety of factors (Alpert & Bernstein, 1964), regression to oral sadism (Kolansky, 1960, 1967), anal sadism (Kolansky, 1967), and castration anxiety (Olden, 1946).

Otto Fenichel (1945) developed perhaps the most direct psychoanalytic view of stuttering. According to Fenichel, stuttering develops as a result of a fixation centered in the anal stage of psychosexual development. Thus, stutterers are thought to have strong sado-masochistic tendencies; stuttering symbolically representing oral defecation.

Working within the Adlerian framework, Nystul and Muszynska (1976) argue that stuttering reflects a failure in speaking which has become generalized to one's self-concept. In other words, because the individual fails to speak correctly, he comes to view himself as a failure in general. Fear of failure in speaking leads to the development of anticipatory anxiety which takes two forms, psychic and physiological. Psychic anxiety, which develops first, leads to physical anxiety which is manifested in increased muscle tension in the neck and throat. As psychic and physiological anxieties interact the individual becomes too distressed to speak correctly. Failure to speak correctly reinforces the whole process.

Psychodynamic accounts of stuttering are subject to several criticisms. First, they have failed to generate consistent clinical or empirical support. Second, they tend to make the interpretation of the dysfunction fit the observation--in a sense, psychodynamic theories cannot fail to explain stuttering. If one is not oral sadistic, then one is anal sadistic, and so on. Perhaps the most damaging problem, however, is that psychoanalytic theory has failed to provide a reasonably successful therapeutic intervention for stutterers.

Neurophysiological Theory

Neurophysiological theories tend to emphasize disorganization in neurohumoral mechanisms or in brain lateralization as etiologic factors in stuttering. For example, Fibriger (1971) links stuttering to physiological tremor in the motor speech apparatus. Recording electromyographic activity of the muscles of the mouth and chin, Fibriger found that stutterers show high amplitude physiological tremor which differs in frequency (8 Hz) from tremor which occurs during fluent speech (10 Hz). (Physiological tremor contains Hz components up to about 30 Hz and is characterized by higher amplitudes with decreasing frequencies.) The frequency of tremor during stuttering is similar to the frequency found in morbidly anxious individuals and in normal individuals when under conditions of stress. Although several investigators have reported slower frequency and higher amplitude EEG alpha activity to be associated with stuttering (e.g., Fritzell, Petersen, & Sellden, 1965), Lippold and

Novotny (1974) argue that this alpha activity is an artifact of physiological tremor. Interestingly enough, the tremor activity Fibinger reports tends to occur during pre-phonation, a finding which at least is consistent with the hypothesis offered by Wingate (1976) and Langova (1967).

Fibriger suggests that parental concern over normal developmental speech dysfluency during the preschool years creates a stressful situation for the young child. Physiological tremor is induced by stress indirectly and by increased adrenaline secretion directly. Every speech situation, being stress producing, leads to increased adrenaline secretion which causes physiological tremor and stuttering accompanied by parental anxiety. Parental anxiety in turn induces stress, and the cycle repeats.

There are at least two major problems with Fibriger's (1971) analysis. First, it does not answer the basic question of why some individuals stutter during stressful interactions and why some do not. Second, if higher levels of blood adrenaline induce stuttering, then why do many individuals fail to stutter when placed in emotionally frustrating or anger arousing situations? Both of these conditions are associated with increased adrenaline secretion. Obviously, Fibriger's theory needs to be able to account for these problems before it can be accepted as a full explanation of stuttering. Nevertheless, the approach does show promise and does link physiological activity with the speech act and with stress.

Several authors have alluded to dysfunctions in the organization of the central nervous system (CNS) as important determinants

of stuttering. Quite some years ago, Orton (1927) suggested that mixed cerebral dominance played an important role in the etiology of language disorders in general, and in stuttering specifically. According to Orton, poor lateralization of speech centers in the left hemisphere leads to a rivalry between the two hemispheres over the dominance of speech functions. Accordingly, stutterers have great difficulty integrating the units of motor sequences into sequential language because of poorly established cerebral lateralization.

Orton's theory was supported by Travis (1931), who based his hypothesis on a report that a disproportionate number of stutterers are left-handed (Bryngelson, 1927). Travis assumed that forcing originally left-handed individuals to use their right hand would lead to poorly established cerebral lateralization for speech functions and to ambidextarity. If the left-handed individual was allowed to use the left hand, speech centers would be well lateralized in the right cerebral hemisphere and stuttering would not develop. In thwarted left-handers, lateralization for hand dominance and for speech functions would be poor, leading to mixed dominance, rivalry between the hemispheres, and stuttering.

Unfortunately, empirical evidence for mixed dominance in stutterers is poor and conflicting. At present, no neurophysiological evidence exists which indicates that hemispheric centers for hand function and speech function are closely associated, or that disorganization of one of the centers is accompanied by disorganization of the other center. Whereas it is true that thwarted left-handed individuals can be found among stutterers, many are not

ambidextrous. In any event, cerebral dominance aspects of stuttering have received renewed interest in recent years (e.g., Brajovic & Brajovic, 1974, 1976) and cannot be dismissed completely.

Recently, the dichotic listening task and bilateral tachistoscopic word perception task have been employed in studies attempting to test the Orton-Travis hypothesis. These tasks are used to assess speech perception. In the dichotic listening task, pairs of different words (syllables or digits) are presented simultaneously to both ears via headphones. The subject's task is to report what has been heard in any order desired. Between ear differences in accuracy are measured. The assumption is that because contralateral connections between the ears and the cerebral hemispheres are stronger than are ipsilateral connections, better right ear scores should be reported for normal individuals because the speech centers are in the left hemisphere. Conversely, no significant differences should occur for stutterers who are assumed to have poorer left hemispheric organization for speech. Results of the studies testing this hypothesis are conflicting. Some authors have found the expected difference (e.g., Curry & Gregory, 1969) and some have not (Sussman & McNeilage, 1975).

Using the bilateral tachistoscopic word perception task, Moore (1976) found differences between stutterers and nonstutterers. Nonstutterers showed a significant right visual half-field preference. In other words, they recalled significantly more words presented to the right visual field than words presented to the left visual field. On the other hand, stutterers showed no differences in recall

as a function of right-left visual field presentation. These data are consistent with the Orton-Travis hypothesis if one considers disturbances in speech perception as an important dimension of stuttering. But can one do so? Without sufficient evidence that speech perception influences speech production such as that involved in stuttering, it is difficult to see how perception tasks can assist in our understanding of stuttering as a product of poorly established cerebral lateralization for speech function. Unfortunately, an adequate test for measuring cerebral lateralization for speech production has yet to be developed.

Learning Theory

Without question, learning theory and therapeutic interventions based on learning theory dominate contemporary approaches to stuttering. Wishner (1947, 1950, 1952) generally is recognized as having been the first to apply notions of instrumental learning to the problem of stuttering. According to Wishner (1947) stuttering is learned and persists because it is instrumental in avoiding punishment, which in this case consists of disapproval associated with the original nonfluent utterance. Stuttering represents an attempt to avoid painful reactions contingent upon stuttering. However, as Wingate (1966) pointed out, ". . . the avoidance response (stuttering) persists even though it does not avoid the punishment (painful reactions)."

Wishner (1947) also applied instrumental escape learning to the problem of stuttering. According to this explanation, the

speech situation raises the level of tension in stutterers. Stuttering is seen as instrumental in tension reduction and is, as such, reinforcing. Thus, stuttering provides the individual a way to escape from an aroused state of tension. Because stuttering is associated with tension reduction, it is reinforced and persists. But, is this really the case? Whereas it can be accepted that speech situations raise the level of tension in many stutterers and that higher levels of tension can contribute somehow to stuttering, clinical observations suggest that stuttering does not lead to tension reduction. My own clinical observations suggest that stuttering often is accompanied psychologically with embarrassment and avoidance of social interaction, and physiologically with evidence of marked increases in arousal level (e.g., blushing, palmar sweating, tachycardia). If stuttering was tension reducing, one would expect more positive outcomes after the occurrence of a stuttering episode.

According to Goldiamond's (1965) operant model, stuttering begins as normal dysfluency associated with early speech development. These early dysfluencies are positively reinforced, perhaps by parents who consider the behavior "cute" or by direct parental imitation of the behavior. After a time, aspects of the speech situation become discriminative occasions for stuttering. However, as the speech dysfluency increasingly comes under stimulus control, parents shift their view of the dysfluency from a behavior perceived to be "cute" to one perceived to be unacceptable. Consequently, parents begin to punish the child for the dysfluent speech.

Punishment, whether from others or from self, creates tension, anxiety, or fear associated with speaking and shows up as stuttering.

The role of anxiety or fear in stuttering is brought to the fore in Brutten and Shoemaker's (1967) two-factor theory of stuttering. According to the two-factor view of stuttering, there are two aspects involved with the stuttering act: one associated with the speech act itself, and one associated with correlates of the speech act (e.g., eyeblinks, articulatory postures, etc.). Conditioned emotional responses are thought to account for the emotional component of stuttering, whereas instrumental conditioning is thought to account for stuttering itself as well as for the correlates of stuttering. Brutten and Shoemaker argue that stuttering develops in a three-stage sequence. Initially, speech dysfluencies are caused by poorly organized cognitive and motor skills. The speech dysfluencies become associated with negative emotional states (i.e., frustration, anxiety, fear). Then through classical conditioning the fluency failures and negative emotions become associated with certain stimuli, as for example, arise in interpersonal speech interactions where parents may give nonverbal facial stimulus cues of disapproval. Finally, as the range of stimuli to which the negative emotional responses become associated increases, the speech act itself acquires stimulus value for eliciting discriminative cues.

As Wingate (1977) points out, the major problem with the learning theory explanations of stuttering is that many stutterers do not have negative emotions, and the specific stimuli sufficient to evoke stuttering have not been well detailed.

A Note on the Role of Heredity in Stuttering

There are recurrent suggestions in the literature that stuttering is due to hereditary rather than to environmental factors. However, there is little evidence to support a pure hereditary view of stuttering etiology. To be sure, many investigators have reported a higher incidence of stuttering among children of stutterers than among children of nonstutterers (Andrew & Harris, 1964; Bryngelson & Rutherford, 1937; Darley, 1955; Freund, 1952; Gutzman, 1924; Wepman, 1939; West, Nelson, & Berry, 1939; Wingate, 1964). Nevertheless, efforts to link stuttering to hereditary mechanisms have failed to partial cultural learning factors from hereditary factors. Even the study of monozygotic twins has failed to provide convincing evidence to support a hereditary explanation of stuttering (Sheehan & Costly, 1977). Yet, there is an obvious biological component to stuttering. Sheehan and Costly (1977) argue that the 5:1 ratio of male to female stutterers provides evidence for a biological predisposition in males, most likely polygenetically transmitted. Coleman (1976) has attempted to link genetic mechanisms in stuttering with the sex chromosomes. Considering all current evidence, Sheehan and Costly suggest that an hereditarian explanation can account for about 25% of stutterers but they do not hold firmly even to this estimate. Clearly, there are no compelling data to support an hereditary explanation of stuttering; certainly none as compelling as learning or psychobiological explanations, or for that matter, psychoanalytic ones.

Summary

The theories noted above are representative of those that have been offered as explanations for the etiology of stuttering. To date, no single theory has garnered sufficient empirical evidence in its support to permit the complete dismissal of competing theories, although there clearly is a preference for cognitive learning theories to the near exclusion of psychodynamic theories.

What the current literature does support is that stuttering is a disorder of multifactorial causation, including both biological and psychological factors. Stuttering is a complex dysfunction which has a variety of clinical symptoms that are present in various degrees from one individual to another. Stutterers differ in the frequency of stuttering, the intensity of stuttering, the types of observable characteristics of stuttering, and the age at which stuttering first develops. Stutterers also vary greatly in the degree to which they respond to different therapeutic interventions. Thus, neurophysiological and psychological factors may contribute to the development of stuttering in different ways for different individuals. In some stutterers neurophysiological factors may play a more important role than psychological factors and in other stutterers the opposite may be the case. According to Cohen and Hanson (1966) the many interactions among neurological and psychological factors probably account for the marked individual differences in onset, persistence, and recovery one finds among stutterers.

Nevertheless, there is a consistency that cuts across theoretical boundaries, namely, the notion that anxiety (nervous tension, fear, upset, etc.) plays a fundamental role in stuttering. Moreover, if one takes a broad view of the role that attention plays in the organization of behavior, most neurophysiological and learning theory accounts of stuttering suggest a dysfunction in attention underlying stuttering behavior. Consequently, it seems appropriate to briefly consider these two processes as they relate to stuttering.

Stuttering and Anxiety

Anxiety produced by some stimulus present in the speech situation has been cited repeatedly as an important psychological factor contributing to the occurrence of stuttering (Fibriger, 1971; Johnson, 1948, 1956; Wishner, 1947, 1950, 1952). In my own clinical practice I have found that the frequency of stuttering among adolescents and adults is most pronounced during the initial interview with the therapist. During medical examinations in such situations, the clinical physiological indices of heightened arousal are obvious (increased heart rate, increased respiration rate, and easily noticeable palmar sweating). Nevertheless, there is no clear-cut relationship between autonomic nervous system activity and anxiety in stutterers. Some investigators have reported no ANS correlates of stuttering whereas others have reported reduction in systolic blood pressure, increases in skin conductance (see Perkind, 1971), and increases in tonic heart rate (Brajovic & Brajovic, 1974, 1976).

Patient self-reports indicate that much stuttering occurs in some anxiety producing situations, as for example, when taking an oral examination in school, when buying a movie ticket and being pressured to move quickly by other customers, and when speaking with others with whom one is trying to make a favorable impression. Upon occasion I have gone with my patients to shops or to travel agencies when they had to acquire some information. Many of them report that they have a fear of failure concerning fluent speech. Others report that they are afraid that others will embarrass them, laugh at them, or be impatient with them should they stutter. Although all stutterers do not have difficulty in such situations, some show a pronounced increase in the severity of stuttering and in some instances the severity is so great that the individual cannot speak at all.

Based on the patients that I have observed in my own clinic, children who stutter are less anxiety-ridden in speech situations than are adolescents and adults. Puberty appears to mark the point developmentally when anxiety emerges as an important concomitant of stuttering. Thus, it is tempting to hypothesize that anxiety may play a different role in inducing stuttering in different age groups. Since adolescence also is a period of great self-examination as well as a period when significant changes in the nature of interpersonal relationships occur, the emergence of anxiety as a correlate of stuttering would suggest a strong cognitive-learning component to the emotional reaction. Although anxiety may lead to increased frequency, intensity, and persistence of stuttering, it

has yet to be solidly demonstrated that anxiety is a primary component of stuttering. Moreover, there are many stutterers who are not particularly anxiety-ridden.

Psychophysiological Measures of Anxiety

Palmar sweating, a physiological indice of sympathetic nervous system activity, has been considered for years to be a reliable ANS measure of such emotional states as fear and anxiety. For example, emotions such as fear should be accompanied by activation of any defensive emotion represented in the skin reflex, such as sweating. Skin resistance as a measure of arousal or activation is registered by electrodes which are placed on a portion of the skin surface which is rich in sweat glands. Kuno (1934) linked emotional sweating to high arousal level and argued that the response was limited to the palms of the hands and the soles of the feet and these sites have remained the preferred recording placements. When a small current is driven through the skin it acts as a resister. The amount of resistance that the skin has depends on the amount of sweat present on the skin surface. Since sweat is a better conductor than skin per se, it induces lowering of skin resistance. Thus, the degree of decreased skin resistance depends upon the degree to which sweat gland activity increases. Today, skin conductance (SC), the reciprocal of skin resistance, is the preferred unit of measurement. The phasic SC response (SCR) has a typical latency of about 1.5 sec and requires from .5 sec to about 5 sec to reach its peak.

Repeatedly, electrical activity recorded on the skin surface has been shown to be reflected in tonic elevations (general activation, arousal, or anxiety) and phasic reactions (specific activation or orientation reactions and/or conditioned responses) to situational demands. Mason (1941) correlated changes in electrodermal activity with various emotional and cognitive states. The results of her study suggested that conditions associated with a high degree of uncertainty elicit larger amplitude skin resistance changes than do conditions associated with a high degree of certainty. Mason argued that the significance of a situation with regard to its relevance in attaining a goal toward which an individual was motivated was associated with the extent of electrodermal change. Edelberg (1972) found that threatening situations are powerful stimuli for eliciting electrodermal change based on increased palmar conductance. Wilson (1967) obtained perfect separation between normal subjects and those with phobias by comparing their electrodermal responses to spiders and landscapes.

In one of the few published studies linking electrodermal activity to stuttering, Bruten (1963) compared fluent and dysfluent subjects on the adaptation task (repeated oral readings of the same passage). Decrease in stuttering frequency during repeated readings of the passage was associated with decrease in palmar sweating. Fluent speakers showed no relationship between repeated reading and electrodermal activity.

Assuming that speech situations produce anxiety in stutterers and that anxiety is associated with increased sympathetic activity

resulting in increased palmar sweating, the extent of galvanometric change should differ between stutterers and fluent speakers. Conversely, speech situations should not produce significant anxiety in fluent speakers. Thus, in the present experiment, SC is recorded and used as a measure of the subject's activation level. Tonic activation is reflected by changes in SC level (SCL) which indicate changes in arousal level over the course of the experiment. Phasic activation is reflected by changes in the SCR, which indicates the subject's response to specific stimulation.

Stuttering and Attention

Cvetko Brajovic, director of the Institute for Psychophysiological and Speech Disorders, Belgrade, Yugoslavia, has developed a set of therapeutic procedures for speech dysfunction based mainly on the neurophysiological and cognitive-learning theory traditions in speech pathology (Brajovic & Brajovic, 1974, 1976; Brajovic, Brajovic, & Ivanus, 1974). Brajovic and his associates argue that an attentional dysfunction is one of the key factors (although not the only factor) contributing to the development and persistence of stuttering. Aspects of the attentional dysfunction which have corresponding therapeutic exercises relate mainly to the neural feedback and control mechanisms which link the ascending reticular activating system (activation-arousal), the pyramidal and extrapyramidal motor systems (muscular control), the thalamic and hypothalamic projection systems (emotional reactivity), and the central neural integration system (cognitive control). Brajovic's

therapeutic techniques have claimed enormous success compared to most other therapeutic methods applied to stuttering and to dysfunctions involving speech defects (Brajovic, Fitzgerald, Brajovic, & Ivanus, 1974; Brajovic, Fitzgerald, Novakovic, & Bojanovic, 1974; Brajovic, Fitzgerald, Polomcic, Cuk, & Bojanovic, 1974; Brajovic, Fitzgerald, Polaincic, Djurdjevic, Cuk, Mihajlovic, & Dolnicar, 1976; Brajovic, Negovanovic, Brajovic, Fitzgerald, Djurdjevic, Bojanovic, & Tufegdzic, 1975; Fitzgerald, 1977). Brajovic and Brajovic (1974) reported an 80% success rate on the basis of a 3-year study of their therapeutic method. Success was defined as discontinuation of therapy and the absence of stuttered speech.

Although success in the Brajovic therapy requires the stutterer to develop highly organized powers of selective attention and to integrate a variety of attention-demanding tasks, to date there has been no direct empirical support for the notion that attentional dysfunction is a prominent component of the stuttering act. Nevertheless, there is sufficient clinical and empirical evidence of an indirect sort to justify the hypothesis that attentional dysfunction is one characteristic of the stutterer.

