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VOICE REACTION TIME AND CORTICAL AUDITORY RESPONSE
LATENCIES IN STUTTERING AND NONSTUTTERING ADULTS

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DEDICATION

This dissertation is dedicated to my wife, Alexandra Rowland Molt. Without her love, support, encouragement, and belief, I could not have accomplished what I have. Her sacrifice these past seven years has been as great as my own; she deserves the diploma as much as I do.

I also take this opportunity to thank my family:

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And finally, my daughter, Kristen Alexandra, and my son, Ryan Douglas, who were asked to give up their Daddy on far too many evenings and weekends.

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ABSTRACT

The purpose of the present study was to examine cortical auditory system activity of stutterers as a possible explanation of slower voice reaction time responses previously reported for stutterers. Mean latencies of the auditory electroencephalic responses (AER) N100 (N1) and P200 (P2) and mean voice reaction time (VRT) to an auditory stimulus were investigated for adult stutterers and normal speakers. Two conditions were utilized. One condition (AER-VR) involved recording AERs during a voice reaction time task. In this condition each subject phonated a previously specified speech sound (/^/) as quickly as possible upon hearing the onset of a 1000 Hertz (Hz) frequency tone. AERs were recorded for the 1000 Hz. stimulus tone. In the second condition, listening- only (AER-LO), AERs were recorded while the subjects listened to the same 1000 Hz. stimulus tone without producing a phonatory response. The subjects participating were eight stutterers and eight nonstutterers, with the two groups matched for age and sex. All subjects presented normal hearing sensitivity for pure tone auditory stimuli and exhibited no abnormal neurological symptoms. All members of the stuttering group presented a childhood onset of stuttering.

Each subject completed the AER-VR condition first. In this condition each subject was instructed to phonate the sound / $\wedge$ / as rapidly as possible upon hearing the onset of a 1000 Hz. tone presented at a level 50 dB above her/his auditory threshold. Tone stimuli were presented in seven sets of 20 tones each, yielding a total of 140 tone presentations. The subjects were informed that the initial set would be utilized solely for practice purposes. Tones were presented with randomly assigned inter-stimulus-intervals of five to eight seconds. A three minute rest period was allowed between sets. Voice reaction time was judged as the time between stimulus tone onset and the onset of a phonatory response detected by a contact microphone placed over the laryngeal area. Because AER responses to the acoustic stimulus also were being recorded during this condition, subjects were instructed to maintain a relaxed body posture with fixed gaze. They were requested to minimize all body movement during presentation of sets. A five mm thick bite block placed between the upper and lower rear molars was utilized to reduce jaw motion during phonation. Only perceptibly fluent utterances were utilized for study. Trials were discarded in which obvious struggle behavior was observed or in which vocal response latency exceeded one second.

Upon completion of the AER-VR condition and after a short rest, each subject heard five additional sets of the tones. No physical response was required in this listening-only condition. AER latencies again were monitored during each set.

Electroencephalographic signals were recorded from a vertex site referenced to linked ear electrodes during a constant two second interval beginning just prior to the onset of each stimulus tone. Computer sampling of these two channels, the microphone channel, and an eye movement artifact channel every 7.5 ms allowed rejection of responses to any trial contaminated with excessive eye movement prior to computer summation of the recorded EEG signal. The averaged waveform obtained through the computer summation procedures was examined for latency of the N1 and P2 peak values. The computer also provided values of VRT latency for all non-artifacted trials excluding the initial practice set of tones.

Five hypotheses relating voice reaction time and auditory processing as measured by auditory electroencephalic response latency were tested in the present study. Single factor analysis of variance (ANOVA) computations were utilized to examine differences between stutterers and normal speakers for each of the factors VRT, AER N1 latency in the AER-LO and the AER-VR conditions, and AER P2 latency in each of the two response

conditions. The difference in AER latency between the two conditions (referred to as latency shift) and the difference between stutterers and nonstutterers in latency shift was also examined through single factor ANOVA computations to determine the effect of a phonatory response on AER latency. Pearson Product-Moment Correlation Coefficients were calculated to examine the relationship between VRT and AER latency. A summary of these statistical treatments relative to the five hypotheses follows:

Hypothesis 1

The stuttering group will be slower than the nonstuttering group in initiating phonation to an auditory stimulus.

The stutterers in the present study were statistically significantly slower than the nonstutterers in voice reaction time, supporting the hypothesis. Additionally, the relationship between voice reaction time and stuttering severity was examined post-hoc. There was not a statistically significant relationship between VRT and severity of stuttering.

Hypothesis 2

Stutterers will exhibit longer N1 and P2 AER latencies to a pure tone stimulus in both the AER-LO and AER-VR conditions.

The stuttering group in the present study did not exhibit significantly longer AER latencies in either condition. There was only one condition in which a statistically significant difference was present between groups. This was for the AER N1 response in the listening-only condition, and in this condition the stutterers had significantly shorter N1 responses than nonstutterers. The results of the present study do not support the hypothesis that stutterers have less efficient auditory processing than nonstutterers, at least as indicated by N1 and P2 latency measures..

Hypothesis 3

There will be a relationship between auditory processing and voice reaction time. Subjects with later occurring N1 and P2 responses will display longer VRT than subjects with earlier occurring AER components.

There was only one statistically significant correlation when the VRT measures and the various AER measures for all subjects were compared. This was for VRT with the P2 component during the VRT condition. The correlation coefficient was 0.39, indicating that the relationship was not robust. When the measures were compared by groups there were no statistically significant correlations for the stuttering group. The results of the

present study do not support the hypothesis that voice reaction time is related to auditory processing efficiency.

Hypothesis 4

The addition of a phonatory response requirement will increase auditory processing as indicated by an increase in N1 and P2 latency over that recorded in a passive listening situation.

There was not a statistically significant difference in N1 latency between the two conditions. The addition of the phonatory response requirement did not affect N1 latency. There was a statistically significant increase in P2 latency in the VRT condition over that in the L0 condition. The majority of preparatory phonatory activity in a VRT task would likely be occurring in the time interval after normal N1 occurrence and around the time of normal P2 occurrence. This result supports the hypothesis that auditory processing may be affected as the amount of neural demands increase.

Hypothesis 5

The effect of the additional neural demands of phonation will have a greater effect on the stutterers than on the nonstutterers.

There were no statistically significant differences between stutterers and nonstutterers in the amount of latency shift observed between the listening-only and

phonatory response conditions. The results of the present study do not support the hypothesis that stutterers are affected more than nonstutterers by the presence of added neural demands.

The results of this study indicate that stutterers require a significantly longer time than nonstutterers to initiate phonation to an auditory stimulus. As stutterers displayed a significantly shorter N1 response latency to an auditory stimulus in the listening-only condition, and no significant difference was observed between stutterers and nonstutterers for the P2 response latency in the listening-only condition, nor for either AER component in the voice reaction condition, it would appear that slower initial cortical-level auditory processing of the stimulus by stutterers would not be a feasible explanation for the VRT discrepancy. This conclusion is further supported by other results from the present study. No relationship between voice reaction time and the AER latencies in either condition was observed for the stuttering subjects. While the addition of a phonatory task did increase the latency of one of the AER components (P2) as hypothesized, there was not a significant difference between stutterers and nonstutterers in the amount of this increase. Relative to the type of auditory system activity examined, stutterers responded much the same as nonstutterers.

The results of the present study suggest that the slower VRT response in the stuttering group observed in the present study and in previous studies apparently arises from factors other than the auditory system. Such factors may be deficiencies or errors in motor program retrieval, motor programming, central-to-peripheral efferent motor transmission, or in abnormal laryngeal position or activity. Additionally such non-neurophysiologically based factors such as learned compensatory laryngeal patterns, hesitancy, or psychological factors may contribute to the VRT discrepancy between the groups.

While the results do not support the original contention that the observed difference in voice reaction time between the groups is attributable to inefficient auditory processing, one significant finding in the present study does have implications for the role of central auditory impairments in stuttering. As was hypothesized, the additional demands of the rapid phonatory production in the VRT condition significantly increased the latency of the P2 AER component. This hypothesis was developed from prior reports that indicated that the latency of some of the endogenous AER components increased as neural processing demands increased (Ritter, 1978; Teuting, 1978). While no difference between stutterers and nonstutterers in the amount of P2 latency

shift was observed in the present study, a relatively simple processing task and a relatively simple phonatory task was involved. There are indications in the literature that stutterers may possess a less efficient neurological system (e.g., the central auditory processing research such as that of Curry and Gregory, 1969; Hall and Jerger, 1978), or utilize a different neurological pattern (e.g., Moore and Haynes, 1980). If this is true, the shift in neural processing/neural efficiency observed in the present study may change dramatically when the demands of maintaining a normal conversational level/rate of speech are present. The stutterers with a less efficient or inadequate processing system theoretically would be affected to a much greater degree, and a breakdown in the ongoing flow of speech would not be unexpected. Therefore the significant shift in processing observed in the present study becomes relatively important. Apparently even relatively simple phonatory tasks place observable demands on the neural system. The demands of conversational speech may be excessive for some stuttering subjects.