Sherrard (1975) attempted to induce stuttering in fluent speakers by setting up two conditions, an anxiety condition and a divided attention condition. Sherrard used a masking task in which auditory feedback was masked with noise and pure tone. There were four conditions and two groups. In the first condition, subjects were required to read a prescribed passage. In the second condition, subjects read the passage while at the same time they traced a

stylus maze with their right hand (divided attention task). The third condition was the same as the first except that auditory feedback was masked. The fourth condition was the same as the second with the addition of auditory feedback masking. One group of subjects received electric shocks to their left hand administered randomly during the divided attention task. The other group did not receive shocks. Results of the study were intriguing. Whereas electric shocks cannot be considered to induce anxiety in the same way that speech situations induce anxiety for stutterers, the results connected with divided attention can be related to some elements of stuttering behavior. Compared to the reading-only task, the divided attention task results revealed a significant increase in stuttering-like speech. In the masked auditory feedback condition there was a significant decrease in stuttering-like speech both in the reading only condition and the divided attention condition. Sherrard related her results to stuttering in general, suggesting that division of attention between auditory feedback and other features of the speech situation are the immediate causes of stuttering. Thus, in speech situations stutterers are thought to divide their attention between auditory feedback and other features of speech (for example, ideation, word selection, etc.). Anxious attention to auditory feedback, according to Sherrard, can lead to false alarm reactions (reactions to erroneous perception of signals) and to disruption of smooth verbal performance. Masking auditory feedback leads to termination of the "distracting" stimulus and to

termination of divided attention. The result is a decrease in stuttering as seen in the masked auditory feedback situation.

The results of Sherrard's study as well as the results of other research indicate that there are significant decreases in stuttering frequency when stutterers cannot hear the outcome of their speech (Cherry, Sayers, & Marland, 1956; Murray, 1969; Maraist & Hutton, 1957; Burke, 1969). These studies suggest that attentional dysfunction may contribute to the occurrence of stuttering. In my own clinical practice, I have often heard stutterers emphasize that in speech situations their first concern is the outcome of their speech. Their attention appeared to be directed more to "How will I speak," than to "What will I say?" Some of my patients claim that they help themselves in speech situations by playing with a coin in their hand, passing the coin from finger to finger. Perhaps they inadvertently discovered a way to distract their attention from the speech situation.

As I have mentioned previously, many stutterers evidence signs of heightened anxiety (arousal) during initial medical interviews conducted at our clinic. However, many stutterers do not show these symptoms. From my own experience and that of my colleagues, it seems that stutterers who do not show physiological signs of high arousal (who either have normal or bradycardic heart rate, regular respiration rate, minimal muscular tension) are more difficult to cure with Brajovic's therapeutic methods, independent of the severity of stuttering. Perhaps different etiologic factors are present in these two subgroups of stutterers. It seems most likely that

sympathetic arousal would be the predominant ANS state in stutterers who show physiological signs of high arousal, whereas in the other subtype autonomic balance may be either normal or with a predominance of parasympathetic activity. Moreover, it seems that anxiety and attentional dysfunction may be crucial factors in inducing stuttering in the latter group. Several authors have shown that high arousal is correlated with a narrowing of attention (Callaway & Stone, 1960; Easterbrook, 1959; Hockey, 1970; Wachtel, 1968; Zaffy & Brunning, 1966). Anxiety in speech situations, being a state of high arousal for the speaker, may narrow attention in stutterers so that they attend primarily to auditory feedback. Other features of the speech context (word selection, cognitive content, ideation) may be less attended to. Thus, the high arousal stutterer may begin to speak before thinking through what he wants to say, or how he wishes to express his thoughts. Not having been selected in an appropriate manner, several words may compete and be discharged at the same time, thus precipitating stuttering. Observations that the speech of most stutterers is faster than normal and that even some fluent speakers stutter in certain speech-anxiety situations have been reported (Bode & Bruten, 1963).

It cannot be stated too strongly that the validity of the above speculations is dependent at least on the initial demonstration of attention deficit in stutterers. Until such demonstration is made, discussion of types of stutterers remains speculative at best. Nevertheless, if stutterers while speaking attend anxiously to auditory feedback and attend less to other internal and external

information, it would seem that a task requiring attention to external information would show differences in performance between stutterers and fluent speakers. The reaction time (RT) task provides one situation where one can assess performance as it relates to attention. In the simple RT task one must attend to two stimuli, one which "warns" the subject preparatory to the presentation of the other or "respond" stimulus. The time interval between the two signals is the preparatory interval (PI). If one requires the subject to speak during the PI and if speaking is associated with attentional dysfunction in stutterers then one should observe poorer RT performance as a result of disorganization in the attending process. Moreover, psychophysiological dependent variables associated with attention (heart rate and heart rate variability) and anxiety (SC) should reflect differences in RT performance. Prior to specifying the specific hypotheses to be tested in the present study, let us turn briefly to consider psychophysiological correlates of attention, with specific emphasis on the RT task.

Psychophysiological Measures of Attention

Phasic heart rate (HR) change, particularly HR deceleration, has been indicated by many researchers as reflecting attentional preparation for the intake of environmental stimulation (Hahn, 1973; Krupski, 1975; Lacey, 1959, Lacey & Lacey, 1974; Lacey et al., 1963; Luria, 1973; Obrist et al., 1974; Sroufe et al., 1973). Lacey (1959) argued that attention to external events is associated with HR deceleration, whereas cognitive processing, inferentially involving

motivated rejection of irrelevant external events, is associated with HR acceleration. The RT task has been used in most of the studies to test Lacey's hypothesis about cardiac correlates of attention. Most researchers have reported that HR deceleration occurring just after the onset of the warning signal is positively correlated with RT latency (Hahn, 1973; Krupski, 1975; Obrist et al., 1974; Sroufe et al., 1973).

In a study conducted with children selected to conform to the description of "minimal brain dysfunction," in whom attentional dysfunction is assumed to be primary, Sroufe et al. (1973) found that the children had significantly slower RT and significantly less HR deceleration to the onset of the warning signal than did normal children. Normal children showed significant negative between subjects correlation between mean magnitude of HR deceleration and average response latency--children with larger average HR deceleration had faster RTs. Similar results were reported by Lacey and Lacey (1974) in a RT study with normals. Krupski (1975) compared normal adults with retarded adults and found that retardates had significantly slower RTs and lower magnitude HR decelerations following the warning stimulus. While most authors agree that HR deceleration can be accepted as an indice of some component of attentional processing, Porges (1970) questions this hypothesis.

In a study using fixed and variable preparatory intervals, Porges (1972) found that subjects who exhibited the greatest decrease in heart rate variability (HRV) during the PI tended to have faster RTs. A significant relationship also was found between

the magnitude of the respiratory change from the pre-respond period to the post-respond period and RT. The greater the increase in respiratory amplitude following the warning signal, the shorter was the response latency. At the same time, no significant relationship between the degree of HR deceleration prior to the respond signal and RT was found. Porges assumes that HR deceleration during the PI in a fixed PI RT task includes a component of temporal conditioning (see Fitzgerald & Porges, 1971). More recently, he has elaborated his theory concerning the physiological responses associated with various kinds of attention and distinguishes two physiological responses, one reactive and one sustained (Porges, 1976). The reactive component of attention is composed of vagally mediated short latency responses (orientation reactions) which are generally decelerative and occur within approximately 1 sec after the warning stimulus is presented. This response is followed by a longer latency response which occurs approximately 2-6 sec following stimulus change. The magnitude and direction of these long latency responses are a function of stimulus signal value (accelerative in response to important or intense stimuli, and decelerative in response to novel or mild stimuli). These responses are primarily vagally mediated. When accelerative, then vagal inhibition is probably coupled with generalized sympathetic activation. When decelerative, vagal discharge masks sympathetic effects. The sustained component of attention is more active, instrumental, and is manifested by a reduction in HRV as well as reduction in respiratory amplitude variability. The sustained component of the attentional

reaction occurs approximately 6 sec following onset of the warning signal and continues during the period required of the subject to sustain attention (the upper limit of this reaction has yet to be specified). According to Porges, the sustained attention response is vagally mediated and reflects increased parasympathetic influence on HR resulting in greater coupling between HR and respiration. Heart rate is often reduced in the situations of sustained attention although reduction in HRV can occur without a directional change in HR. The sustained attention response also includes increased respiration rate and decreased respiration amplitude.

Keeping in mind psychophysiological studies using the RT task, use of a fixed PI should provide a situation in which development of the attentional response is maximized. Consequently, the present study employs such a procedure. In addition, measurement of HR, HRV, respiratory rate, and amplitude throughout the RT task will allow a test of Porges' hypothesis concerning the various components of the attentional process in the RT task. Finally, this technique should provide a means of testing the main purpose of the present study, the determination of whether stutterers and nonstutterers show differences in attention.

Hypotheses

The present study was designed to investigate attentional and anxiety correlates of speech dysfluency using a fixed PI RT task. Based on the preceding literature review, the following hypotheses were developed:

Reaction Time Performance

1. If auditory feedback is a distracting stimulus for stutterers, then they should be less attentive to external task demands when they are required to speak than when they are not required to speak. Thus:

a. stutterers should have longer latency RTs when speaking than when not speaking,

b. nonstutterers should have no differences in RT latency as a function of speaking or not speaking, and

c. stutterers should differ from nonstutterers during speaking but not during nonspeaking conditions.

Attention

2. If stutterers are less attentive to external task demands when they are required to speak than when they are not required to speak, then

a. stutterers should show less HR deceleration, less decrease in HRV, and less increase in respiration amplitude during the PI during speech compared to nonspeech conditions;

b. nonstutterers should exhibit no differences in HR deceleration, HRV, or respiratory amplitude as a function of speaking or not speaking;

c. stutterers should show less magnitude HR deceleration to the warning signal in the speaking condition than do nonstutterers; and

d. they also should show less decrease in HRV during the PI in the speaking condition than do nonstutterers and less increase in respiration amplitude following the reaction signal in the speaking condition compared to nonstutterers; and

e. there should be no difference between stutterers and nonstutterers in HR and respiration in the nonspeech condition.

Anxiety-Arousal

3. If speech situations induce anxiety in stutterers, then

   a. stutterers should show significantly higher SCLs (tonic) and lower magnitude SCRs (phasic) than nonstutterers during speaking, but not during nonspeaking conditions;

   b. stutterers should show higher SCLs and lower magnitude SCRs in speaking than in nonspeaking conditions; and

   c. nonstutterers should exhibit no differences in SC activity between speaking and nonspeaking conditions.

CHAPTER III

METHOD

Subjects

Twenty young male adults (10 stutterers, 10 nonstutterers) participated in the study. Stutterers ranged in age from 18 to 30 years (Mean = 22.6 years), and nonstutterers ranged in age from 19 to 24 years (Mean = 21.6 years). Stutterers were contacted through the Michigan State University Department of Audiology and Speech Sciences. Only stutterers with no current or previous history of neurological dysfunction related to speech function were accepted as research participants. Each stutterer received financial remuneration for his participation. Nonstutterers were obtained from introductory psychology classes and received extra course credit for their participation. Only nonstutterers with no current or previous history of speech dysfluency were accepted into the study.

Apparatus

The reaction time (RT) stimuli consisted of two lights (one red, one green) mounted on a wall in a sound attenuated experimental booth (ambient noise level 50 dB re .2000 dynes/cm^2), located approximately 2 ms distance from the subject. Stimulus presentation and duration was controlled automatically by a series of electronic timers. Standard electronic timers were used to record RT, which

was obtained from a button-push device. Physiological activity was recorded continuously on a Grass Model 7 polygraph as were all stimulus events. A Grass Model PT 5A volumetric pressure transducer was used in conjunction with a Phipps-Bird pneumograph to record thoracic respiration.

Dependent Variables

Heart Rate (HR)

For recording HR, EKG Leads II and III were used. Prior to the application of the electrodes, the recording sites (left leg, right arm, left arm) were cleaned with 70% ethanol. Grass Ag electrodes (contact area .12 cm^2) filled with Beckman Offner Paste (sodium cloride base) were attached to the recording sites and connected with the polygraph.

Skin Conductance (SC)

SC was recorded from the hypothenar eminence of the subject's nonpreferred hand. Prior to applying Ag Ag/Cl electrodes (surface area .78 cm^2) the subject's palm was prepared with 70% ethanol, slightly abraded, and allowed to dry (see Bundy & Fitzgerald, 1975). The electrolytic medium was a Unibase preparation (Lykken & Venables, 1971). Electrodes were applied about 1.5 cm apart.

Respiration

Respiration was registered by placing a Phipps-Bird pneumograph (rubber bellows type) around the subject's thoracic area. Inspiration and expiration pressure changes in the bellows were

transduced into voltage by a Grass volumetric pressure transducer. The transducer was connected to a Grass Model 7 polygraph located in a room adjacent to the subject room.

Reaction Time (RT)

The RT signal consisted of two lights, a warning light (red) and a reaction light (green). The warning signal was of 1 sec duration, and the reaction signal was of 500 msec duration. Onset and offset of each light was registered on-line on the polygraph. The RT task required the subject to push a button when the reaction signal came on. Latency of button pushing was registered electronically and recorded on the polygraph in msec.

Questionnaires

Three questionnaires were administered to stutterers in order to assess their reactions to speech situations (Stutterer's Self Rating of Reactions to Speech Situations), their reactions toward stuttering (Iowa Scale of Attitudes Toward Stuttering), and their levels of social sensitivity and anxiety (Willoughby Questionnaire). These are clinical instruments used to establish diagnosis and treatment for stutterers. Nonstutterers completed the Willoughby Questionnaire and the Iowa Scale but not the Stutterer's Self Rating Scale.

The Stutterer's Self Rating of Reactions to Speech Situations consists of 40 items each rated on a 5-point scale for each of four categories: Avoidance, Reaction, Stuttering, and Frequency. For example, to answer the item, "Introducing one person to another,"

one can choose one of five statements in each category. For the Avoidance category, a rating of 1 indicates that the subject never tries to avoid the situation, whereas a rating of 5 indicates that the subject avoids the situation at all costs. For the Reaction category, a rating of 1 indicates that the subject definitely enjoys speaking in the situation, whereas a rating of 5 indicates that the subject dislikes speaking in the situation very much. For the Stuttering category, a rating of 1 indicates that the stutterer stutters seldom if at all in a given situation, whereas a rating of 5 indicates that stuttering is severe in the given situation. Finally, for the Frequency category, a rating of 1 indicates that the stutterer often finds himself in situations that involve speech, whereas a rating of 5 indicates that the stutterer seldom finds such situations. Each category is scored as a sum of the ratings per item divided by the number of items answered. A Low average represents a positive self-rating and a High average represents a negative self-rating.

Shumak's (1942) analysis of 95 adult male stutterers' (17-30 years old) responses to the Self-Rating questionnaire revealed the following normative data for each of the categories: Avoidance (M = 2.31, SD = .66, Range = 1.02-4.20); Reaction (M = 2.57, SD = .62, Range = 1.41-3.97); Stuttering (M = 2.54, SD = .60, Range = 1.20-3.92); and Frequency (M = 3.80, SD = .45, Range = 2.39-4.72). Shumak also found correlations of .52 between Avoidance and Stuttering, .84 between Reaction and Avoidance, and .57 between Stuttering and Reaction categories. Correlations corresponded to

specific situations presented in each of the items of the questionnaire. (For more details regarding this instrument the reader is referred to Johnson, Darley, & Spriestersbach, 1952).

The Iowa Scale of Attitudes Toward Stuttering consists of 45 items each of which is rated on a 5-point scale ranging from 1 (Strongly Agree) to 5 (Strongly Disagree). The subject is instructed to circle the rating that best describes his own feelings. The questionnaire is scored by adding the number of Strongly Agree responses and multiplying by 4, adding the number of Moderately Agree responses and multiplying by 3, adding the number of Moderately Disagree responses and multiplying by 2, and adding the number of Strongly Disagree responses and multiplying by 1. The four scores then are added to give an aggregate "total" score. The total score is then divided by the sum of items answered across categories. A low score represents better attitudes toward stuttering, whereas a high score is thought to reflect an unsatisfactory attitude toward stuttering. According to Johnson et al. (1952), scores ranging from 1.0 to 1.4 reflect positive attitudes and considerable tolerance for stuttering; scores ranging from 1.4 to 2.2 reflect average or moderate attitudes toward stuttering; and scores above 2.2 reflect poor attitudes and considerable intolerance for stuttering. Split-half reliability of the scale is reported to be .89 (Johnson et al., 1952).

The Willoughby Questionnaire is an index of social sensitivity and anxiety often used as a clinical tool in therapeutic work with stutterers. The questionnaire consists of 25 items each of which is

rated on a 5-point scale ranging from 0 (no, never) to 4 (practically always, entirely, etc.). The higher the number used in answering the item, the higher the subject is thought to be in anxiety and social sensitivity. Scores range from 0-19, 20-39, 40-59, 60-79, and 80-100 for normal, mild, moderate, moderately extreme, and extreme anxiety and social sensitivity, respectively.

General Procedure

Before the experiment began, subjects were shown the subject room and given a general explanation of what was expected of them in terms of the RT task. When their questions had been answered, they were asked to read and sign an informed consent statement (see Appendix A).

Table 1 illustrates the experimental procedure. Within each group, subjects were randomly assigned to one of two condition orders (Speaking-No Speaking; No Speaking-Speaking). In the Speaking condition, subjects were instructed to read a sentence immediately after the warning signal came on, to finish reading the sentence before the reaction signal came on, and to push the RT button as quickly as possible after the reaction signal came on (see Appendix B for detailed instructions administered to subjects). In the No Speaking condition, subjects were instructed to prepare to push the button when the warning signal came on and to push the button as quickly as possible after the reaction signal came on.

When the experimenter was assured that the subject understood the task, the electrodes and bellows were attached and the polygraph

Table 1

Experimental Design Used to Study Attentional Processes and Anxiety (Arousal) in Stutterers and Nonstutterers During a Simple RT Task

Group	Order	Baseline	Reaction Time Trials			
			1-10	Baseline	11-20	Baseline
Stutterers (N = 10)	A (N = 5)	30 sec	PI Condition: Speaking	30 sec	PI Condition: No Speaking	30 sec
	B (N = 5)	30 sec	PI Condition: No Speaking	30 sec	PI Condition: Speaking	30 sec
Nonstutterers (N = 10)	A (N = 5)	30 sec	PI Condition: Speaking	30 sec	PI Condition: No Speaking	30 sec
	B (N = 5)	30 sec	PI Condition: No Speaking	30 sec	PI Condition: Speaking	30 sec

was checked to be certain that all physiological events were being registered free of artifact. From that point on the procedure followed that depicted in Table 1. A 30-sec period of baseline physiological activity was registered prior to Trial 1. Trials 1-10 consisted of the assigned condition for the PI. Figure 1 illustrates a single RT trial. As indicated, the PI was a constant 12 sec. Intertrial intervals varied randomly from 15 to 30 sec ($\underline{M}$ = 22.5 sec). There were 10 trials in each condition. After the first 10 trials were completed a second 30-sec baseline registration was made.

Between trials 10 and 11 a 5-min rest period was used to read instructions for the next condition and to check electrode placements and polygraph signals. Prior to Trial 11, a third 30-sec baseline registration was made. During trials 11-20 each subject performed the condition to which he was assigned. A fourth 30-sec baseline registration was made immediately after trial 20. Following the RT task subjects went to a second experimental room and completed the questionnaires.