TABLE OF CONTENTS

CHAPTER	PAGE
I. INTRODUCTION, REVIEW OF THE LITERATURE, AND STATEMENT OF THE PROBLEM	1
Introduction	1
Stutterers and Voice Reaction Time	1
Auditory System and Voice Reaction Time	3
Evoked Potential Recordings and Voice Reaction Time	6
Purpose of the Present Study	8
Review of the Literature	8
Reaction Time Studies	10
Summary of VRT Research	25
Influence of Audition on Speech Production	28
Evaluation of Auditory Function in Stutterers	30
Auditory Evoked Responses	38
Summary	46
Statement of Purpose	47
Hypothesis 1	48
Hypothesis 2	48
Hypothesis 3	49
Hypothesis 4	50
Hypothesis 5	51
II. METHODS	52
Subjects	52
Stutterers	52
Nonstutterers	53
Instrumentation	54
Auditory Stimulus	54
Determination of Phonatory Response	63
Calibration of Instrumentation	65
Procedures	65
Electrode Placement	65
Instructions to Subjects	70
Stimulus Presentation	71
Computer Analysis of Data	72
Computer Data Recording	72
Wave Latency Measurement	73
Reliability Evaluation	76
Reliability of Latency Measures	76
Reliability of VRT Measures	77
III. RESULTS	79
Voice Reaction Time	80
Experimental Question 1	80

CHAPTER

PAGE

Slow AER Latency, AER-LO Condition	85
Experimental Question 2	85
Experimental Question 3	86
Slow AER Latency, AER-VR Condition	88
Experimental Question 4	88
Experimental Question 5	89
VRT/AER Relationships	92
Experimental Question 6	92
AER Latency Shift Between Conditions	97
Experimental Question 7	97
Experimental Question 8	99
AER Latency Shift Between Groups	100
Experimental Question 9	101
Experimental Question 10	101
IV. DISCUSSION	103
Hypothesis 1: Voice Reaction Time	
Differences	106
Difference Between Stutterers and	
Nonstutterers	106
Variability of VRT Measures	107
Increased VRT Measures in the Present Study	109
Overlap in VRT Measures Between Groups	110
Relationship Between VRT Measures and	
Stuttering Severity	110
Hypothesis 2: Slow Auditory Electroencephalic	
Response Latency	112
N1-LO Discrepancy	114
Lack of Significant Differences in	
AER Latencies	118
P2-VR Recording Difficulties	124
Hypothesis 3: VRT/AER Relationships	127
VRT with N1-LO Latency	128
VRT with P2-VR Latency	129
Hypothesis 4: Increase in AER Latency Between	
Conditions	130
N1 Latency Shift Between Conditions	131
P2 Latency Shift Between Conditions	132
Hypothesis 5: AER Latency Shift Between	
Groups	137
N1 Latency Shift Between Groups	137
P2 Latency Shift Between Groups	138
V. SUMMARY AND CONCLUSIONS	140
Summary	140
Hypothesis 1	141
Hypothesis 2	142
Hypothesis 3	143
Hypothesis 4	143

CHAPTER	PAGE
Hypothesis 5	144
Conclusions	144
Implications for Further Research	147
Auditory Processing and Stuttering	147
Voice Reaction Time and Stuttering	148
Differential Diagnosis of Etiological Components	149
Neurological Demands and Stuttering	150
Response Variability and Stuttering	151
BIBLIOGRAPHY	152
APPENDICES	161
APPENDIX A. STATEMENT OF INFORMED CONSENT	162
APPENDIX B. CASE HISTORY QUESTIONS	163
APPENDIX C. ORAL MECHANISM EVALUATION FORM	164
APPENDIX D. TABLE D-1	165
APPENDIX E. TABLE E-1	166
VITA	167

LIST OF TABLES

TABLE		PAGE
1.	Mean, Range, and Standard Deviation Values in Milliseconds for Stutterers and Nonstutterers on Voice Reaction Time (VRT), N1 and P2 Latency for Listening-only (LO) and Voice Reaction (VR) Conditions, and N1 and P2 Latency Shift (LS)	83
2.	Summary of Single Factor Analysis of Variance Between Stutterers and Nonstutterers for the Variables Voice Reaction Time (VRT), N1 and P2 Latency in the Listening-only (LO) and Voice Reaction (VR) Conditions, and N1 and P2 Latency Shift.	84
3.	Correlation Coefficients for the Variables Voice Reaction Time (VRT) and N1 and P2 Latency for the Listening-only (LO) and Voice Reaction (VR) Conditions. Coefficients are Provided for all Subjects Combined and Subjects by Groups.	93
D-1.	Mean Voice Reaction Time (VRT) Values, and Range and Standard Deviation Values for Individual Subjects in the Present Study.	165
E-1.	Mean Values (in Milliseconds) of the Auditory Electroencephalic Responses N1 and P2 and Amount of Latency Shift (LS) between Listening-Only (LO) and Voice Reaction (VR) Conditions for Each Subject in the Present Study.	166