"Distraction" Sentences

Construction of the "distracting" sentences used during the Speaking condition took into account both clinical and empirical evidence suggesting that stuttering occurs more frequently on initial consonants than on initial vowels. Clinical experience suggested that consonants such as /p/, /t/, /k/, /d/ appear to be especially difficult for stutterers to negotiate. Empirical research tends to support these clinical observations. Although Wingate (1976)

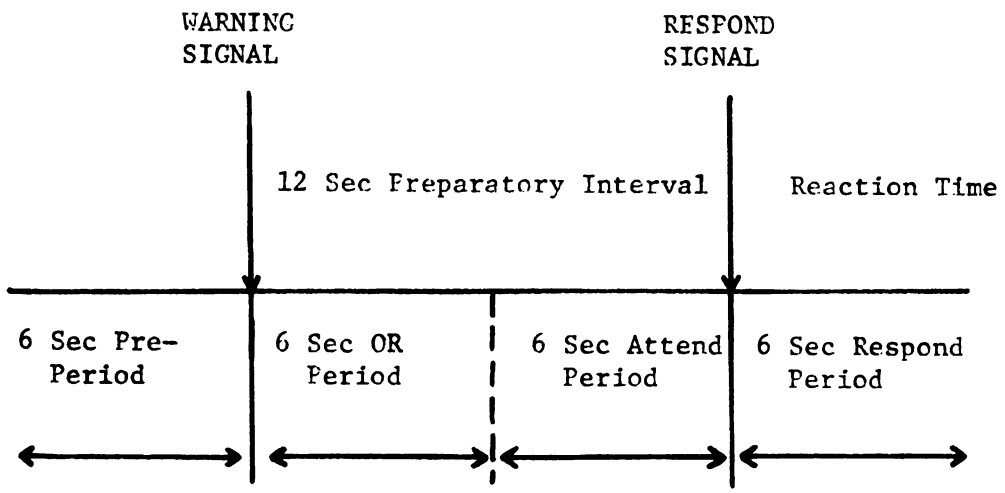

Figure 1. Reaction time task on a single trial and illustration of the four periods used in data analysis.

has argued for stressed vowels as the immediate elicitor of stuttering blocks, it is nevertheless the case that it is the consonant that the stutterer sounds during the block, not the vowel. Bloodstein (1975) reports that initial consonants are more difficult for stutterers than are initial vowels. According to Brown (1938), consonants are more important for speech intelligibility and meaning, involve a greater measure of anticipatory tension, and are more easily perceived as difficult to utter.

Ten sentences were constructed initially and exposed to review. Several of these sentences were found to contain words that in and of themselves were too difficult to pronounce. On the basis of this review, the sentences were either modified, retained, or replaced with new sentences. The final set of 10 sentences (one for each trial) was pretested on 10 fluent speakers for readability and duration of reading time. The 10 distraction sentences and their mean length of utterance were:

1. Measles is a particularly frequent disease among children ($\underline{M}$ = 2.93 sec).
2. Interpersonal conflicts are often dangerous for mental health ($\underline{M}$ = 3.0 sec).
3. The novel "Point Counter Point" was written by Aldous Huxley ($\underline{M}$ = 3.0 sec).
4. Waylan Jennings and Dolly Parton are popular country singers ($\underline{M}$ = 2.81 sec).
5. St. Peter's Cathedral is a masterpiece of renaissance architecture ($\underline{M}$ = 3.37 sec).
6. The battle for Yorktown was crucial in the American Revolution ($\underline{M}$ = 3.50 sec).
7. Systematic desensitization is effective in the treatment of phobias ($\underline{M}$ = 3.5 sec).
8. The movie "One flew over the cuckoo's nest" won several Oscars last year ($\underline{M}$ = 3.43 sec).

9. General Custer was defeated by the Sioux Indians at Little Big Horn ($\underline{M}$ = 3.12 sec).

10. Parkinson's disease is a serious neurological disorder ($\underline{M}$ = 2.62 sec).

None of the fluent speakers had difficulty pronouncing any of the words. Thus, it was concluded that even dysfluent speakers should be able to complete the sentence during the 12-sec PI even if a stuttering block should occur.

Scoring of Physiological Variables

HR, respiration, and SC were scored for each baseline period and for each trial. As indicated in Figure 1, each trial was divided into four Periods: a Preperiod, consisting of the 6 sec prior on the onset of the warning signal; an OR Period, consisting of the 6 sec immediately after onset of the warning signal; an Attend Period, consisting of 6 sec immediately prior to the onset of the reaction signal; and a Respond Period, consisting of the 6 sec immediately following onset of the reaction signal.

Two measures of HR were derived: the reciprocal of HR, heart period (HP) and heart period variability (HPV). For each complete R-R interval in each second of each period, the distance between cardiac R waves was measured in mm. The distance in mm was then divided by the polygraph paper speed (25 mm/sec) with the resulting value indicating HP for that second. HP and HPV scores were obtained for each trial in all four periods in both conditions and submitted to statistical analyses. HP was analyzed for seconds and for Periods. For the second-by-second analysis, a HP difference score

was calculated for each second during the OR Period, the Attend Period, and the Respond Period. These three Periods comprised the 18 sec of data collection on each trial following the onset of the warning signal. The HP difference score for seconds consisted of the difference between the HP of the last second of the Preperiod and the HP of each of the following 18 sec of a trial. The HP difference scores for Periods consisted of the difference between the mean HP of the Preperiod and the mean HP for each of the OR, Attend, and Respond Periods.

Respiration frequency was scored by counting the number of inspirations (initiations of respiratory cycle) in each of the Periods for each trial. Respiration amplitude was scored by measuring in mm the extent of polygraph pen deflection for each respiratory cycle initiated in each Period. Mean respiration amplitude was computed and used in the statistical analysis.

SC was measured in mm of pen deflection for each SCR that began during the Preperiod and Attend Period, and for each SCR that began within .6-6 sec following the warning and reaction signals. Any positive deflection of the pen which exceeded 1 micromho was considered an SCR. Tonic SC (SCL) was scored for each baseline Period and each trial Period. SCL was scored as the average of two readings of SCL, one at the beginning and one at the end of each Period noted above. Finally, electrodermal frequency (EF) was scored for each Period as the number of SCRs that began during that Period.

Thus, the following dependent variables were scored and submitted to statistical analyses: HP, HPV, respiration rate, respiration amplitude, SC, SCL, and EF. Each dependent measure was analyzed with repeated measures analyses of variance. Interactions were tested with simple or simple-simple effects tests. Relationships among dependent variables were examined with correlational statistics.

CHAPTER IV

RESULTS

Separate analyses of variance were computed for RT, HP, HPV, Respiration Frequency, Respiration Amplitude, log SCL, SCR, EDF, Base level HP, Base Level HPV, and Base level log SCL. Appendix C contains analysis of variance summary tables for each of these analyses. When significant interactions occurred, simple effects analyses of variance were performed on the conditions for the interacting variables. Tukey multiple comparisons were performed to test for significant differences between means (at $p < .05$). Pearson product moment correlations were computed for the relationship between scores on the Willoughby measure of anxiety and log SCL, between HP and RT, and between HPV and RT. Group differences on questionnaire data were analyzed with t tests.

Reaction Time Performance

Mean RT did not differ significantly between Groups, Orders, or Conditions. Although RT did improve as a function of trials (F (9,144) = 5.08, $p < .0005$), there were significant interactions of Group x Order x Trials (F (9,144) = 2.08, $p < .035$) and Order x Conditions x Trials (F (9,144) = 2.73, $p < .006$). A test for simple effects indicated that when subjects were divided into stuttering and nonstuttering groups, the Group x Order x Trials interaction

was a result of RT differences between trials for stutterers in Order 1 (F (9,144) = 3.29, $p < .01$) and for nonstutterers in Order 2 (F (9,144) = 2.60, $p < .01$). As illustrated in Figure 2, the marked reduction in RT from trial 1 to trial 2 suggests that either a practice effect or a warm-up effect was responsible for this interaction.

Figure 3 illustrates the interaction of Order x Condition x Trials. Simple effects analyses showed that this interaction resulted from differential decreases in RT across trials in the SPK condition of the SPK-NSPK order (F (9,288) = 4.13, $p < .01$) and in the NSPK condition of the NSPK-SPK order (F (9,288) = 2.44, $p < .01$). In addition, RT was significantly greater in the SPK than in the NSPK condition in (a) the SPK-NSPK order on trial 1 (F (1,160) = 14.69, $p < .005$) and (b) in the NSPK-SPK order on trial 9 (F (1,160) = 11.59, $p < .005$).

Finally, as illustrated in Figure 4, there was a significant interaction of Order x Condition (F (1,16) = 5.08, $p < .039$). A test for simple effects indicated that the interaction was a function of a significant difference in mean RT for Order 2 (F (1,16) = 7.44, $p < .025$) indicating greater RT in the SPK condition compared to the NSPK condition.

The Order x Condition and Order x Condition x Trials interactions may have been caused by the performance of one of the stutterers. This subject had extraordinarily slow RTs in each condition, but especially during the Speaking condition, which in his case was the second condition to occur in the experimental setting. His mean RT latency for the Speaking condition was 758 msec. Moreover, his

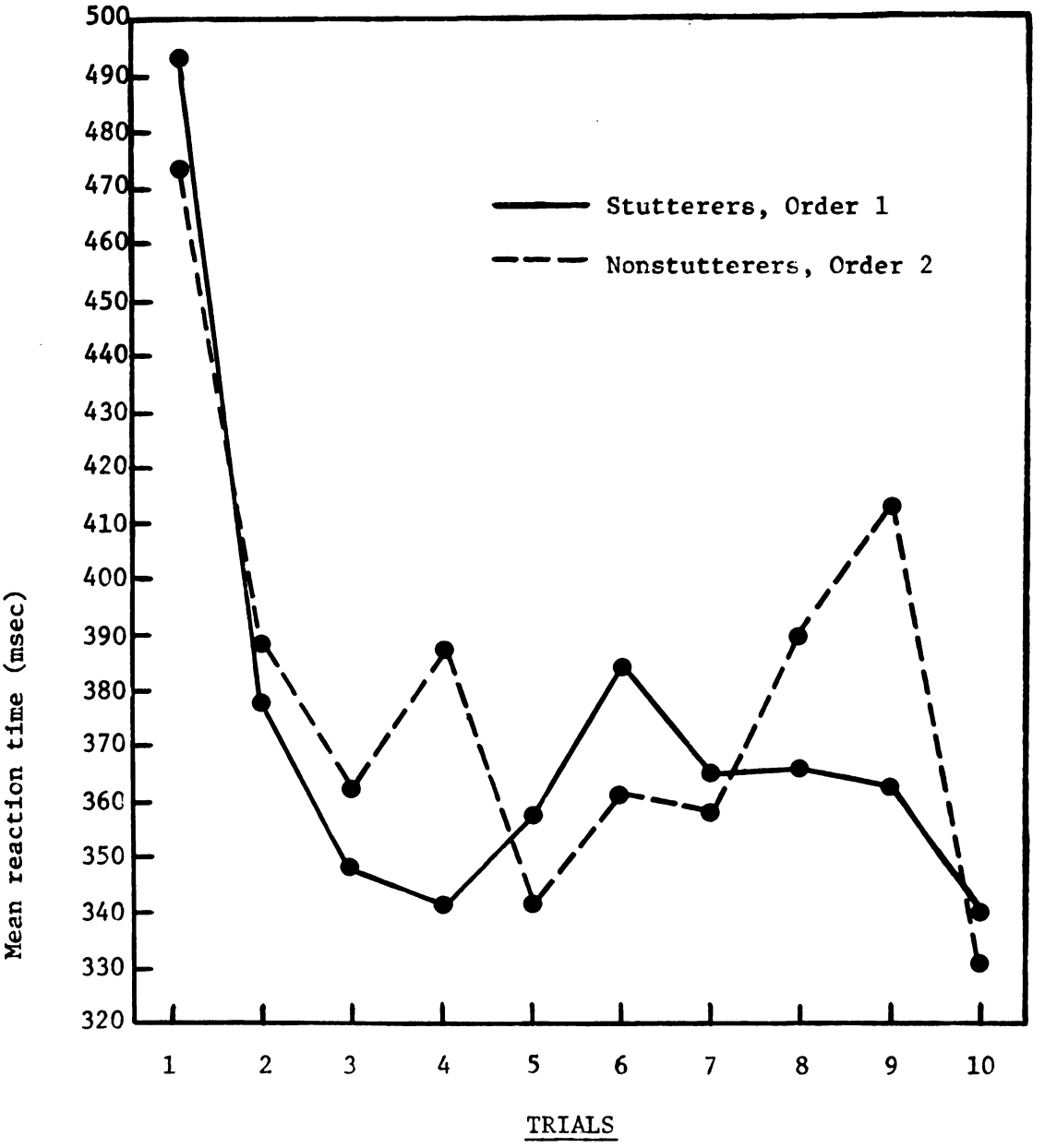

Figure 2. Mean reaction time per trial as a function of group and order.

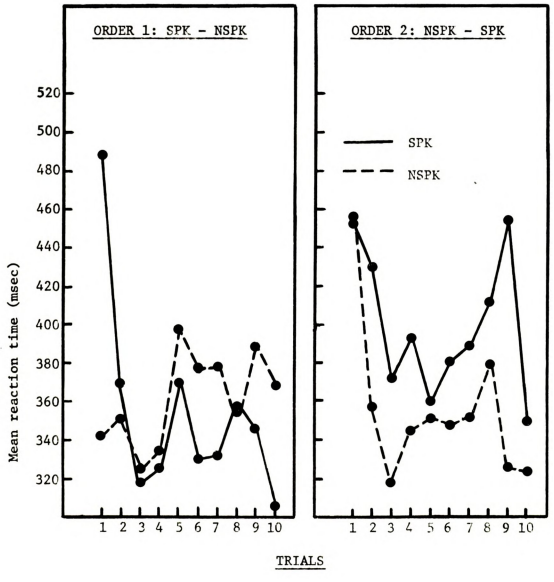

Figure 3. Mean reaction time per trial as a function of order and condition.

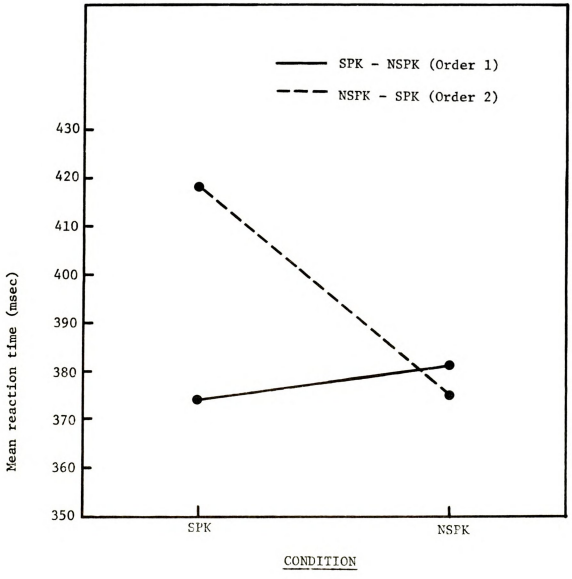

Figure 4. Mean reaction time as a function of the interaction of order and condition.

RT latencies of 1400 msec on trial 6 and 990 msec on trial 9 are sufficiently beyond the normal range expected for this task to have caused the elevated RT depicted in Figure 4 for the SPK condition in the NSPK-SPK order. Thus it seems reasonable to expect that if this subject's data were excluded from the analyses the Order x Condition interaction would not have appeared.

Summary of RT Results

Two of the three hypothesized effects were not supported: stutterers' RT did not differ as a function of not speaking during the PI; stutterers and nonstutterers did not differ in RT latency as a function of speaking and not speaking during the PI. On the other hand, the hypothesis that nonstutterers' RT would not differ between the PI conditions was supported. The interaction obtained and depicted in Figures 3 and 4 appears to have been spuriously influenced by the RT data from one subject in the stuttering group. Thus it seems reasonably clear that stutterers and nonstutterers did not show meaningful differences in RT performance either between groups or within groups, with the exception of the one stutterer noted above.

Heart Period

Data for mean second x second HP changes from the last Pre-period second were analyzed in separate analyses of variance for stutterers and nonstutterers since the combined analysis would have exceeded the available core memory capacity of the CDC 6500 computer.

However, a combined analysis of variance was performed on the mean HP changes from the Preperiod mean.

Seconds Analysis for Stutterers

The analysis of variance of second x second HP for stutterers indicated significant main effects for Seconds (F (17,136) = 2.50, $p < .002$) and Conditions (F (1,8) = 20.73, $p < .002$). Figure 5 illustrates changes in mean HP over the 18 sec for all trials combined. As indicated in Figure 5, HR accelerated during the first few seconds of the OR Period, steadily decelerated through the Attend Period, and accelerated following the Respond signal. However, the Seconds main effect was involved in significant interactions of Conditions x Seconds (F (17,136) = 2.50, $p < .002$) and Trials x Seconds (F (153,1224) = 1.34, $p < .005$). The Conditions x Seconds interaction, illustrated in Figure 6, indicates that stutterers had higher overall HR during the SPK condition than during the NSPK condition. Moreover, during the Attend Period stutterers showed a biphasic HR response during the SPK condition but showed a rather flat decelerative HR pattern during the NSPK condition. A test for simple effects indicated significant differences between conditions in mean HP for seconds 3 (F (1,144) = 32.03, $p < .005$), 6 (F (1,144) = 7.79, $p < .005$), 9 (F (1,144) = 31.08, $p < .005$), and 18 (F (1,144) = 10.24, $p < .0005$).

Figure 7 indicates that the temporal conditioning influence typical of HP during a fixed PI RT task was only partially in evidence from trial 1 to trial 10. Although HR deceleration did move

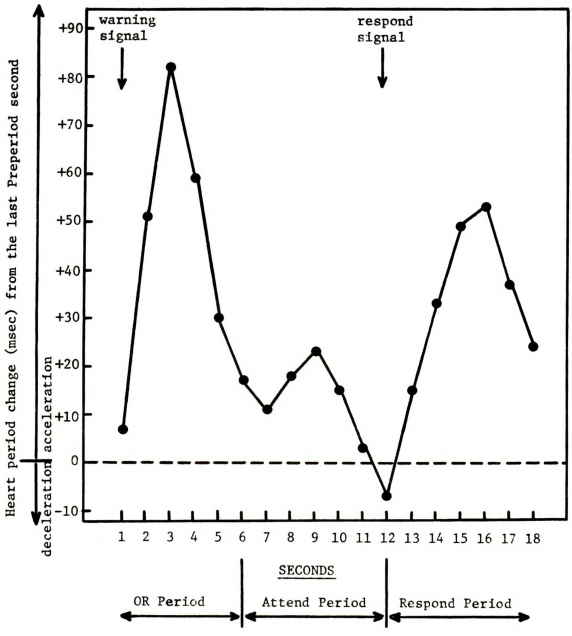

Figure 5. Second by second changes in heart period for stutterers as a function of seconds.

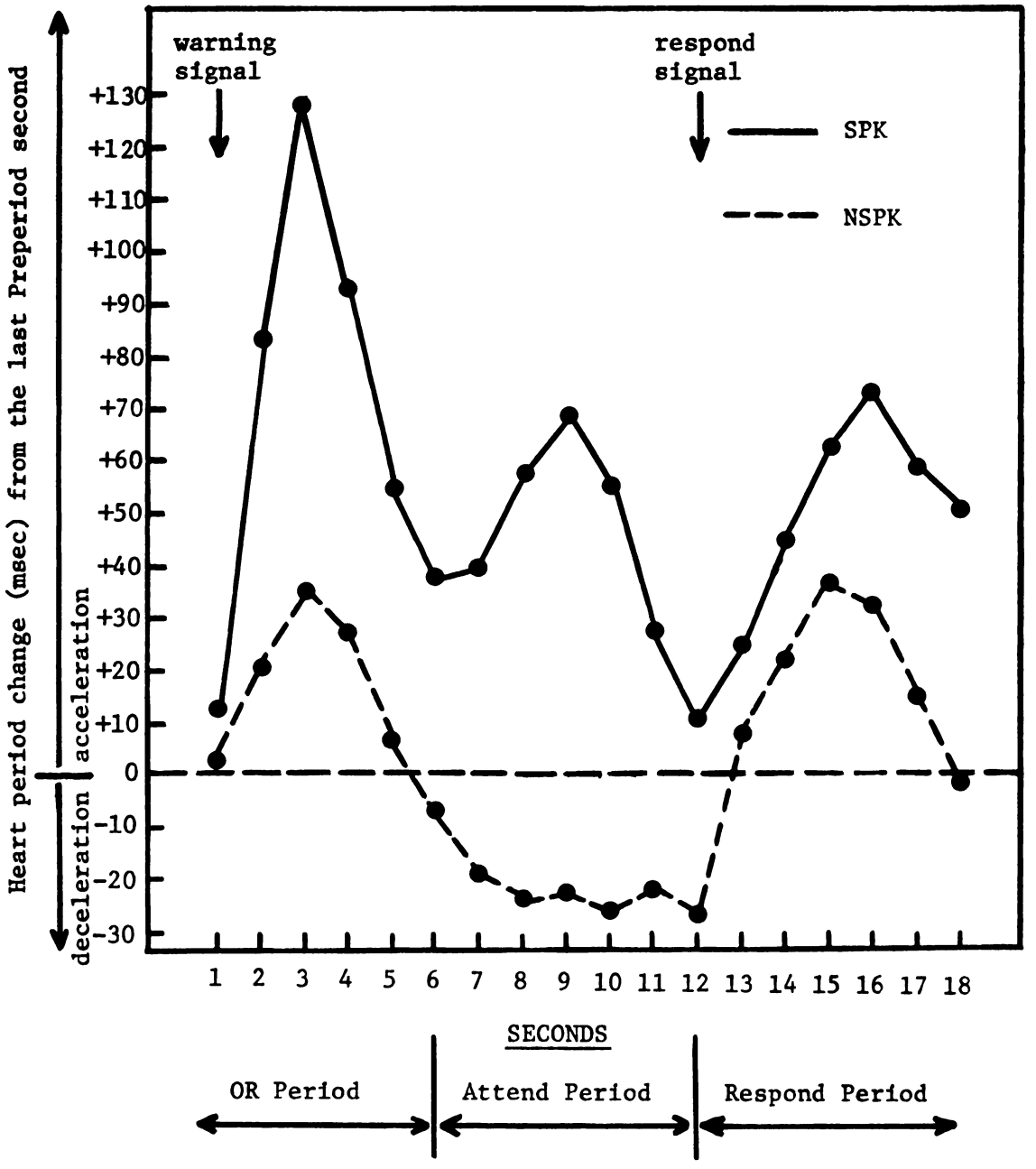

Figure 6. Second by second changes in heart period for stutterers in the speaking and nonspeaking conditions.