LIST OF FIGURES

FIGURE	PAGE
1. Schematic Representation of the Four Classes of Auditory Electroencephalic Responses. . . .	39
2. Block Diagram of the Instrumentation Employed in Recording the Stimulus Tape.	61
3. Illustration of the Timing Sequence of Computer Trigger Tone and Auditory Stimulus Tone Utilized in Recording the Stimulus Tape. . . .	64
4. Instrumentation Arrangement for Intensity Calibration of the Auditory Stimulus Tone and the Trigger Pulse.	66
5. Instrumentation for Recording Voice Reaction Time and Auditory Electroencephalic Responses. .	67
6. Illustration of Electrode Placement for Recording Auditory Electroencephalic Responses from sites Cz, C3, C4, P3, P4, and for Monitoring Eye Movement.	69
7. Illustration of Summated Waveform Recorded from Site Cz (Vertex) During the Listening-only Condition (Nonstuttering Subject).	74
8. Scatter Plot Illustrating the Overlap of Mean Voice Reaction Time Values for Individual Stutterers and Nonstutterers.	82
9. Scatter Plot Illustrating Latency Functions for N1 and P2 Responses by Individual Subjects During the Listening-only Condition.	87
10. Scatter Plot Illustrating Latency Functions for N1 and P2 Responses by Individual Subjects During the Voice Reaction Condition.	90
11. Illustration of Voice Reaction Time and Auditory Electroencephalic Response Relationships for Individual Stuttering Subjects During Listening-only and Voice Reaction Conditions. .	95
12. Illustration of Voice Reaction Time and Auditory Electroencephalic Response Relationships for Individual Stuttering Subjects During Listening-only and Voice Reaction Conditions. .	96

LIST OF ABBREVIATIONS

ABBREVIATION	DEFINITION
AER	Auditory Electroencephalic Response
LO	Listening-only Condition
LRT	Laryngeal Reaction Time
ms	Milliseconds
N1	Auditory Electroencephalic Response N100
NRT	Neural Reaction Time
P2	Auditory Electroencephalic Response P200
VOT	Voice Onset Time
VR	Voice Reaction Condition
VRT	Voice Reaction Time

CHAPTER I

INTRODUCTION, REVIEW OF THE LITERATURE, AND STATEMENT OF THE PROBLEM

Introduction

Stutterers and Voice Reaction Time

A considerable amount of recent research activity in stuttering has dealt with voice reaction time (VRT). In the simple voice reaction time paradigm a subject is instructed to begin phonating a specified sound as rapidly as possible upon perceiving some previously specified stimulus or stimulus parameter. Within such a paradigm stutterers as a group have been reported as consistently exhibiting statistically significant slower phonation initiation time to an auditory stimulus than nonstutterers (Adams and Hayden, 1976; Cross and Luper, 1979; Reich, Till, and Goldsmith, 1981).

The voice reaction time procedure originally was utilized to explore the possibility of aberrant laryngeal function in stutterers. Once the presence of this slower phonatory initiation time had been identified in stutterers, research emphasis switched to analyzing the effect of such factors as age, presence of other speech or language difficulties, and alterations in stimulus and

response modalities and parameters on reaction time measures. Such research had as its focal point the explanation of why such a reaction time deficit was observed for stutterers (McFarlane and Prins, 1978; Cross and Luper, 1979; Cullinan and Springer, 1980; and Reich, Till, and Goldsmith, 1981).

Several investigators have utilized stimulus modalities other than the auditory mode to determine if the response deficiency is modality-specific. Starkweather, Hirschmann, and Tannenbaum (1976) reported that stutterers exhibited the same significantly slower VRT pattern to visual stimuli (a light flash). The authors speculated that their results implied the presence of a more general neurologic processing or response deficiency. Later studies (McFarlane and Prins, 1978; Prosek, Montgomery, Walden, and Schwartz, 1979) did not find that responses to visual stimuli yielded statistically significant differences in VRT between stutterers and nonstutterers. The majority of studies reporting significant VRT differences have utilized an auditory stimulus.

Other investigators have centered their attention on the response modality to determine if the VRT discrepancy is simply an indication of an overall neurological reaction time deficit. McFarlane and Prins (1978) attempted to remove the laryngeal response factor from

their design by monitoring as their response electromyographic (EMG) activity from the upper lip during phonation of a nonsense syllable, which they termed "neural reaction time" (NRT). They also utilized several stimulus modalities and examined reaction time for a simple lip movement in an attempt to remove speech context from their design. Statistically significant differences in neural reaction time between stutterers and nonstutterers were observed only for the verbal response, and only to an auditory stimulus. Other researchers (Prosek, Montgomery, Walden, and Schwartz, 1979; Reich, Till, and Goldsmith, 1981) examined reaction time for finger movements such as button pushing (manual reaction time). Neither group reported statistically significant differences between stutterers and normal speakers on these measures and concluded that the reaction time differences observed in other studies reflected only speech related reaction time differences.

Auditory System and Voice Reaction Time

Indications from VRT research that stutterers may have a neurologically-based difficulty in initiating phonation have implications for evaluating and treating the disorder. Inconsistencies in reported VRT research, however, indicate the need for further exploration into voice reaction time differences observed between

stutterers and normal speakers. While stutterers consistently have been reported as exhibiting statistically significant slower VRT, the reason remains obscure. Such a reaction time discrepancy could be explained on the basis of deficits in any of the myriad neurologic processes required to make the response (e.g., auditory processing, stimulus interpretation, response formulation, motoric programming, or peripheral transmission). Several VRT studies (Adams and Hayden, 1976; McFarlane and Prins, 1978; Cross and Luper, 1979; Reich, Till, and Goldsmith, 1981) have implicated the auditory system as providing a possible source, as reaction time differences are most often observed to an auditory, rather than some other modality stimulus. This hypothesis is strengthened by other types of research that have also demonstrated auditory system deficiencies in stutterers (e.g., Curry and Gregory, 1969; Hall and Jerger, 1978; Toscher and Rupp, 1978; Molt and Guilford, 1979; Decker, Healey, and Howe, 1982).

The possibility that auditory system differences may contribute to stuttering has been proposed for several decades. Early reported research in auditory function of stutterers was in conjunction with the cerebral dominance theory of stuttering, with Tomatis (1954) speculating that a unilateral hearing loss might affect normal dominance acquisition patterns for language, resulting in a mixed

dominance in stutterers. While Tomatis's theory did not become accepted, a resurgence of interest in the relation between auditory problems and stuttering came about from the report that altering the auditory feedback of stutterers' own voices through delayed auditory feedback (Goldiamond, 1965) or masking (Cherry and Sayers, 1956) often resulted in fluent speech. The possible relationship between auditory system function and stuttering was further supported by the earlier report of Lee (1951) who observed that delaying the auditory feedback of a normal speaker's voice to his ears by a slight increment resulted in stuttering-like behavior.

Studies examining peripheral hearing acuity have generally reported no statistically significant differences between stutterers and normal speakers (Van Riper, 1971). Studies of retrocochlear auditory processes, however, have often revealed subtle but statistically significant differences between these two groups. Curry and Gregory (1969) reported finding a smaller right ear advantage for stutterers than for nonstutterers using a dichotic listening task. Results on a subtest of the Synthetic Sentence Identification test (Speaks and Jerger, 1965) consistently have indicated possible brainstem auditory processing inefficiency in stutterers (Hall and Jerger, 1978; Toscher and Rupp, 1978; Molt and Guilford, 1979). Results of other central

auditory tests have been equivocal. Auditory brainstem responses elicited by evoked potential recording techniques have not demonstrated statistically significant differences in brainstem response latency between stutterers and nonstutterers, although a greater degree of variability in response has been reported for stutterers (Decker, Healey, and Howe, 1982).

Evoked Potential Recordings and Voice Reaction Time

Auditory evoked potential recordings appear promising for directly investigating auditory system activity within a VRT design. In evoked potential recordings the ongoing electrical activity of the brain is recorded during a specified time period preceding, during, and following a stimulus presentation. When these recordings are made for large numbers of stimulus presentations, computer averaging techniques summing the values recorded from the many trials can be utilized to filter out the majority of brain activity extraneous to the response to the stimulus.

Auditory electroencephalic response (AER) recording techniques, such as explained above, have fostered analysis of auditory system responses as incoming stimuli travel along the neurologic pathways. Davis (1976) listed four such categories of AER responses: (1) early or brainstem evoked responses, a series of seven distinctive

waves occurring in the first 10 ms following stimulus onset; (2) middle components, including the first cortical response; (3) slow components, in the interval 75 to 250 ms following stimulus onset and including N100 and P200, early cortical responses thought to reflect selective processing of stimulus parameters (Hillyard and Picton, 1978); and (4) the late potentials, P300 and the contingent negative variation, that are thought to reflect information processing and decision-making.

As was previously mentioned, the methodology for recording AER responses would fit within the VRT paradigm. Centering the AER recording interval around the auditory signal used as the reaction time stimulus would allow recording of auditory system response latency for various levels of the auditory system. Such procedure could provide vital information of slower auditory system response latencies contributing to the observed difference in voice reaction time. Brainstem auditory responses were examined by Decker, Healey, and Howe (1982), who reported no statistically significant difference between stutterers and nonstutterers in mean response latency for the various waves. Little information is available on latencies of the later-occurring responses for the stuttering population. The present study examined the slow cortical auditory responses N1 and P2, as they apparently are the

most readily observable of the initial cortical-level responses to the stimulus (Davis, 1976).

Purpose of the Present Study

The purpose of the present study was to further examine auditory system activity of stutterers as a possible explanation of the slower voice reaction time responses observed for stutterers. To do this, the latency of the slow cortical AER responses N1 (N100) and P2 (P200) to the stimulus tone utilized for eliciting the VRT phonatory response was compared between stutterers and nonstutterers. AER latency to the stimulus tone was determined in two conditions, one in which a phonatory response was required (AER-VRT) and one in which the subjects listened to the stimulus tone without providing a phonatory response. The listening-only condition (AER-LO) was utilized to obtain normative data on the slow AER responses of stutterers and to provide a way for determining if the latency of the AER responses was affected by the additional neural requirements of preparing for a phonatory response.