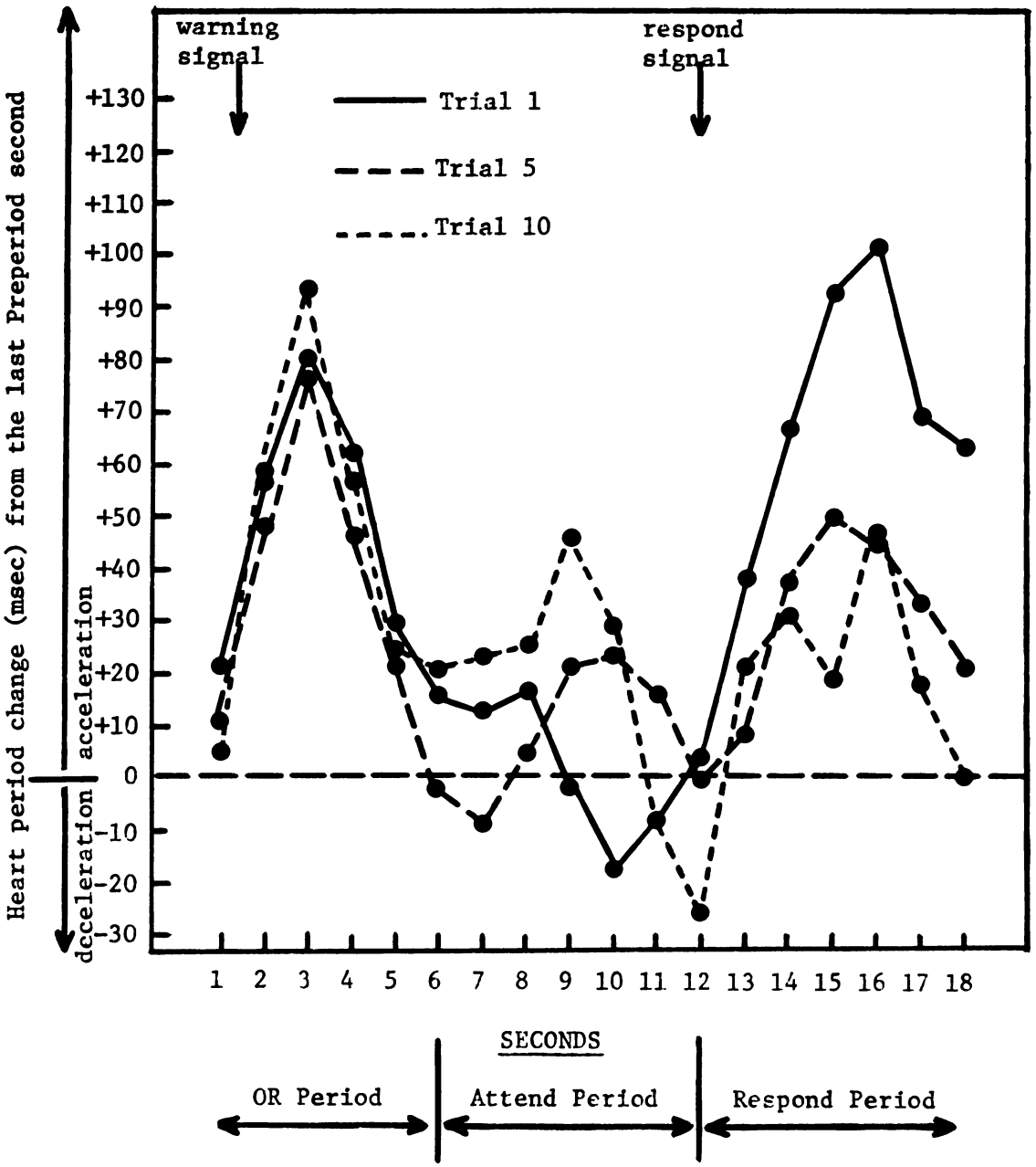

Figure 7. Second by second changes in heart period for stutterers on trials 1, 5, and 10.

temporally closer to the respond signal by trial 10, the HR pattern also showed an atypical acceleration during the early seconds of the Attend Period. Inspection of the curves in Figure 6 suggests that HR patterns in the SPK condition account for the atypical HR pattern on trial 10 (Figure 7).

Finally, the main effect for Conditions was a function of the significant interaction of Order x Conditions (F (1,8) = 20.73, $p < .002$). Figure 8 illustrates this interaction. A test for simple effects indicated that the Order x Condition interaction resulted from a greater over-all change in HP in the SPK condition than in the NSPK condition in the SPK-NSPK order (F (1,8) = 76.94, $p < .025$), but there were no differences between conditions in the NSPK-SPK order.

Seconds Analysis for Nonstutterers

The analysis of variance of second x second mean HP for nonstutterers indicated significant main effects for Seconds (F (17,136) = 15.79, $p < .0005$) and Conditions (F (1,8) = 8.10, $p < .02$). Figure 9 illustrates changes in mean HP over the 18 sec for all trials combined. The major difference between the curve depicted in Figure 9 and its counterpart for stutterers in Figure 5 occurs approximately between the sixth and ninth seconds of the PI. Whereas nonstutterers are beginning to accelerate briefly at the sixth second just prior to a long gradual deceleration beginning at the seventh second and continuing to the Respond signal, stutterers do not begin their deceleration to the Respond signal until the

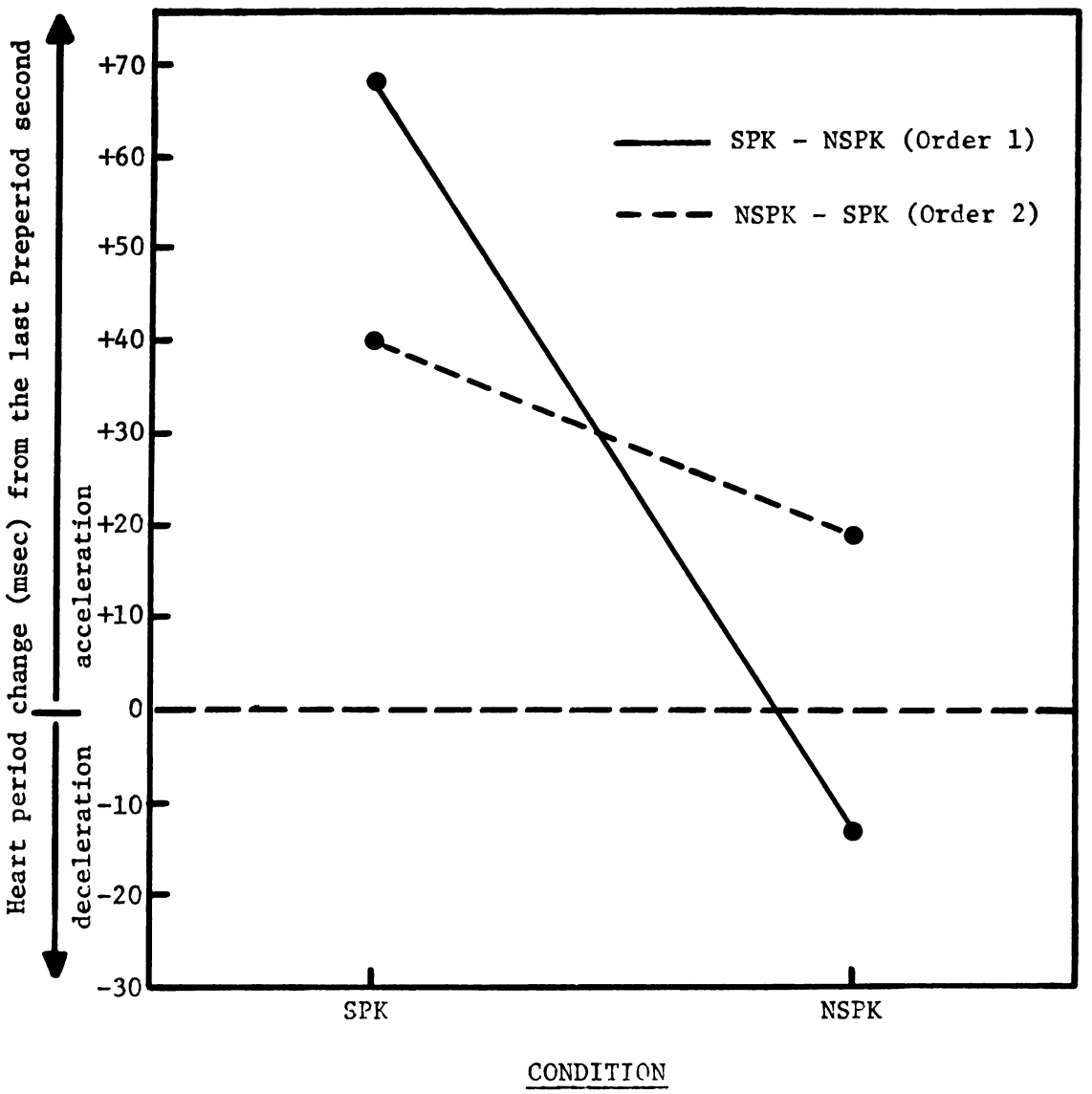

Figure 8. Stutterers' mean heart period for seconds by seconds analyses as a function of conditions and orders.

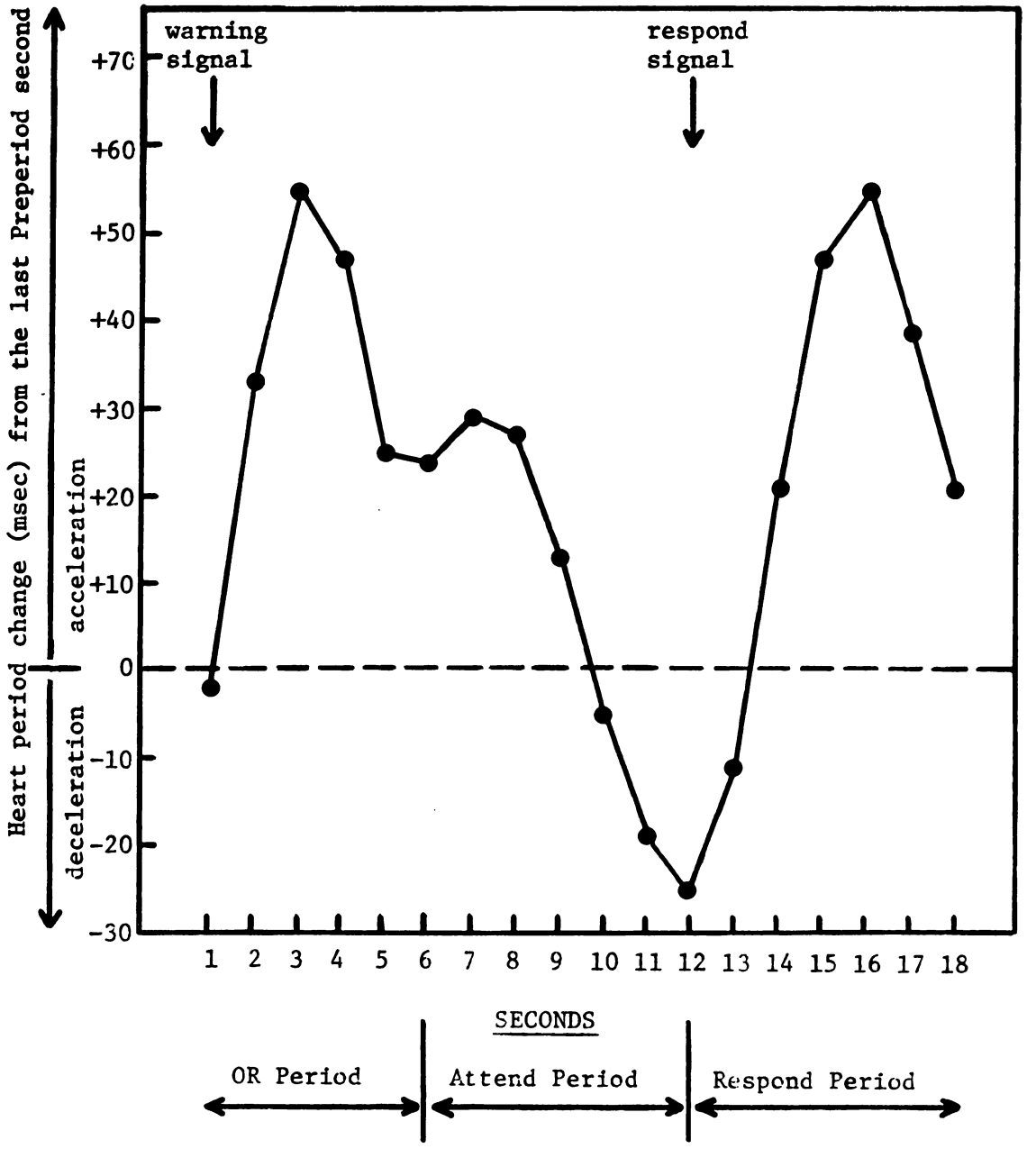

Figure 9. Second by second changes in heart period for nonstutterers as a function of seconds.

ninth second. Comparison of Figure 10 with Figure 6 suggests that this difference can be accounted for by the HR pattern for the SPK condition.

As was the case for stutterers, the Seconds main effect for nonstutterers was qualified by significant interactions of Conditions x Seconds (F (17,136) = 7.18, $p < .0005$) and Trials x Seconds (F (153,1224) = 1.49, $p < .0005$). The Conditions x Seconds interaction illustrated in Figure 10 indicates that nonstutterers had smaller overall HR changes during the NSPK condition compared with the SPK condition. A test for simple effects indicated significant differences in mean HP for seconds 3 (F (1,144) = 10.79, $p < .005$), 6 (F (1,144) = 14.93, $p < .005$), 7 (F (1,144) = 22.86, $p < .005$), and 18 (F (1,144) = 8.12, $p < .005$).

Finally, in contrast to the results for stutterers, the significant Trials x Seconds interaction illustrated in Figure 11 reflects the typical temporal conditioning effect found in the fixed PI RT task. Thus, on trial 10, nonstutterers showed a gradual deceleration through the Attend Period, more marked HR deceleration in close proximity to the Respond signal (see Figure 7 for comparative stutterer responses).

Periods Analyses

The analysis of variance of mean HP difference scores for the OR, Attend, and Respond Periods indicated significant main effects for Conditions (F (1,16) = 29.40, $p < .0005$), Trials (F (9,144) = 3.00, $p < .003$), and Periods (F 2,32) = 26.43, $p < .0005$). These

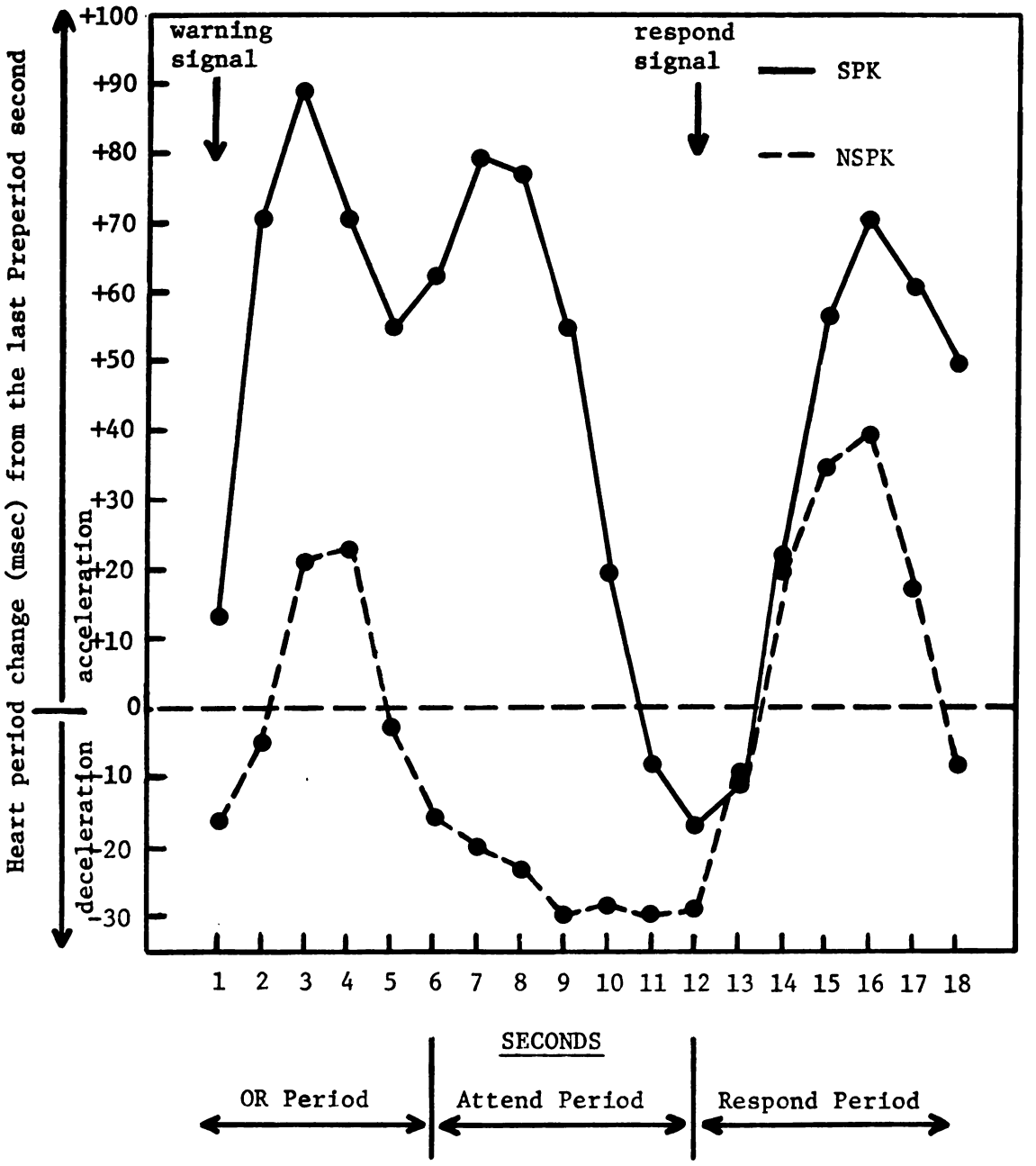

Figure 10. Second by second changes in heart period for nonstutterers in the speaking and nonspeaking conditions.