Review Of The Literature

A resurgence of interest in the possibility of a physiological basis for stuttering has been observed within the last decade. Initiated by Wingate's (1969,

1970) implication of a possible phonatory role in the etiology of stuttering, a considerable amount of recent research activity has been concerned with laryngeal and phonatory activity of stutterers. Common examples of methodology in these investigations include electromyographic records (Freeman and Ushijima, 1975, 1978; McLean and Cooper, 1978); spectrographic analysis of voice onset time (Di Simoni, 1974; Agnello, 1975; Hillman and Gilbert, 1977; Metz, Conture, and Caruso, 1979; Starkweather and Meyers, 1979; Hand, 1979); direct laryngeal observation (Conture, McCall, and Brewer, 1977) and voice initiation/voice reaction time measures (Hayden, 1975; Adams and Hayden, 1976; McFarlane and Prins, 1978; Cross and Luper, 1979; Cullinan and Springer, 1980; Watson and Alfonso, 1982). While the methodology across studies has varied widely, the majority of published reports have indicated aberrant phonatory patterns, observed by some even within perceptually fluent utterances.

Perhaps no area has generated as much research interest and demonstrated as consistent an indication of poorer performance by stutterers when compared to nonstutterers as the area of voice initiation/voice reaction time. A brief review of results and conclusions within this area follows.

Reaction Time Studies

A reaction time experiment typically consists of a condition in which the subject is instructed to initiate (or terminate) a specific behavioral response or set of responses upon presentation of a predetermined stimulus. The response (or set of responses) can be triggered to either onset or offset of the cueing stimulus, with the response varying greatly in complexity requirements across designs. Complex reaction time experiments often involve the differentiation of the target stimulus from similar stimuli and/or a choice of responses dependent upon the particular cueing stimulus presented. Simple reaction time designs eliminate differentiation and choice and their respective required processing time, in that only one stimulus is presented and one response is required. Since the subject is aware of both, he is able to "preorganize" his response, yielding a measure of the minimal time required for the simplest sensory, integrative, and execution components of the nervous system. Reaction time (RT) is then defined as the time interval between the presentation of a cue stimulus and the onset of a subject's response (Kofeld, 1969).

Various response types (e.g., manual, verbal) to various stimuli (e.g., visual, auditory) have been utilized in investigating reaction time differences between stutterers and nonstutterers. Predominating these

studies have been investigations into voice reaction time (VRT), often interchangeable in terminology across studies with voice initiation time (VIT).

Hayden (1975) examined "speech initiation time" for which stutterers and normal speakers were instructed to initiate the oral reading of a sentence as soon as possible after hearing the onset of a 1000 Hertz (Hz) tone. Ten sentences were constructed so that the first word in each began with the syllabic /*ɑ*/ (e.g., "almonds are nuts"). Hayden reported that stutterers were statistically significantly slower than the nonstutterers in initiating phonation in running speech.

Speculating that the stutterer's slowness in initiating speech may be dependent on the presence of stuttering, Adams and Hayden (1976) sought to simplify the motoric response required in Hayden's original study, thus reducing the probability of the subject stuttering during production. Ten stuttering and ten normal speaking adult subjects were required to initiate the production of /*ɑ*/ following the onset of a 1000 Hz tone (VIT), sustain phonation, and then terminate production (voice termination time; VTT) as soon as possible upon tone offset. Tones were presented in "trains" of three tones and each subject participated in three trains. Reaction time measures for both initiation and termination of phonation were averaged across each train. A significant

practice effect was noted, as VIT and VTT measures for both groups decreased from train to train. Stutterers were significantly slower than nonstutterers in both initiating and terminating phonation for initial and final trains. The authors noted that VITs and VTTs continued decreasing through the end of the third train, indicating that optimum VRT performance had not yet been obtained.

In an attempt to determine if the increased voice reaction time reported for stutterers was related to auditory processing, as had been theorized by several authors, Starkweather, Hirschmann, and Tannenbaum (1976) examined VRT to a visual stimulus. A series of 26 different CV syllables, including phonemes representing a broad range of place and manner of production, were presented on a screen in front of the subjects. Subjects were instructed to produce the syllable orally as quickly as possible upon presentation of a light flash. Initiation of phonation was detected via a contact microphone, and the resulting signal was low-pass filtered to remove the majority of supraglottal sounds. As in other studies a significant practice effect was noted. Stutterers as a group were slower than nonstutterers (the difference was statistically significant) in initiating phonation and this slower response encompassed a broad range of syllable types. The mean latency difference in VRT between groups for the production of each syllable was

65 ms. This slower VRT to visual stimuli was proposed as indicating that causal factors for slower VRT in stutterers could not be attributed primarily to a deviation of auditory function; they suggested instead a more central dysfunction.

McFarlane and Prins (1978) attempted to remove the process of phonation from their reaction time measures by measuring "neural response time" (NRT). Their adult stuttering and nonstuttering subjects verbally produced /pæ/, /bæ/, or a nonverbal motor movement control task (lip closure) to visual (light flash) and auditory (1000 Hz tone to right or left ear) stimuli offset. Neural response time was defined as the time interval between stimulus offset and onset of electromyographic activity recorded from the orbicularis oris superior muscle (upper lip closure). As evidence at that time implicated aberrant laryngeal activity to stuttering (Schwartz, 1976; Freeman and Ushijima, 1975; Perkins, Rudas, Johnson, and Bell, 1976), McFarlane and Prins' design essentially removed phonation from the reaction time paradigm. Slower mean measures for the stutterers as a group were obtained on overall NRT across all stimulus modes and response tasks. A statistically significant difference between stutterers and nonstutterers was observed, however, only for NRT to auditory stimuli (30 ms mean latency difference between the two groups). The authors noted that

considerable overlap was observed between performances of individual stutterers and normal speakers. The NRTs of some of the stutterers were faster than the NRTs of some of the nonstutterers, indicating that a single factor distinguishing normal speaker and stutterer was unlikely. McFarlane and Prins theorized that excessive error in the timing of speech events is one of the factors that predisposes breaks in the speech flow to which speakers could learn to attend and react to in a manner that comes to be called stuttering. In contrast to Starkweather, et al's. (1976) interpretation of response time to visual stimuli, McFarlane and Prins attributed the difference in reaction time between auditory and visual stimuli to a reduction of task complexity on the verbal task and the extensive pretest practice allowed in their study.

Early VRT research (Hayden, 1975; Adams and Hayden, 1976) had utilized small numbers of responses and reported significant practice effects occurring across all trials, with stutterers' VRTs more closely approximating those of nonstutterers as trials continued. Cross, Shadden, and Luper (1979) investigated VRT in adult stutterers and normal speakers during 100 trials, in sets of 20 each, seeking an "optimum" VRT for between-group comparisons. They utilized the onset of a 4000 Hz tone as the auditory stimulus and also examined the data for performance differences between right and left ear presentations. The

investigators reported that stutterers exhibited longer VRT (mean latency difference was 79 ms) than nonstutterers for all presentation sets even when the subjects were responding at apparently a fully adapted or optimal VRT. As had been previously noted by McFarlane and Prins (1978), no statistically significant differences were observed between right and left ear presentations. Also of interest was the considerably greater variability of stutterers' performances, with almost a three times greater overall standard deviation as compared to nonstutterers.

Adult stutterers and normal speakers served as subjects for these initial VRT investigations, leaving unanswered the question of whether the longer VRTs observed for stutterers resulted from the development of the stuttering problem with age or contributed as a disruptive factor in the early development of fluent speech. To obtain information relative to this question, Cross and Luper (1979), examined VRT in stutterers and normal speakers as a function of age. They utilized 27 stutterers and 27 nonstutterers in groups of nine at each of three age levels of five years, nine years, and adult (15 years of age or older). Subjects initiated the / $\wedge$ / sound in response to the onset of a 1000 Hz tone. A practice set was provided, followed by 55 VRT presentations in five sets of 11 each. The initial

response of each set was disregarded in statistical analysis of the data to reduce the effect of attentional factors. A statistically significant reduction in mean voice reaction time occurred between the five and nine year old and between five year old and adult subjects in both groups. The VRTs of the stutterers were statistically significantly slower than those of the nonstutterers at each of the three age levels. The difference in mean VRT between stutterers and normal speakers was 79 ms for the five year old group, 50 ms for the nine year old group, and 32 ms for the adult group. Since the stutterers were slower than the nonstutterers at each age level, and since the magnitude of the difference was greatest at the youngest age levels, the authors ruled out learning as the most appropriate explanation for the slower reaction time observed in stutterers. Cross and Luper (1979) theorized that VRT differences between the groups suggested a more basic disruption in the neurophysiological processes, not restricted to speech production alone.