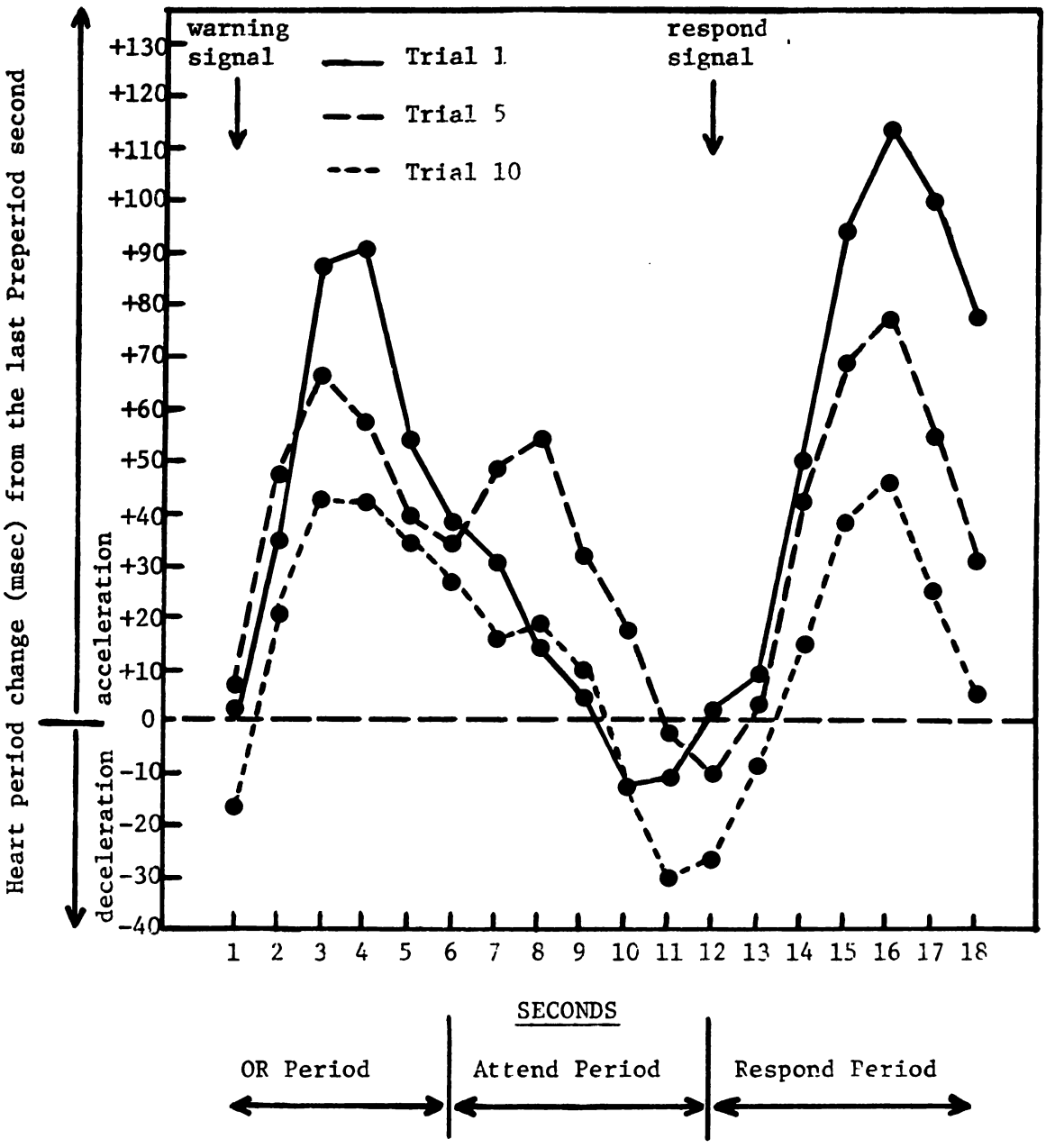

Figure 11. Second by second changes in heart period for nonstutterers on trials 1, 5, and 10.

main effects are best understood within the framework of the significant interactions that occurred.

Simple effects tests of the significant Conditions x Periods interaction (F (2.32) = 6.22, $p < .005$) indicated that there were significant differences between conditions for the OR Period (F (1,48) = 25.80, $p < .01$), the Attend Period (F (1,48) = 33.43, $p < .01$), and the Respond Period (F (1,48) = 35.93, $p < .01$). These differences are illustrated in Figure 12. Tukey comparisons indicated that the significant differences in mean HP difference scores within the SPK condition were for the OR Period-Attend Period comparisons. On the other hand, for the NSPK condition there were significant differences in mean HP difference scores for the OR Period-Attend Period comparison and the Attend Period-Respond Period comparison.

Simple effects analysis of variance for the Trials x Periods interaction (F (18,288) = 3.48, $p < .0005$) revealed significant differences in mean HP difference scores for the Attend Period (F (9,432) = 2.54, $p < .01$) and the Respond Period (F (9,432) = 5.61, $p < .01$) at trials 1,2,3,4,6,8, and 10. The Trials x Periods interaction along with significant interaction of Group x Order x Trials (F (9,144) = 2.01, $p < .04$) are best interpreted in light of the second x second HP changes discussed above. For example, the HP change score for the Attend Period during the SPK condition (Figure 12) is most likely the result of the Attend Period HR acceleration noted in the second x second analysis for stutterers in the SPK condition.

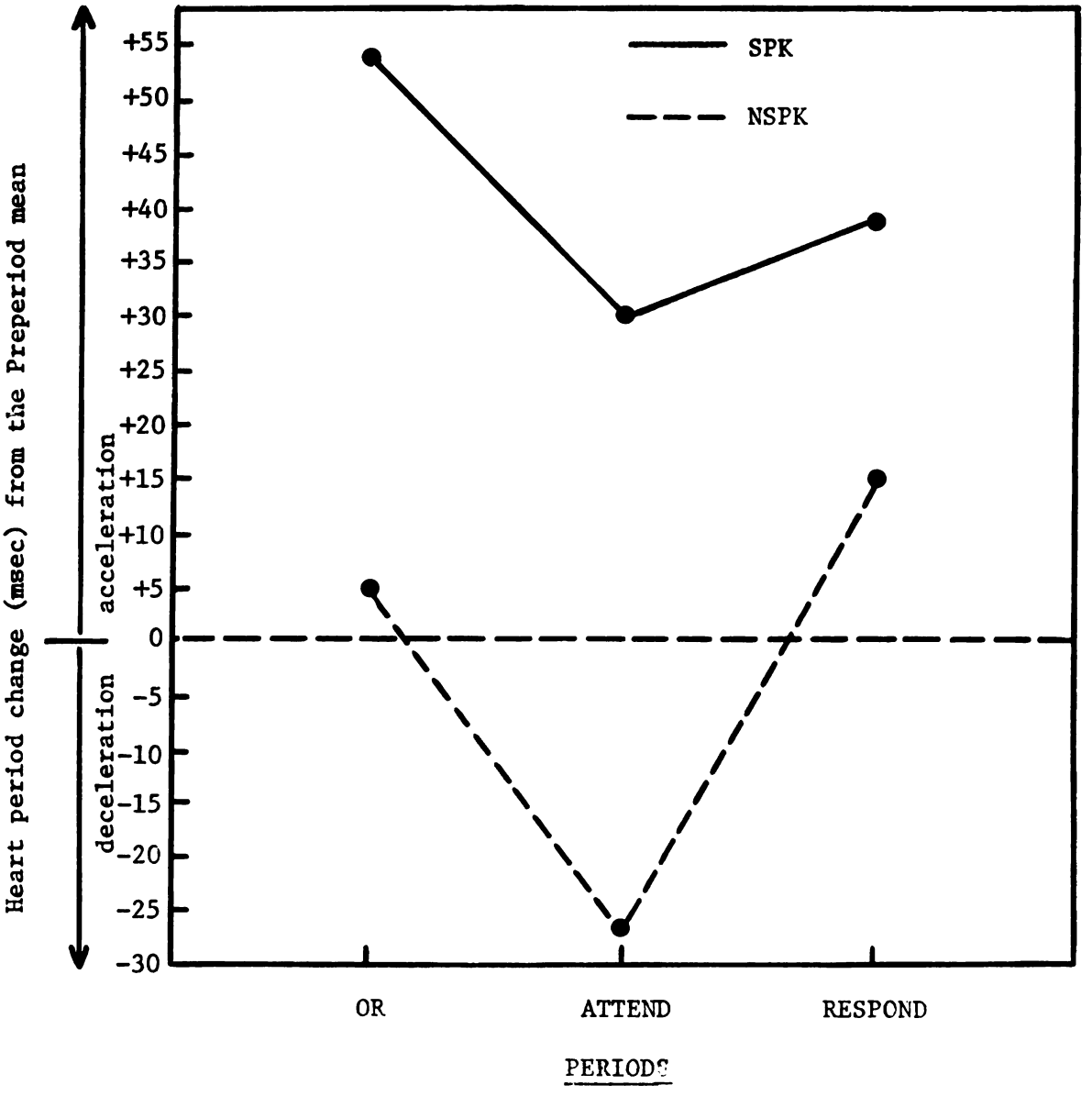

Figure 12. Mean heart period difference scores for periods as a function of conditions.

Heart Period Variability

The analysis of variance for mean HPV indicated a significant main effect for Conditions (F (1,16) = 7.97, $p < .012$). Mean HPV was greater during the SPK condition (M = -1399.18) than during the NSPK Condition (M = 237.00). In addition to the Conditions main effect there also were significant interactions of Groups x Trials (F (9,144) = 2.70, $p < .006$), Order x Trials (F (9,144) = 1.98, $p < .045$), and Groups x Orders x Trials x Periods (F (18,288) = 1.80, $p < .025$).

Figure 13 illustrates the Groups x Trials interaction. Simple effects tests showed that this interaction could be accounted for by the marked trial 3 variability (F (1,160) = 8.31, $p < .005$) for stutterers.

Figure 14 illustrates the Order x Trial interaction. Simple effects tests indicated that this interaction was a result of greater trial to trial variability within the SPK-NSPK order (F (9,144) = 2.77, $p < .025$).

Summary of Heart Period Results

The results provided support for one of the hypotheses and partial support for another. As hypothesized, stutterers and non-stutterers did not differ in HR during the nonspeech condition. In fact, the HR patterns for each group were characteristic of those typically associated with the RT task (Krupski, 1975; Porges, 1972).

Although hypotheses concerning stutterers' and nonstutterers' HR patterns during the SPK and NSPK conditions were not supported,

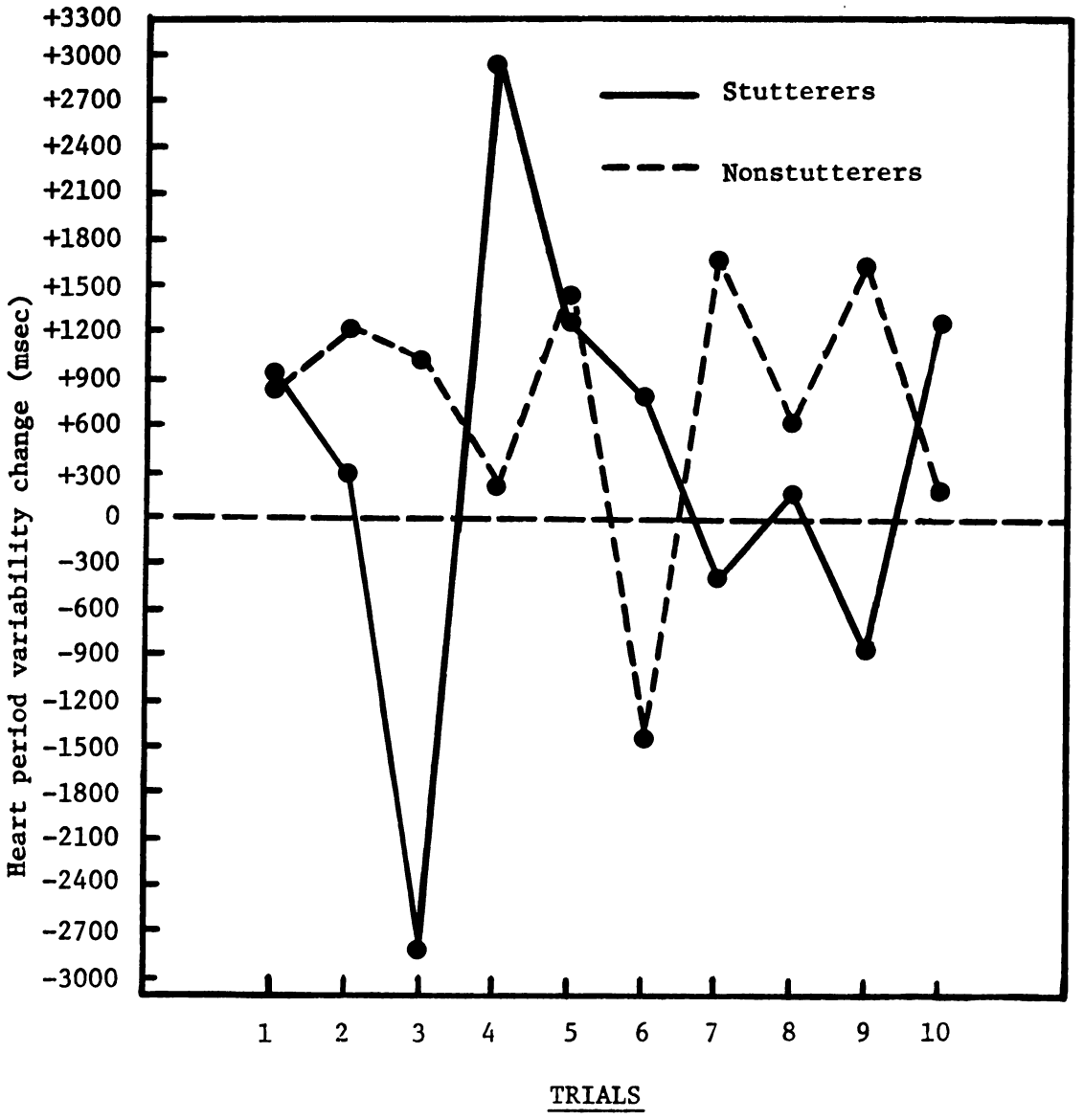

Figure 13. Mean heart period variability change from the Preperiod mean for each trial as a function of groups.

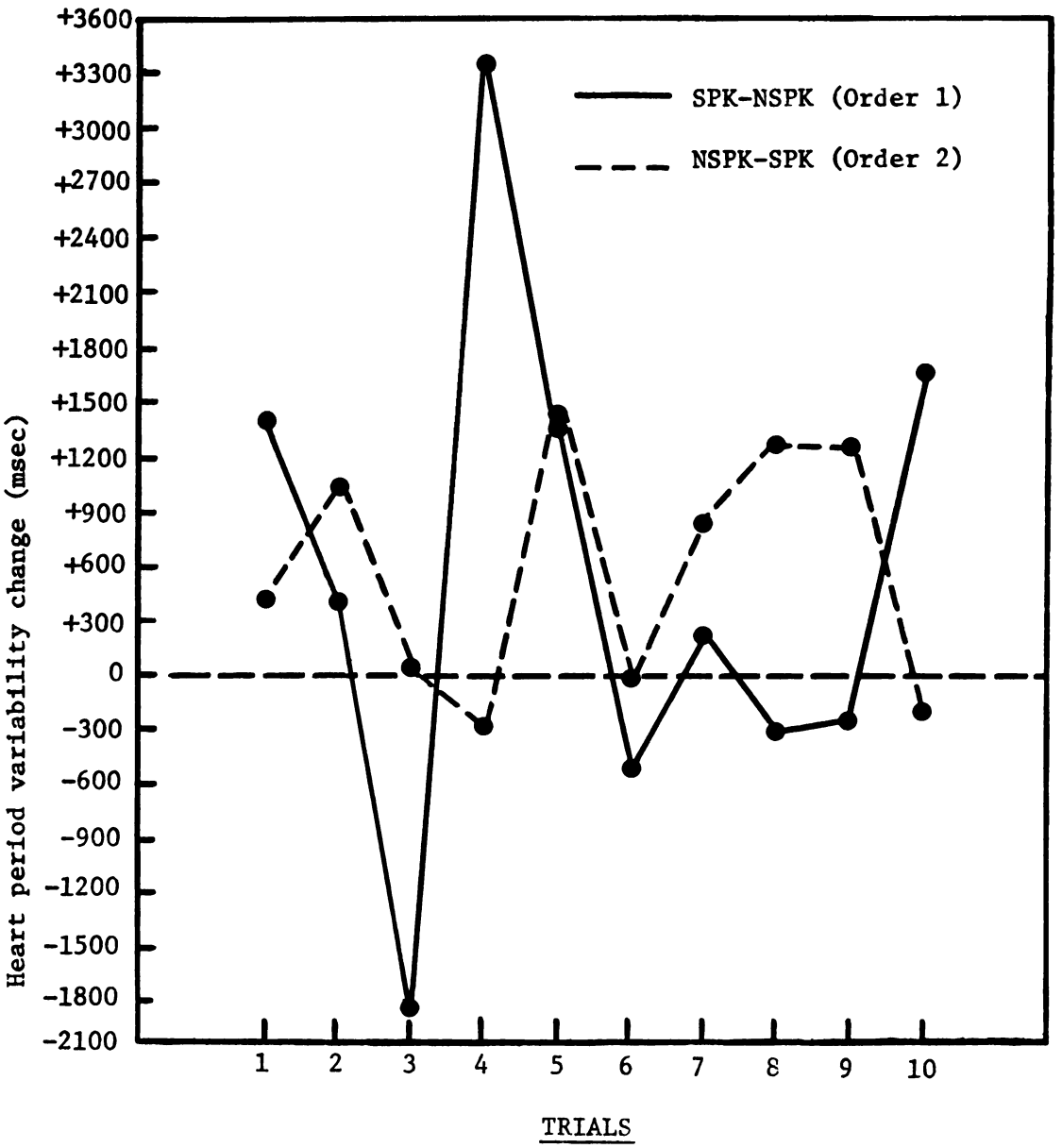

Figure 14. Mean heart period variability change from the Preperiod mean for each trial as a function of orders.

response typographies during the SPK condition clearly were different than those during the NSPK condition for both groups. Comparison of the response typographies illustrated in Figures 6 and 10 indicates that stutterers had a 2-sec delay in the formation of the decelerative HR response typically correlated with attention (Lacey, 1959; Luria, 1973; Obrist et al., 1974). Stutterers also failed to show the anticipatory HR acceleration to the respond signal noticeable in the HR pattern for nonstutterers. Moreover, stutterers apparently failed to benefit from the temporal conditioning component of the fixed PI over trials (Figures 7 and 11) compared to nonstutterers. These differences provide indirect support for the hypothesis that attentional disruption is a component of stuttering.

Respiration Amplitude

The analysis of variance for mean differences in respiration amplitude (millimeters) showed a significant main effect for Conditions (F (1,16) = 18.49, $p < .001$). There was a greater change in respiration amplitude during the SPK condition (M = -3.45, SD = 10.17) than during the NSPK condition (M = -.18, SD = 7.63). In addition, there was a significant main effect for Period (F (2,32) = 12.52, $p < .0005$). Mean difference scores and standard deviations for each of the Periods were as follows: OR Period (M = -3.88, SD = 9.60), Attend Period (M = .37, SD = 8.25), and Respond Period (M = 1.95, SD = 9.04).

The main effect for Conditions is qualified by a Groups x Conditions interaction (F (2,32) = 12.52, p < .005). A simple effects test showed that this interaction could be accounted for by a significantly greater mean respiration amplitude change for stutterers in the SPK condition (F (1,32) = 5.55, p < .025).

The main effect for Periods is qualified by a significant interaction of Conditions x Periods (F (2,32) = 10.41, p < .0005). A test for simple effects showed that this interaction could be accounted for by a significant difference in mean respiration amplitude change in the SPK condition at the OR Period (F (1,48) = 37.97, p < .01). Tukey tests for multiple comparisons indicated that the mean respiration amplitude change was significantly greater during the OR Period in the SPK condition than during the NSPK condition.

Finally, there was a significant Order x Conditions x Trials interaction (F (9,144) = 2.24, p < .023). Means for this interaction are summarized in Table 2.

Respiration Frequency

The analysis of variance for mean change in respiration frequency showed a significant main effect for Conditions (F (1,16) = 8.03, p < .012), indicating that the change in respiration frequency was significantly greater for the SPK condition (M = -1.98, SD = .91) than for the NSPK condition (M = .006, SD = .77). A significant main effect for Periods (F (2,32) = 3.94, p < .03) indicated differences in mean respiration frequency change among the OR Period

(M = -.05, SD = .94), the Attend Period (M = .015, SD = .74), and the Respond Period (M = .25, SD = .84).

Table 2

Mean Change in Respiration Amplitude (Millimeters) from the Preperiod Mean for the Order x Conditions x Trials Interaction

Trial	Order			
	SPK-NSPK		NSPK-SPK	
	Condition		Condition	
	SPK	NSPK	SPK	NSPK
1	-7.00	-0.61	-3.72	-1.22
2	.04	.23	-7.77	1.98
3	-1.74	2.51	-1.67	-1.77
4	-4.15	- .13	-2.44	.33
5	-4.70	-2.78	-7.17	-2.00
6	-8.03	- .01	3.83	1.23
7	.45	-2.60	-5.28	.03
8	-5.60	.02	-5.76	-1.93
9	-1.50	- .42	-4.16	.93
10	-3.25	.76	-6.77	1.67

The Condition main effect is qualified by a significant interaction of Group x Order x Condition (F (1,16) = 5.20, $p < .037$). This interaction, illustrated in Figure 15, is further qualified by a significant interaction of Group x Order x Condition x Trial (F (9,144) = 2,79, $p < .005$).

Summary of Respiration Results

Hypotheses concerning respiration changes were only partially supported. Contrary to the hypothesized effects, both stutterers

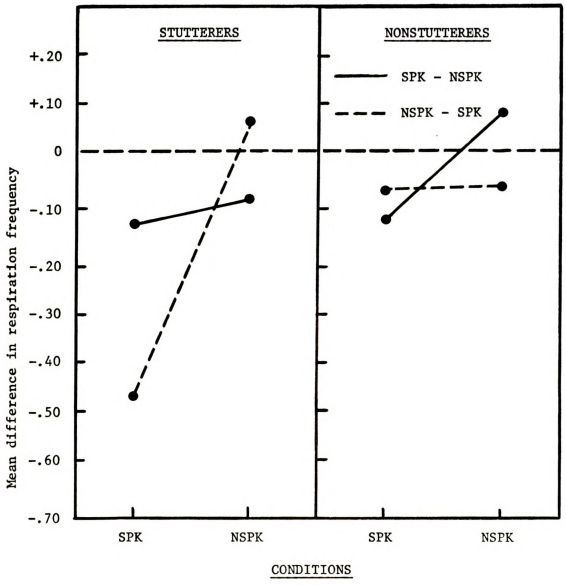

Figure 15. Changes in mean difference respiration frequency for stutterers and nonstutterers as a function of conditions and orders.

and nonstutterers showed greater change in respiration amplitude during the SPK condition compared to the NSPK condition. In addition, there were no differences between groups in respiration amplitude as a function of Periods; both groups showed less amplitude change during the OR Period compared to other Periods. Analysis of the Groups x Conditions interaction suggested that the difference between conditions could be accounted for by greater respiration amplitude changes in stutterers during the SPK condition. Thus, while both stutterers and nonstutterers had smaller amplitude respirations while speaking, the effect was most prominent among stutterers. The results for respiration frequency closely paralleled those for respiration amplitude.

Log Skin Conductance Level

Analysis of variance for mean log SCL yielded a significant main effect for Conditions (F (1,16) = 4.52, $p < .049$), indicating that SCL was higher during the SPK condition (M = 1.97 micromhos) than during the NSPK condition (M = 1.89 micromhos). Mean log SCL also differed among Periods (F (3,48) = 2.91, $p < .044$). Mean log SCL in micromhos for each Period were as follows: Preperiod (1.925), OR Period (1.936), Attend Period (1.942), and Respond Period (1.925). The significant main effect for Trials (F (9,144) = 2.76, $p < .005$), illustrated in Table 3, indicated that mean log SCL decreased over trials.

The significant Periods main effect was qualified by significant interactions of Conditions x Periods (F (3,48) = 3.21, $p < .031$) and

Trials x Periods (F (27,432) = 2.69, p < .0005). Figure 16 illustrates the Conditions x Periods interaction. Simple effects tests showed that these interactions resulted from significant periods differences for the SPK condition (F (3,96) = 4.42, p < .025). Tukey multiple comparisons indicated that the Conditions x Periods interaction could be accounted for by the following significant differences among Periods for the SPK condition: OR > Pre; Attend > Pre; and Attend > Post. No other comparisons were significantly different. Inspection of the means suggested that the Trials x Periods interaction was a result of elevated mean log SCL which occurred on trials 2, 3, and 4.

Table 3

Mean Log Skin Conductance Level for Each Trial

Trial	Mean	SD
1	1.929156	.35943614
2	1.983848	.28465643
3	1.979840	.30077803
4	1.968009	.33446227
5	1.935404	.40362843
6	1.898831	.50382540
7	1.915768	.41243511
8	1.905957	.40265952
9	1.902732	.43295010
10	1.906341	.43275978

The significant interaction of Group x Order x Period (F (3,48) = 2.88, p < .045) is illustrated in Figure 17. Simple effects tests

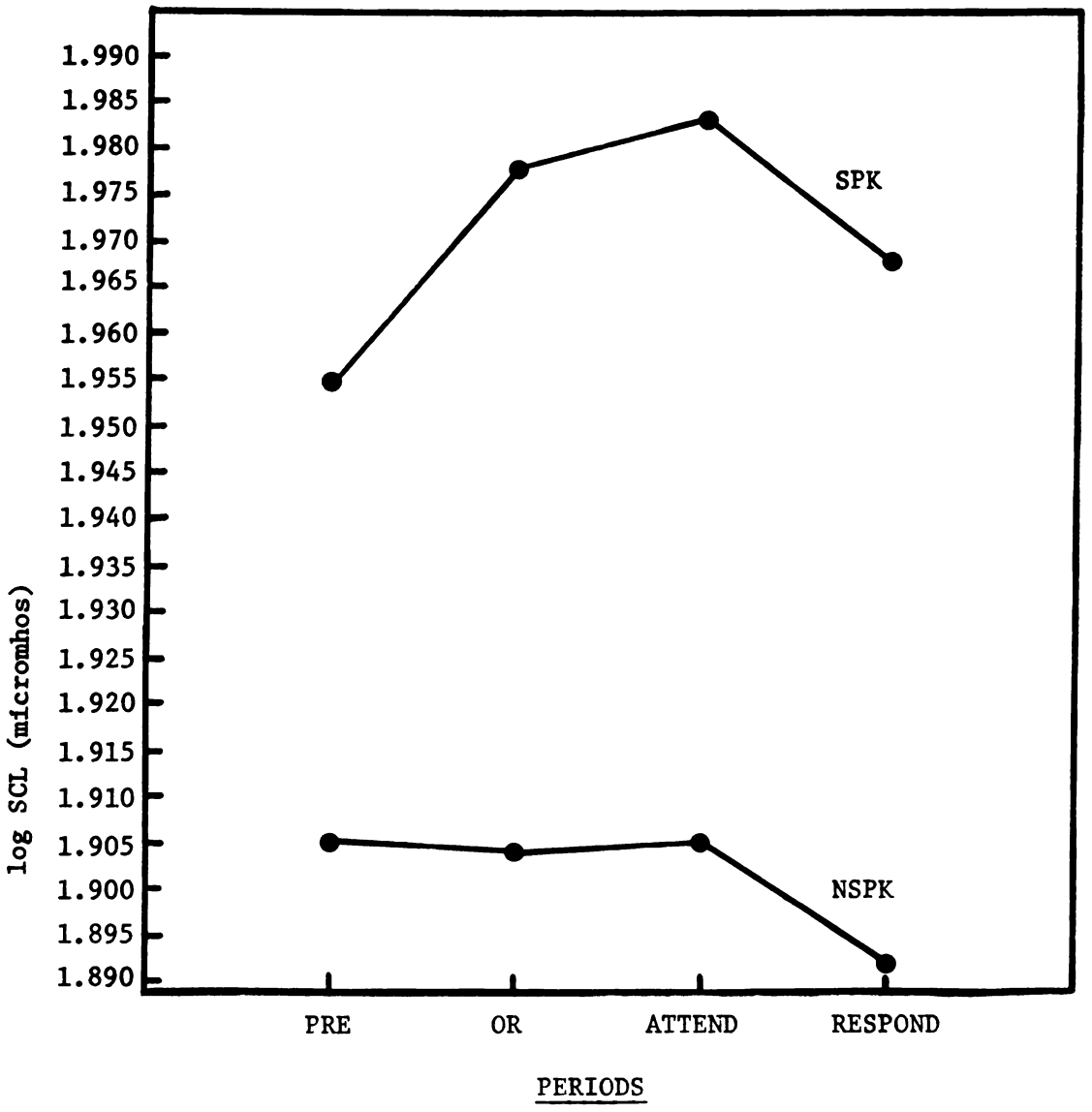

Figure 16. Mean changes in log SCL during the SPK and NSPK conditions for each period.

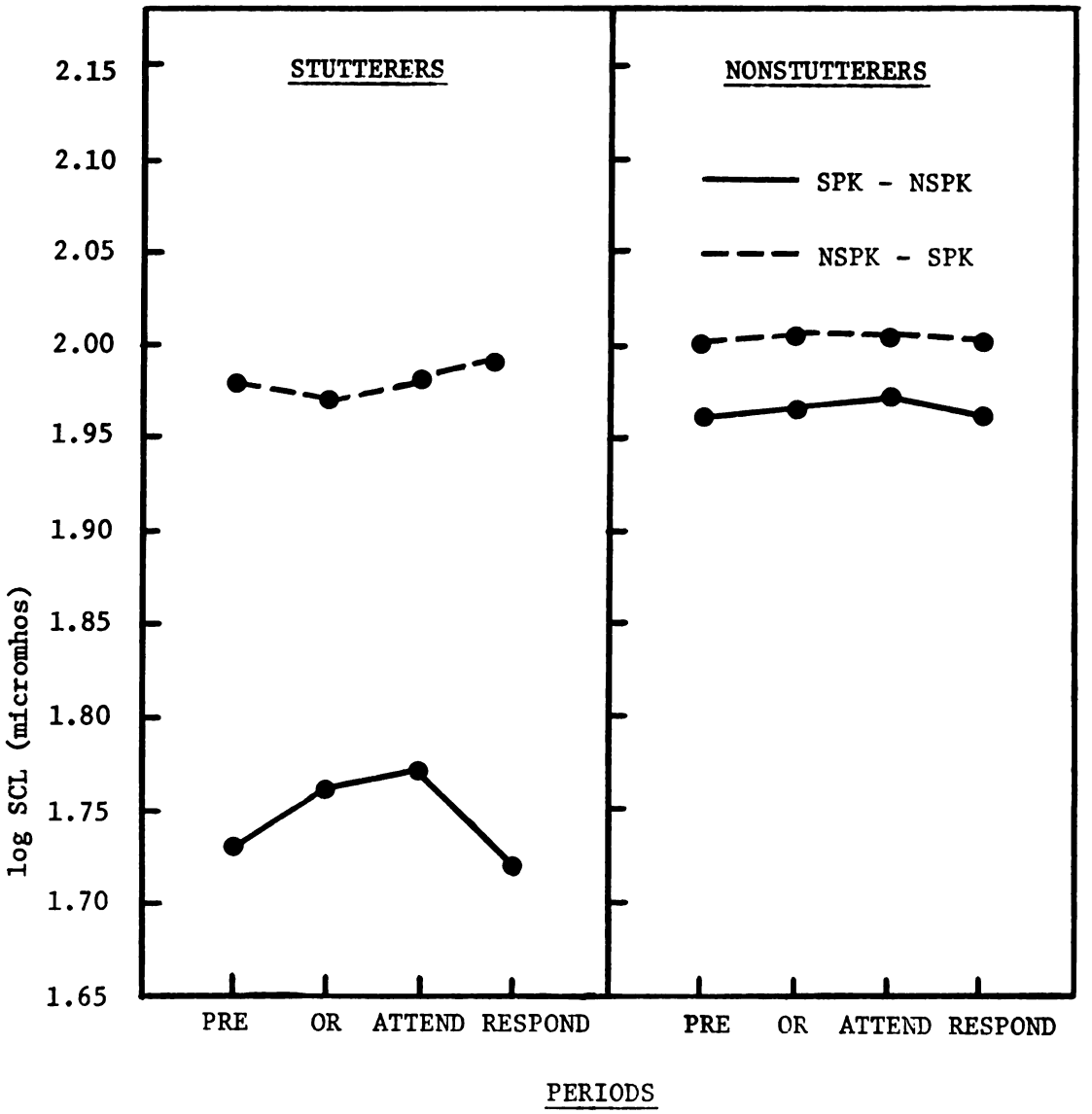

Figure 17. Mean log SCL for each period for stutterers and nonstutterers in SPK-NSPK and NSPK-SPK orders.

showed that this interaction resulted from mean log SCL differences for stutterers in the SPK-NSPK order (F (3,48) = 6.02, $p < .01$).

Skin Conductance Response

The analysis of variance for SCR indicated significant main effects for Trials (F (9,144) = 4.81, $p < .0005$) and Periods (F (3,48) = 21.33, $p < .0005$). Inspection of the means indicated that there was a decrease in mean SCR amplitude over trials. The significant Trials x Periods interaction (F (27,432) = 3.47, $p < .0005$) reflects a marked decrease in SCR during the OR Period for trials 1 and 2 (see Table 4).

Table 4

Mean SCR Amplitude in Micromhos for Each Period and Each Trial

Trials	Periods			
	Pre	OR	Attend	Respond
1	1.0175	17.3575	3.1275	9.5300
2	2.0025	10.6050	3.0325	7.5750
3	1.5250	8.5700	1.7125	7.6500
4	1.7075	6.1475	2.2000	7.0800
5	1.0250	8.3100	2.0550	7.4000
6	1.3675	7.9325	1.3000	7.6326
7	1.5250	9.0875	2.3500	8.1575
8	2.2375	7.8300	3.0125	8.8625
9	2.1575	8.0300	1.6300	9.1500
10	1.8500	7.4250	1.5500	7.6676

The Periods main effect is qualified by a significant interaction of Conditions x Periods (F (3,48) = 7.18, $p < .0005$). Simple effects tests showed that this interaction could be attributed to

differences in mean SCR for both the SPK and NSPK conditions at the Respond Period (F (1,64) = 12.50, $p < .01$). Tukey multiple comparisons (see Table 5 for the appropriate means) indicated that these differences were significant.

Table 5

Mean Skin Conductance Amplitude (Micromhos) for Each Period for the SPK and NSPK Conditions

Periods	Conditions	
	SPK	NSPK
Preperiod	1.893	1.390
OR Period	10.159	8.099
Attend Period	2.109	2.285
Respond Period	5.514	10.627

Electrodermal Frequency

Analysis of variance for mean EDF among the four Periods indicated that subjects had higher EDF during the SPK condition (M = 1.00) than during the NSPK condition (M = .77) (F (1,16) = 10.97, $p < .004$). In addition, there was a significant difference in EDF among Periods (F (3,48) = 38.97, $p < .0005$). Mean EDF for the four Periods were as follows: Preperiod (.58), OR Period (1.19), Attend Period (.67), and Respond Period (1.12).

The Periods main effect is qualified by a significant interaction of Conditions x Periods (F (3,48) = 8.32, $p < .0005$). Simple effects tests showed that there were significant differences between

conditions for the Preperiod (F (1,64) = 14.82, $p < .01$) and the OR Period (F (1,64) = 25.50, $p < .01$) and between Periods for the SPK condition (F (3,96) = 30.39, $p < .025$) and the NSPK condition (F (3,96) = 19.14, $p < .025$). Table 6 contains the appropriate means for the Condition x Period interaction. Tukey multiple comparisons showed that mean EDF was significantly greater for the Pre- and OR Periods in the SPK condition when compared to the NSPK condition. Tukey multiple comparisons also revealed significant differences among Periods within the SPK condition as follows: OR > Pre; OR > Attend; OR > Respond; Respond > Attend; Respond > Pre. Similar differences occurred between Periods within the NSPK condition (OR > Pre; OR > Attend; Respond > Attend; Respond > Pre; Attend > Pre).

Table 6

Mean Electrodermal Frequency for Each Period for the SPK and NSPK Conditions

Periods	Conditions	
	SPK	NSPK
Preperiod	.775	.390
OR Period	1.440	.935
Attend Period	.630	.710
Respond Period	1.175	1.060

Summary of Electrodermal Results

Only one of the three hypothesized effects was supported: stutterers had higher SCLs and lower magnitude SCRs during the SPK condition than during the NSPK condition. Contrary to expectations, nonstutterers showed the same pattern. When the Group x Order x Periods interaction was analyzed with simple effects tests, it was shown that stutterers had higher SCL during the OR and Attend Periods compared to the Preperiod and Respond Period in the SPK-NSPK order for the SPK condition. This suggests that stutterers were more responsive to the warning signal when they were required to perform the speaking task first as compared to when they were required to perform the speaking task last.

There was no evidence to support the expected differences among stutterers and nonstutterers as a function of speaking or not speaking. Moreover, none of the differences in SCR or EDF were attributable to factors within groups other than the one effect for SCR noted above.

Base Level Analyses for HP, HPV, and SCL

Analyses of variance for mean HP, mean HPV, and mean SCL during the four base level periods revealed no significant differences.

Questionnaires

Stutterers (M = 1.93, SD = .51) and nonstutterers (M = 1.68, SD = .37) did not differ significantly on the Iowa Scale of Attitudes Toward Stuttering (t (9) = 1.22, ns). Mean scores for both

groups fell within the range indicating average to moderate tolerance for stuttering.

Stutterers (M = 53.10, SD = 20.30) and nonstutterers (M = 24.3, SD = 8.30) did differ significantly on the Willoughby Questionnaire (t (9) = 4.128, $p < .05$). The mean score for stutterers falls toward the upper normative range for moderate anxiety and social sensitivity. In contrast, the mean score for nonstutterers falls toward the lower end of the normative range for mild anxiety and social sensitivity. Moreover, it should be noted that the variation was considerably greater among stutterers than among nonstutterers.

Stutterers' scores on the Stutterers' Self Rating of Reactions to Speech Situations placed them above the normative mean reported by Shumak (1942) for three of the four categories. Categories in which stutterers scored toward the negative self-rating end of the continuum were Reaction (M = 2.84, SD = .50), Stuttering (M = 2.91, SD = .47) and Frequency (M = 4.12, SD = .47). Stutterers scored on the average toward a positive self-rating on the Avoidance category (M = 2.72, SD = .54).

Correlational Analyses

Heart Period and Reaction Time

Pearson product moment correlations were computed for mean second x second HP at seconds 3, 9, and 12 with RT for the SPK and NSPK conditions for all subjects combined and for stutterers and nonstutterers separately (see Table 7). As can be seen in Table 7, although most correlations were negative and all were low, the

negative correlations were in the direction that suggested a relationship between slower RT and HR acceleration.

Table 7

Pearson Product Moment Correlations Between Mean HP Seconds and RT for Stutterers and Nonstutterers

Conditions	Seconds		
	3	9	12
	Stutterers and Nonstutterers		
SPK	-.11	-.18	-.17
NSPK	-.06	.13	-.16
	Stutterers Only		
SPK	.06	.18	-.16
NSPK	-.07	-.08	-.14
	Nonstutterers Only		
SPK	-.13	.09	-.11
NSPK	-.17	-.29	-.21

Pearson product moment correlations also were computed for mean HPV and RT for each Period. As indicated in Table 8, although all correlations were low and all but one were negative they too are in the expected direction of slower RT and greater HPV.

SCL and the Willoughby Questionnaire

Pearson product moment correlations were computed for mean base level log SCL and mean scores on the Willoughby Questionnaire.

These correlations are summarized in Table 9 for stutterers and Table 10 for nonstutterers.

Table 8

Pearson Product Moment Correlations Between Mean HPV and Mean RT for Stutterers and Nonstutterers

Condition	Periods		
	OR	Attend	Respond
SPK	-.10	.00	-.13
NSPK	-.23	-.29	-.16

Table 9

Pearson Product Moment Correlations for the Willoughby Questionnaire and Base Level Periods Log SCL for Stutterers

		Pre O_1	Post O_1	Pre O_2	Post O_2	Anxiety
		SPK-NSPK Order (O_1)				
Base Periods	Pre O_1	1.00				
	Post O_1	.87	1.00			
	Pre O_2	.88	.99	1.00		
	Post O_2	.99	.86	.87	1.00	
	Anxiety	-.84	-.52	-.54	-.88	1.00
		NSPK-SPK Order (O_2)				
Base Periods	Pre O_1	1.00				
	Post O_1	.99	1.00			
	Pre O_2	.97	.96	1.00		
	Post O_2	.98	.97	.94	1.00	
	Anxiety	.86	.88	.83	.77	1.00

Table 10

Pearson Product Moment Correlations for the Willoughby Questionnaire and Base Level Periods Log SCL for Nonstutterers

		Pre O_1	Post O_1	Pre O_2	Post O_2	Anxiety
		SPK-NSPK Order (O_1)				
Base Periods	Pre O_1	1.00				
	Post O_1	.98	1.00			
	Pre O_2	.95	.93	1.00		
	Post O_2	.91	.95	.94	1.00	
	Anxiety	.17	.10	.01	.06	1.00
		NSPK-SPK Order (O_2)				
Base Periods	Pre O_1	1.00				
	Post O_1	.99	1.00			
	Pre O_2	.98	.98	1.00		
	Post O_2	.99	.99	.99	1.00	
	Anxiety	-.04	-.14	-.15	-.14	1.00

As indicated in Table 9, stutterers showed high negative correlations between anxiety scores on the Willoughby Questionnaire and base level log SCL for the SPK-NSPK order and correlations in the opposite direction for the NSPK-SPK order. In each instance, the magnitude of the correlations accounts for a substantial portion of the variance. Correlations for nonstutterers (see Table 10) differed in two ways from those for stutterers. First, correlations for nonstutterers were markedly lower, independent of direction, and in fact can account for considerably little of the variance. Second, the directions of the correlations were opposite those for

stutterers; i.e., they were positive for the SPK-NSPK order and negative for the NSPK-SPK order.

Summary of Correlational Analyses

Reaction time was not correlated with HP, suggesting that all subjects had sufficient time to recover from speaking in order to perform the RT task. This is particularly interesting in light of the previous results indicating disrupted HR patterns among stutterers. Obviously, disruption of attention suggested by the HR patterns for stutterers was not great enough to interfere with performance on the task used in this study.

The significant correlations between SCL and scores on the Willoughby anxiety measure for stutterers and the lack of such correlations for nonstutterers suggests that anxiety played a more influential role in stutterers' tonic levels of arousal than it played in nonstutterers' levels of arousal. Interestingly enough, stutterers appeared to be influenced by the order in which they experienced the SPK-NSPK conditions. Tonic SCLs in the SPK-NSPK condition were inversely related to anxiety scores, whereas those in the NSPK-SPK order were positively related to anxiety scores.

CHAPTER V

DISCUSSION

The purpose of the present experiment was to investigate psychophysiological correlates of attention and anxiety in stutterers and nonstutterers. While seldom clear-cut, the results did provide support for attentional and anxiety-arousal differences between the groups. Equally important, however, was the fact that the results provided strong indications for the future directions research in this area should take.

Reaction Time Performance

None of the hypotheses regarding overall differences in RT were confirmed for main between-groups comparisons. Stutterers and nonstutterers did differ in RT from trial 1 to trial 2 as a function of the order in which conditions were presented. Reaction time was slower on trial 1 for stutterers when the speaking condition occurred first, but for nonstutterers RT was slower on trial 1 when the nonspeaking condition occurred first. At first glance, these differences may seem to reflect either practice effects or warm-up effects. However, if they are attributable to such effects it is difficult to see why the effects would have been specific to only two of the four possible combinations of groups x conditions. What does seem clear is that the specific situational demands were not

sufficiently difficult to disrupt RT performance for either stutterers or nonstutterers.

Pretesting of the sentences with nonstutterers indicated that mean duration of sentence utterance was about 3 sec. Despite the fact that stutterers did block while reading the distraction sentences, they obviously had ample time remaining in order to prepare for the RT task. However, there was one notable exception to this group tendency. Nine of the 10 stutterers clearly would have met the clinical criteria for classification as mild stutterers. On the other hand, it was equally clear that one of the stutterers would have been diagnosed as severe. (These classifications were not done systematically in the present study. The observations are based on the levels of fluency shown by the subjects before, during, and after the experiment and admittedly are subjective impressions based on my clinical experience with stutterers.) This subject had severe blocks on at least two of the speaking trials which interfered with RT performance. The difference between the RT of the severe stutterer and the RTs of other stutterers suggests that there may be important differences within stutterers that should be explored in future research. Although the severe stutterer completed the sentences before the respond signal came on, he spent considerably longer speaking the sentences during the PI due to the marked blocking that occurred. Perhaps these blocks sufficiently distracted him from the task to interfere with his performance. This particular subject's physiological data will be analyzed in the future to see if physiological correlates of attention and anxiety-arousal also

distinguish him from the rest of the stutterers in the present study.

Based on the present results, several directions for future research are suggested. The fact that the distraction sentences did not seem to affect mild stutterers' RT performance in a 12-sec PI RT task suggests at least four possible manipulations to be considered in future studies involving mild stutterers. The first manipulation involves the length of the PI interval. If the PI were shorter it should enhance task difficulty. This effect would at least be consistent with similar studies of PI effects (Krupski, 1975). The second manipulation involves the nature of the sentences themselves. It is possible that the distraction sentences were too easy. If Wingate's (1976) hypothesis is correct regarding the role of stressed vowels as the immediate source of stuttering, sentences specifically constructed to emphasize movement into stressed vowels may be more interfering than the sentences in the present study which were constructed to reflect critical consonants. In fact, if Wingate's (1976) hypothesis is correct, one should be able to construct specific sentences predictive of the degree to which they should elicit stuttering blocks. The third manipulation involves the nature of the task. Speech pathologists frequently report that stutterers have less difficulty reading than they do spontaneously speaking (e.g., Brajovic & Brajovic, 1974). This is particularly the case when the reading material is familiar--called the adaptation effect in speech pathology literature. If a task could be constructed which incorporated aspects of spontaneous speech while

retaining sufficient experimental control of the spoken material, task difficulty may be increased to the point that the hypothesized differences in RT performance would appear. The fourth manipulation involves the various combinations of the previous three in a series of parametric studies. Of course, all of these suggestions for future research are predicated on the assumption that a relationship between stuttering and RT performance can be isolated. The lack of group differences in RT in the present study certainly challenges this assumption. Nevertheless, when one considers the physiological data in the present study the assumption seems to have merit.

Stuttering and Attention

Analysis of the HP correlates of attention strongly implicates attentional dysfunction in stutterers when they are required to speak. Specifically, the second x second analysis of HP during the Attend Period of the speaking condition indicated that stutterers experienced a 2-sec delay in the formation of the decelerative HR response typically considered to be associated with attention (Lacey, 1959; Luria, 1973; Obrist et al., 1974). In addition, the magnitude of their decelerative response was considerably less than that of nonstutterers. For example, HR decelerations for stutterers did not drop below baseline levels during the speaking condition, whereas those for stutterers did. The fact that HR deceleration patterns during the nonspeaking condition were virtually identical for stutterers and nonstutterers provides additional support for the

contention that the speaking task was responsible for the attentional dysfunction noted above. Moreover, the topography of the HR patterns for both groups during the nonspeaking conditions was virtually identical to those that commonly appear in the RT literature.

Further evidence that speaking disrupted attention in stutterers is provided by the failure of the temporal conditioning component of the fixed PI to facilitate the formation of the decelerative HR pattern associated with attention in the fixed PI task (Fitzgerald & Porges, 1971; Krupski, 1975). Nonstutterers showed a uniphasic HR deceleration pattern that increased in magnitude and moved temporally closer to the respond signal from trial 1 to trial 10. Stutterers showed a pattern which differed during the early recruitment phase of the response and at the terminal phase of the response. By trial 10, nonstutterers showed a HR decelerative pattern that began on the 4th second of the PI and continued gradually to the 11th second at which point an anticipatory acceleration began. Stutterers showed a biphasic pattern. They began to decelerate at the 3rd second of the PI but showed an acceleration from the 7th to the 9th seconds which in the speaking conditions corresponds to the time immediately following completion of their reading. This acceleration was followed by a deceleration to the respond signal with no anticipatory acceleration evident in the pattern.

In terms of Porges' (1976) model of attention, stutterers showed disruption in the long latency portion of the reactive component of attention. This makes logical sense when one considers

basic differences and similarities between stutterers and nonstutterers. Stutterers and nonstutterers differ fundamentally in the degree of dysfluency characteristic of their speech, not in their basic cognitive and emotional characteristics in nonspeech situations. Thus, one should expect that any attentional differences between the two groups should be ones associated with speech production itself, and the present data seem to reflect this.

In general, the HR seconds analyses were supported by the analyses for HR performed for each Period of the RT trials. The analyses of HPV gave general support to the greater disruptive effect of the SPK-NSPK order on stutterers' physiological activity. The fact that neither HP nor HPV was significantly correlated with RT may reflect the type of PI used in the present study. Porges (1972) has argued that the relationship between HPV and RT is best demonstrated with a variable PI. In the present study the more conservative fixed PI was used to allow for the possibility that temporal conditioning would facilitate the recruitment of the HR decelerative response. The variable PI eliminates the possibility that the subject may develop a temporal expectancy regarding the relationship between the stimuli and therefore should enhance the task demands for the subject. Consequently, one may add the use of the variable PI to the list of manipulations to be investigated in future research.

It is tempting to speculate on the possible implications of the present results for specific therapeutic treatment techniques used with stutterers. For example, one can find aspects of temporal

conditioning in several therapeutic methods used with stutterers (Van Riper, 1973). These include breath control, paced or slow speech, metronome pacing of breathing, and time estimation. While these methods appear to help some stutterers achieve fluency, they are far from being uniformly successful therapeutic interventions. The present results would suggest that pacing or rhythmic therapeutic techniques may not be the most efficacious methods to facilitate the development of attention in stutterers. Of course, in the present experiment subjects received only a total of 20 trials (and only 10 in each condition), whereas hundreds and hundreds of "trials" usually are involved in the therapeutic situation. Therefore, it would be interesting to see if additional trials would allow the temporal conditioning component to facilitate the recruitment of the attentional response during the speaking condition for stutterers. Moreover, it would be interesting to use actual rhythmic therapeutic methods (e.g., metronome pacing) as the subject's task.

The HR results support those involving RT in suggesting that PI length may be an important constraint on the development of attention. Working with adult retardates and normals Krupski (1975) found that PI lengths of 4 and 7 sec were associated with poor recruitment of the HR decelerative response in retardates. When a 13-sec PI was used the HR decelerative component of attention became more similar between normals and retardates although the patterns for retardates were more erratic. Sroufe et al. (1973) report analogous findings for differences between minimal brain dysfunction children and normal children. In the present study, a 12-sec PI

was used to allow stutterers sufficient time to finish reading the distraction sentences in the event that stuttering blocks occurred. In fact, since there are no inherent reasons to suspect that stutterers and nonstutterers differ in their RT ability or in attentional processes other than those associated with speaking situations, the long PI may have precluded any relationship between the HR decelerative response and RT from developing. Shorter PI lengths on the order of 7 to 9 sec, matching the intervals where stutterers showed HR acceleration during the speaking condition, may just enable this relationship to develop.

Analyses of respiration amplitude and frequency were consistent with the HP analyses. Speaking was associated with marked reductions in respiration amplitude and frequency during the OR Period; that is, when subjects were speaking. These differences are expected since respiratory pauses typically accompany speech. The marked increase in respiration amplitude during the Attend Period is consistent with the HR acceleration pattern during the same interval for stutterers during the speaking condition. While the method of data recording in the present study did not allow computer generation of weighted coherence measures of autonomic balance, the data are at least consistent with those Porges (1976) reports for his coherence measure obtained from hyperactive children. Until such measurements are possible, one must be satisfied with the hypothesis that stutterers may fluctuate from sympathetic dominance to parasympathetic dominance as a function of whether or not they are experiencing a stuttering block. This interpretation of the present

data would suggest that stutterers shift to sympathetic dominance during speech situations which are accompanied by stuttering blocks and then have a slower recruitment of parasympathetic dominance immediately following the speech situation. Such an hypothesis would account for the disorganized pattern of HR deceleration found in the present study for stutterers in the speaking condition.

Considering the HP and respiration analyses combined, it is possible to hypothesize a behavioral picture of the stutterers' performance during the speaking condition. Stutterers and nonstutterers appear to react similarly during speech situations which do not involve stuttering blocks. When a stuttering block occurs, however, stutterers appear to have a period of time in which their attention narrows (perhaps to their efforts to restrain the block) and becomes disrupted during the next few seconds following the end of the spoken utterance. If the stutterer must continue to speak the attentional disruption may lead to additional blocking in a cyclic or positive feedback fashion. If the stutterer is also anxious about the speech situation his anxiety may further compound the disruptive effects of the positive feedback condition.

Several authors have suggested that high arousal and/or anxiety is associated with narrowing of attention (Callaway & Stone, 1960; Wachtel, 1968; Zaffy & Brunning, 1966). Thus, if the stutterer is anxious during the speech situation his attention may be directed away from such aspects of the speech context as word selection, cognitive content, or ideation. This may cause the stutterer to speak before thinking through what he wishes to say, or before

deciding how to express his thoughts. Interestingly enough, Bode and Bruten (1963) have reported that even fluent speakers stutter in certain speech-anxiety situations.

Stuttering and Anxiety-Arousal

Analyses of electrodermal activity indicated that both stutterers and nonstutterers showed higher levels of arousal (log SCL) during the speaking condition than during the nonspeaking condition. Moreover, both groups showed higher magnitude SCRs during the OR and Respond Periods compared to the Pre and Attend Periods. Higher SCRs during these Periods can be attributed to the elicitation properties of the Warning signal and Respond signal, respectively. The gradual decrement in SCR magnitude over trials for each Period can be attributed to habituation to the task situation. Electrodermal frequency also was higher during the OR Period than in other Periods and in the Attend Period compared to the Pre and Respond Periods. The EDF following the warning signal is expected due to the elicitation properties of the signal. However, the higher EDF during the Attend Period is more difficult to account for. It may reflect a recruitment effect or rebound effect attributable to the SCRs elicited by the warning signal (Bundy & Fitzgerald, 1975), or it may reflect an anxiety-arousal effect analogous to the HR acceleration patterns observed for stutterers during the speaking condition. In any event, it is clear that stutterers and nonstutterers did not differ in their basic arousal reactions as evidenced in log SCL, SCRs, and EDF data.

Nevertheless, there were differences within the stuttering group in their electrodermal activity. SCL for stutterers was different among Periods in the SPK-NSPK order. There stutterers showed higher SCL during the OR and Attend Periods compared to the Preperiod and Respond Period. This suggests that stutterers were more highly aroused by the warning signal during the SPK-NSPK order than during the NSPK-SPK order for the SPK condition.

When one considers the relationship between base level log SCL and anxiety scores on the Willoughby Questionnaire, a totally different picture and a somewhat paradoxical one emerges regarding the relationship of anxiety and arousal for stutterers in this experimental situation. Stutterers did score significantly higher in anxiety on the Willoughby Questionnaire than did nonstutterers. The significant correlations between Willoughby scores and base level log SCL for stutterers and the lack of such correlations for nonstutterers suggests that anxiety levels were different for the two groups. This suggests the intriguing possibility that the ED measures may have been reflecting arousal levels for nonstutterers but reflecting an anxiety-arousal interaction for stutterers.

The presence of high negative correlations between base level log SCL and Willoughby scores for stutterers in the SPK-NSPK order and high positive correlations in the NSPK-SPK order may reflect the time at which the Willoughby Questionnaire was administered. For stutterers in the SPK-NSPK order the Questionnaire was administered some time after they had experienced the nonspeaking condition. For stutterers in the NSPK-SPK order the Questionnaire was

administered immediately after they experienced the speaking condition. In the NSPK-SPK order it may have been the case that anxiety associated with speaking influenced stutterers' answers to the items on the Willoughby scale. Stutterers in the SPK-NSPK order would have had their anxiety reduced by the NSPK condition by the time they took the Willoughby test.

Finally, the fact that stutterers tended to score toward the negative end of three of the four categories of the Stutterer's Self Rating of Reactions to Speech Situations suggested that this sample of stutterers tends to avoid speech situations, dislikes speaking in certain situations, and tends to stutter more in some situations than in others. While these tendencies are at least consistent with an anxiety interpretation of stuttering, they should be accepted with caution due to the lack of solid normative data available for the Self Rating instrument.

Summary

The results of the present experiment provide some support for both attentional and anxiety-arousal correlates of stuttering. However, in neither instance were the findings clear-cut. Nevertheless, the results of the present study strongly suggest the directions future research should take. It was suggested that at least the following factors should be taken into account in subsequent studies: (a) the length of the PI; (b) the nature of the PI, i.e., the use of variable PIs and fixed PIs; (c) the nature of the distraction sentences, i.e., reading versus spontaneous speech;

(d) the composition of the distraction sentences, i.e., whether the sentences are constructed to emphasize critical consonants or movement into stressed vowels; (e) the use of extended trials beyond the 10 used in the present experiment; (f) the nature of the performance task, i.e., using a task that is actually used in therapeutic interventions but which contains some of the components of the present task; (g) recording of dependent variables on magnetic tape for computer analysis so that coherence measures of autonomic balance can be derived; and (h) more precision in the assessment of anxiety as well as arousal so that the arousal-anxiety interaction can be better understood in relation to the act of speaking in stutterers.

Taken as a whole, the results of the present study clearly support the use of electrophysiological dependent variables in the analysis of the correlates of psychological mechanisms associated with stuttered speech. Whether these measures will eventually lead to a clearer understanding of stuttering and to improvements in therapeutic interventions remains an unanswered empirical question.

APPENDICES

APPENDIX A

HUMAN SUBJECTS INFORMED CONSENT FORM

APPENDIX A

HUMAN SUBJECTS INFORMED CONSENT FORM

Michigan State University
Department of Psychology

DEPARTMENTAL RESEARCH CONSENT FORM

1. I have freely consented to take part in a scientific study being conducted by:_____

   under the supervision of:_____

   Academic Title:_____

2. The study has been explained to me and I understand the explanation that has been given and what my participation will involve.

3. I understand that I am free to discontinue my participation in the study at any time without penalty.

4. I understand that the results of the study will be treated in strict confidence and that I will remain anonymous. Within these restrictions, results of the study will be made available to me at my request.

5. I understand that my participation in the study does not guarantee any beneficial results to me.

6. I understand that, at my request, I can receive additional explanation of the study after my participation is completed.

Signed_____

Date_____

APPENDIX B

INSTRUCTIONS FOR THE REACTION TIME SPEECH STUDY

APPENDIX B

INSTRUCTIONS FOR THE REACTION TIME SPEECH STUDY

In this experiment we will place sensors on your skin in order to record your physiological activity. The equipment with which you have been connected will measure your heart rate, the activity of your sweat glands, and your respiration rate while you perform a reaction time task. The equipment is sensitive to movement so please do not make any unnecessary movements that would produce interference on the polygraph recording sheet. There are three parts to this experiment. In the first two parts you will perform a reaction time task while in the third part you will fill out the questionnaires that I will show you later.

A. Instructions for the speaking condition

Part I

In this part of the experiment you should sit relaxed in the armchair with your preferred hand holding the reaction time button. In front of you on the wall are two lights. One of the lights is red and one is green. Your task is to watch these two lights. The red light (point to it) is the warning light. When it comes on you must do two things: First you must prepare to press the button as fast as possible when the green light comes on. Second, you must say one sentence from the list that I gave you (present the list). There will be 10 trials and in each of them you must say a different sentence in the order in which they are written. Be sure to complete the sentence before the green light comes on. When the green light goes on you should press as fast as possible the button you are holding. As soon as the green light goes off, release your pressure on the button. At the end of these 10 trials there will be a brief rest period during which I will give you new instructions. Do you understand what you are to do? Do you have any other questions? OK. After I close the door it will take a few moments to check the equipment. The first light will come on about 1 minute after I say "Ready?" If you find it necessary to talk to me during the experiment, simply do so. I will be able to hear you through the intercom which connects this room with the equipment room.

Part II

In this part of the experiment you should sit relaxed in the armchair with your hand holding the reaction time button. This part differs from the first part in that now you will not say sentences. You will perform the reaction time task without speaking.

Namely, when the red light comes on you have to prepare to press the button. And, when the green light comes on you have to press the button as quickly as possible. As soon as the green light goes off, release your pressure on the button. There will be 10 trials. At the end of these 10 trials I will give you 3 questionnaires to fill out. After completing these questionnaires the experiment will be over. Do you have any questions?

B. Instructions for the nonspeaking condition

These were essentially the opposite of those for the speaking condition, appropriately rephrased for orders in which the non-speaking condition occurred first.

APPENDIX C

ANALYSES OF VARIANCE SUMMARY TABLES

APPENDIX C

ANALYSIS OF VARIANCE SUMMARY TABLES

Table 11

Analysis of Variance Summary Table for Reaction Time (msec)

Source	df	MS	F	p
Between Subjects				
Group (A)	1	19.46025	.28	ns
Order (B)	1	39.40225	.56	ns
A x B	1	53.59225	.76	ns
Sub. w. gps.	16	70.05675		
Within Subjects				
Condition (C)	1	33.30625	2.57	ns
A x C	1	45.58225	3.52	ns
B x C	1	65.79225	5.08	.039
A x B x C	1	36.67225	2.83	ns
C x Sub. w. gps.	16	12.94325		
Trial (D)	9	31.84281	5.08	.0005
A x D	9	5.96414	.95	ns
B x D	9	5.08503	.81	ns
A x B x D	9	13.02725	2.08	.035
D x Sub. w. gps.	144	6.27217		
C x D	9	7.52125	1.19	ns
A x C x D	9	10.04725	1.60	ns
B x C x D	9	17.19614	2.73	.006
A x B x C x D	9	7.63281	1.21	ns
C x D x Sub. w. gps.	144	6.29394		

Table 12

Analysis of Variance Summary Table for Heart Period Seconds for Stutterers

Source	df	MS	F	p
Order (A)	1	11808.44	.04	ns
Error:	8	291770.00		
Condition (B)	1	2423211.00	61.65	.0005
A x B	1	814807.11	20.73	.002
Error:	8	39308.56		
Trial (C)	9	34857.73	.39	ns
A x C	9	46887.70	.51	ns
Error:	72	90501.42		
Seconds (D)	17	106551.82	8.17	.0005
A x D	17	12904.21	.99	ns
Error:	136	13045.82		
B x C	9	33002.47	.35	ns
A x B x C	9	38701.68	.42	ns
Error:	72	93029.60		
B x D	17	30216.05	2.50	.002
A x B x D	17	6564.53	.54	ns
Error:	136	12107.08		
C x D	153	4681.39	1.34	.005
A x C x D	153	3574.05	1.03	ns
Error:	1224	3486.66		
B x C x D	153	4045.58	1.04	ns
A x B x C x D	153	4356.08	1.12	ns
Error:	1224	3883.30		

Table 13

Analysis of Variance Summary Table for Heart Period Seconds for Nonstutterers

Source	df	MS	F	p
Order (A)	1	187200.44	.79	ns
Error:	8	234454.67		
Condition (B)	1	2160900.00	8.10	.02
A x B	1	418177.78	1.57	ns
Error:	8	266463.89		
Trial (C)	9	95389.88	1.39	ns
A x C	9	98991.80	1.45	ns
Error	72	68236.02		
Seconds (D)	17	115581.59	15.79	.0005
A x D	17	7383.20	1.00	ns
Error:	136	7318.60		
B x C	9	61724.94	.83	ns
A x B x C	9	97383.46	1.30	ns
Error:	72	94768.09		
B x D	17	50445.12	7.18	.0005
A x B x D	17	11601.72	1.66	ns
Error:	136	7021.66		
C x D	153	3561.66	1.49	.0005
A x C x D	153	2312.48	.96	ns
Error:	1224	2384.60		
B x C x D	153	2106.26	.86	ns
A x B x C x D	153	3194.71	1.31	.009
Error:	1224	2427.82		

Table 14

Analysis of Variance Summary Table for Heart Period Base Levels

Source	df	MS	F	p
Group (A)	1	16290.63	.15	ns
Order (B)	1	1463.76	.01	ns
A x B	1	11012.12	.09	ns
Error:	16	110939.96		
Condition (C)	1	9600.96	3.74	.071
A x C	1	3527.16	1.37	ns
B x C	1	200.34	.08	ns
A x B x C	1	25.76	.01	ns
Error:	16	2562.24		
Base Period (D)	1	524.29	.17	ns
A x D	1	2933.04	.94	ns
B x D	1	10.22	.01	ns
A x B x D	1	1165.86	.37	ns
Error:	16	2116.86		
C x D	1	551.25	.17	ns
A x C x D	1	773.76	.24	ns
B x C x D	1	32.00	.01	ns
A x B x C x D	1	2773.01	.87	ns
Error:	16	3189.53		

Table 15

Analysis of Variance Summary Table for Heart Period Periods for Stutterers and Nonstutterers

Source	df	MS	F	p
Group (A)	1	954.09	.03	ns
Order (B)	1	2471.07	.09	ns
A x B	1	476.28	.02	ns
Error:swg	16	28555.07		
Condition (C)	1	543320.96	29.40	.0005
A x C	1	5650.68	.31	ns
B x C	1	43440.33	2.35	ns
A x B x C	1	726.96	.04	ns
Error: C x swg	16	18478.61		
Trial (D)	9	20372.94	3.00	.003
A x D	9	3412.69	.50	ns
B x D	9	6414.03	.95	ns
A x B x D	9	13658.96	2.01	.04
Error: D x swg	144	6788.54		
Periods (E)	2	9697.12	26.43	.0005
A x E	2	1182.35	.32	ns
B x E	2	376.23	.10	ns
A x B x E	2	5280.38	1.44	ns
Error: E x swg	32	3669.76		
C x D	9	1841.74	.25	ns
A x C x D	9	5658.01	.76	ns
B x C x D	9	11473.57	1.54	ns
A x B x C x D	9	8335.12	1.12	ns
Error: CD x swg	144	7449.97		
C x E	2	28356.46	6.22	.005
A x C x E	2	1000.97	.22	ns
B x C x E	2	7351.11	1.61	ns
A x B x C x E	2	815.10	.17	ns
Error: CE x swg	32	4557.42		
D x E	18	4644.53	3.84	.0005
A x D x E	18	1262.77	.41	ns
B x D x E	18	948.03	.72	ns
A x B x D x E	18	695.88	.92	ns
Error: DE x swg	288	1209.61		
C x D x E	18	1581.10	1.23	ns
A x C x D x E	18	1103.68	.86	ns
B x C x D x E	18	1979.56	1.55	ns
A x B x C x D x E	18	1787.48	1.40	ns
Error: CDE x swg	288	1275.78		

Table 16

Analysis of Variance Summary Table for Heart Period Variability for Stutterers and Nonstutterers

Source	df	MS	F	p
Group (A)	1	42662907.00	.33	ns
Order (B)	1	27.60	.00	ns
A x B	1	809588.80	.01	ns
Error:	16	125773178.00		
Condition (C)	1	803129425.00	7.97	.012
A x C	1	813791.67	.01	ns
B x C	1	230873778.00	2.29	ns
A x B x C	1	72673035.00	.72	ns
Error:	16	100643875.00		
Trial (D)	9	62088926.00	1.37	ns
A x D	9	122615441.00	2.70	.006
B x D	9	90220287.00	1.98	.045
A x B x D	9	54395342.00	1.19	ns
Error:	144	45398438.00		
Period (E)	2	28526896.00	1.08	ns
A x E	2	19906344.00	.76	ns
B x E	2	2932477.00	.11	ns
A x B x E	2	3271176.00	.12	ns
Error:	32	26335549.00		
C x D	9	65114182.00	1.31	ns
A x C x D	9	49738378.00	1.00	ns
B x C x D	9	83535433.00	1.69	ns
A x B x C x D	9	38503834.00	.77	ns
Error:	144	49476145.00		
C x E	2	143360519.00	3.05	ns
A x C x E	2	63912606.00	1.36	ns
B x C x E	2	28772020.00	.61	ns
A x B x C x E	2	47979949.00	1.02	ns
Error:	32	46944167.00		
D x E	18	12177336.00	1.07	ns
A x D x E	18	14696036.00	1.29	ns
B x D x E	18	17419690.00	1.52	ns
A x B x D x E	18	20567107.00	1.80	.025
Error:	288	11408509.00		
C x D x E	18	9711669.00	.65	ns
A x C x D x E	18	12457982.00	.83	ns
B x C x D x E	18	15293991.00	1.01	ns
A x B x C x D x E	18	19164966.00	1.28	ns
Error:	288	15001712.00		

Table 17

Analysis of Variance Summary Table for Heart Period Variability for Baseline Data

Source	df	MS	F	p
Group (A)	1	15162457.00	.93	ns
Order (B)	1	1872016.00	.11	ns
A x B	1	4166978.00	.26	ns
Error:	16	16308465.00		
Condition (C)	1	26953196.00	3.83	.068
A x C	1	267463.76	.04	ns
B x C	1	2026139.00	.29	ns
A x B x C	1	6103081.00	.87	ns
Error:	16	7032822.00		
Base Period (D)	1	8268530.00	1.18	ns
A x D	1	15962646.00	2.27	ns
B x D	1	1213446.00	.17	ns
A x B x D	1	280809.45	.04	ns
Error:	16	7016284.00		
C x D	1	950327.41	.13	ns
A x C x D	1	20065153.00	2.78	ns
B x C x D	1	25448470.00	3.53	.079
A x B x C x D	1	9996909.00	1.83	ns
Error:	16	7209638.00		

Table 18

Analysis of Variance Summary Table for Respiration Frequency for Stutterers and Nonstutterers

Source	df	MS	F	p
Group (A)	1	4.44	1.99	ns
Order (B)	1	1.40	.63	ns
A x B	1	.30	.13	ns
Error:	16	2.23		
Condition (C)	1	12.61	8.03	.012
A x C	1	2.34	1.49	ns
B x C	1	1.68	1.08	ns
A x B x C	1	8.17	5.20	.037
Error:	16	1.56		
Trial (D)	9	1.28	1.15	ns
A x D	9	.74	.67	ns
B x D	9	.61	.54	ns
A x B x D	9	.83	.75	ns
Error:	144	1.11		
Period (E)	2	7.79	3.94	.030
A x E	2	2.03	1.03	ns
B x E	2	1.30	.66	ns
A x B x E	2	4.98	2.51	ns
Error:	32	1.98		
C x D	9	1.21	1.03	ns
A x C x D	9	1.79	1.52	ns
B x C x D	9	.75	.63	ns
A x B x C x D	9	3.29	2.79	.005
Error:	144	1.18		
C x E	2	.43	.77	ns
A x C x E	2	.77	1.37	ns
B x C x E	2	.25	.43	ns
A x B x C x E	2	.93	1.64	ns
Error:	32	.57		
D x E	18	.37	1.15	ns
A x D x E	18	.32	.99	ns
B x D x E	18	.33	1.02	ns
A x B x D x E	18	.31	.97	ns
Error:	288	.32		
C x D x E	18	.37	1.09	ns
A x C x D x E	18	.29	.86	ns
B x C x D x E	18	.40	1.18	ns
A x B x C x D x E	18	.31	.92	ns
Error:	288	.34		

Table 19

Analysis of Variance Summary Table for Respiration Amplitude for Stutterers and Nonstutterers

Source	df	NS	F	p
Group (A)	1	669.90	1.64	ns
Order (B)	1	80.80	.19	ns
A x B	1	305.42	.74	ns
Error:	16	408.57		
Condition (C)	1	3211.79	18.49	.001
A x C	1	958.37	5.51	.032
B x C	1	166.95	.96	ns
A x B x C	1	660.08	3.80	ns
Error:	16	173.65		
Trial (D)	9	261.87	1.77	ns
A x D	9	102.24	.69	ns
B x D	9	96.11	.65	ns
A x B x D	9	67.96	.46	ns
Error:	144	147.70		
Period (E)	2	1818.13	12.52	.0005
A x E	2	60.45	.42	ns
B x E	2	24.93	.17	ns
A x B x E	2	21.87	.15	ns
Error:	32	145.13		
C x D	9	128.69	1.47	ns
A x C x D	9	96.05	1.09	ns
B x C x D	9	196.35	2.24	.023
A x B x C x D	9	101.35	1.16	ns
Error:	144	87.65		
C x E	2	1500.72	10.41	.0005
A x C x E	2	130.09	.09	ns
B x C x E	2	81.24	.56	ns
A x B x C x E	2	97.93	.68	ns
Error:	32	144.19		
D x E	18	22.10	.59	ns
A x D x E	18	45.76	1.24	ns
B x D x E	18	33.40	.91	ns
A x B x D x E	18	57.60	1.56	ns
Error:	288	36.84		
C x D x E	18	35.98	1.11	ns
A x C x D x E	18	29.74	.92	ns
B x C x D x E	18	14.24	.44	ns
A x B x C x D x E	18	29.31	.90	ns
Error:	288	32.43		

Table 20

Analysis of Variance Summary Table for Skin Conductance Level for Stutterers and Nonstutterers

Source	df	NS	F	p
Group (A)	1	6.63	.55	ns
Order (B)	1	8.43	.70	ns
A x B	1	2.95	.25	ns
Error:	16	12.04		
Condition (C)	1	1.88	4.52	.049
A x C	1	.08	.19	ns
B x C	1	.19	.46	ns
A x B x C	1	.01	.01	ns
Error:	16	.42		
Trial (D)	9	.18	2.76	.005
A x D	9	.11	1.72	ns
B x D	9	.08	1.33	ns
A x B x D	9	.07	1.09	ns
Error:	144	.06		
Period (E)	3	.03	2.91	.044
A x E	3	.01	.24	ns
B x E	3	.02	1.55	ns
A x B x E	3	.03	2.88	.045
Error:	48	.01		
C x D	9	.03	.72	ns
A x C x D	9	.02	.49	ns
B x C x D	9	.02	.58	ns
A x B x C x D	9	.04	.97	ns
Error:	144	.04		
C x E	3	.01	3.21	.031
A x C x E	3	.01	.34	ns
B x C x E	3	.01	.67	ns
A x B x C x E	3	.01	.67	ns
Error:	48	.01		
D x E	27	.02	2.69	.0005
A s C x E	27	.01	.86	ns
B x D x E	27	.01	.69	ns
A x B x D x E	27	.01	1.19	ns
Error:	432	.01		
C x D x E	27	.01	1.27	ns
A x C x D x E	27	.01	.36	ns
B x C x D x E	27	.01	.62	ns
A x B x C x D x E	27	.01	1.32	ns
Error:	432	.01		

Table 21

Analysis of Variance Summary Table for Skin Conductance Responses for Stutterers and Nonstutterers

Source	df	MS	F	p
Group (A)	1	178.76	.21	ns
Order (B)	1	1020.80	1.21	ns
A x B	1	2658.94	3.17	ns
Error:	16	838.07		
Condition (C)	1	185.07	.43	ns
A x C	1	552.48	1.27	ns
B x C	1	44.56	.10	ns
A x B x C	1	3.61	.01	ns
Error:	16	435.80		
Trial (D)	9	158.43	4.81	.0005
A x D	9	37.23	1.13	ns
B x D	9	45.81	1.39	ns
A x B x D	9	30.97	.94	ns
Error:	144	32.93		
Period (E)	3	6046.33	21.33	.0005
A x E	3	43.02	.15	ns
B x E	3	320.87	1.13	ns
A x B x E	3	515.10	1.81	ns
Error:	48	283.52		
C x D	9	24.92	.78	ns
A x C x D	9	25.73	.81	ns
B x C x D	9	41.82	1.31	ns
A x B x C x D	9	2.48	.07	ns
Error:	144	31.90		
C x E	3	960.42	7.18	.0005
A x C x E	3	252.50	1.89	ns
B x C x E	3	17.44	.13	ns
A x B x C x E	3	35.74	.27	ns
Error:	48	133.69		
D x E	27	93.63	3.47	.0005
A x D x E	27	26.53	.98	ns
B x D x E	27	20.31	.75	ns
A x B x D x E	27	35.38	1.31	ns
Error:	432	27.00		
C x D x E	27	22.76	.80	ns
A x C x D x E	27	32.30	1.14	ns
B x C x D x E	27	37.40	1.32	ns
A x B x C x D x E	27	33.92	1.20	ns
Error:	432	28.22		

Table 22

Analysis of Variance Summary Table for Electrodermal Frequency for Stutterers and Nonstutterers

Source	df	MS	F	p
Group (A)	1	17.02	2.57	ns
Order (B)	1	11.73	1.78	ns
A x B	1	14.63	2.22	ns
Error:	16	6.60		
Condition (C)	1	21.39	10.97	.004
A x C	1	3.52	1.80	ns
B x C	1	1.15	.59	ns
A x B x C	1	.01	.01	ns
Error:	16	1.94		
Trial (D)	9	1.02	3.53	.001
A x D	9	.52	1.78	ns
B x D	9	.34	1.18	ns
A x B x D	9	.18	.62	ns
Error:	144	.29		
Period (E)	3	37.76	38.97	.0005
A x E	3	.09	.09	ns
B x E	3	.97	1.00	ns
A x B x E	3	.84	.87	ns
Error:	48	.97		
C x D	9	.22	.63	ns
A x C x D	9	.37	1.04	ns
B x C x D	9	.83	2.34	.017
A x B x C x D	9	.41	1.16	ns
Error:	144	.35		
C x E	3	6.96	8.32	.0005
A x C x E	3	.09	.12	ns
B x C x E	3	1.58	1.89	ns
A x B x C x E	3	.63	.75	ns
Error:	48	.84		
D x E	27	.47	1.45	ns
A x D x E	27	.49	1.50	ns
B x D x E	27	.48	1.49	ns
A x B x D x E	27	.30	.91	ns
Error:	432	.33		
C x D x E	27	.46	1.24	ns
A x C x D x E	27	.33	.89	ns
A x B x C x D x E	27	.36	.98	ns
Error:	432	.36		

Table 23

Analysis of Variance Summary Table for Log Skin Conductance Level for Base Level Periods

Source	df	MS	F	p
Group (A)	1	.72485281	.95	ns
Order (B)	1	1.26454200	1.67	ns
A x B	1	.83187126	1.09	ns
Error:	16	.75662744		
Condition (C)	1	.07842529	.91	ns
A x C	1	.00798001	.09	ns
B x C	1	.02120307	.25	ns
A x B x C	1	.00861540	.10	ns
Error:	16	.08609972		
Period (D)	1	.00128320	.07	ns
A x D	1	.00076014	.04	ns
B x D	1	.03295096	.03	ns
A x B x D	1	.00228338	.13	ns
Error:	16	.01711958		
C x D	1	.09652162	.87	ns
A x D x D	1	.12661178	1.15	ns
B x C x D	1	.00712154	.65	ns
A x B x C x D	1	.12279012	1.12	ns
Error:	16	.11003359		

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