A modification of reaction time presentation methods was utilized by Prosek, Montgomery, Walden, and Schwartz (1979), who measured reaction time in six conditions: manual (push button), voice (Vowel-Consonant word production), and neural (laryngeal area EMG) reaction time

to three different stimuli: visual (light flash), acoustic (1000 Hz tone), and acoustic-speech (VC word). Each task was repeated over five consecutive days, rather than in one single session. No statistically significant differences in manual reaction time (MRT) between their ten adult stutterers and ten normal speakers were observed for any of the stimulus conditions, although both groups displayed slower VRTs for visual than for either acoustic stimuli. Similar to what previous studies had reported, differences closely approximating their selected statistical significance level of 0.05 (their results computed out to the 0.06 level) were noted between stutterers and nonstutterers for reaction time tasks involving a vocal response, with stutterers slower in mean VRT. The longest VRT measure for both groups was noted for the acoustic-speech stimuli, perhaps reflecting the additional processing time required, as this task involved the oral repetition of the VC word heard. While vocal response time was slower for the stutterers, NRT for laryngeal area EMG did not differ appreciably between the two groups for any of the verbal response tasks. It is important to note, however, that the type of EMG arrangement utilized in the study did not allow examination of possible aberrant laryngeal activity that could have delayed acoustically recorded data of vocal activity. The electrode placement did not insure that the

recorded initial activity originated from laryngeal musculature.

Cullinan and Springer (1980) reported results on VIT to an auditory stimulus for stuttering and nonstuttering children that conflict with the results reported by Cross and Luper (1979). The authors used 20 stuttering and 20 nonstuttering children (mean age 7.3 years, range 5.8-11.6) producing vocalization to tone onset and ceasing vocalization (VTT) upon tone offset. The phonatory responses were required in three different stimulus conditions (single tone, tone pairs, and tone pairs with reward-nonreward factor). The authors reported that the stuttering children were significantly slower than the nonstutterers in initiating and terminating phonation. When the stuttering group was subdivided, however, into those children with no other speech or language problems (stuttering-only) and those with other speech and/or language problems such as articulation, language, or learning disabilities (stuttering-plus) only the stuttering-plus group, consisting of eleven children, differed significantly from the nonstuttering children in the two VRT conditions in which reward was not a factor. It is important to note, however, that while the stuttering-only group did not differ significantly from the nonstutterers in either VIT or VTT, all mean VRT values were higher (longer) for the stuttering-only

groups were 59 ms for both speech mode tasks, similar to differences reported in previous studies. The authors theorized that the lack of difference in MRT between groups did not support the presence of an organically based neurologic slowness in stutterers. They proposed instead that their results, which indicated statistically significant differences between the two groups only for tasks requiring a speech response, reflected learned anticipatory fears of phonatory initiation and maladaptive prephonatory sets. The study's use of small group size and small numbers of RT presentations per task (ten), coupled with the fact that VRTs for the nonspeech vocal tasks approached statistical significance allows questioning of their conclusion.

Murphy and Baumgartner (1981) replicated a portion of the Cullinan and Springer study. VIT and VTT for / $\wedge$ / to an auditory stimulus were examined for seven stuttering children reported free of any other speech and/or language problems and eight nonstuttering children. Ages of subjects in the study ranged from four years, six months of age to six years, ten months, with an average age of five years, seven months. No significant differences were noted between groups for either VIT or VTT. The mean VTT data indicated in fact that the stuttering group was actually faster (by nine ms) in terminating phonation than the nonstuttering group. The stuttering children,

however, were slower than the nonstutterers by an average of 47 ms in initiating phonation. The small number of subjects and limited amount of VRT measures taken, considering the wide intrasubject variability reported for initial VRT measures, would conceivably reduce the chance of achieving statistically significant differences between stutterers and nonstutterers even though VIT differences between the two groups approached the magnitude reported in several other studies.

Stuttering is often completely absent or markedly reduced during whispered speech (Van Riper, 1971). This finding has in part prompted speculation that aberrant laryngeal activity may be a causal factor in stuttering. The reports of McFarlane and Prins (1978) and Reich, et al. (1981), that no significant differences in reaction time measures were observed between stutterers and nonstutterers when phonatory activity was removed, provided further indirect support for the theory that aberrant laryngeal function was involved in stuttering. In exploring this concept Venkatigiri (1981) examined VRT for both whispered /a/ and voiced /a/ productions to determine if adult stutterers would differ from nonstutterers in VRT for voiced speech only, or for both voiced and voiceless productions. Only 18 VRT trials were recorded for each task, and only the three fastest reaction time measures were utilized in statistical

computation, possibly as an attempt to deal with the high variability in repeated VRT measures. Stutterers were 23 ms slower on average VRT for production of the voiced /a/ than the nonstutterers, a difference that was not statistically significant. The stutterers as a group demonstrated a mean VRT nine ms faster than that of the normal speakers for the whispered production. Of further note was Venkatigiri's observation that VRT for the phonated production was significantly slower than for the whispered production in the stutterers, while the nonstuttering group took approximately the same amount of time to initiate both voiced and whispered /a/. Venkatigiri speculated that the result did not eliminate aberrant laryngeal activity as a possible factor in stuttering.

A similar investigative approach to VRT performance in stutterers was reported by Hayden, Jordahl, and Adams (1982). Moving from the assumption that slowness in initiating vocal fold activity may play a role in stuttering, Hayden et al. theorized that recording VRT under known fluency enhancing conditions may result in decreased VRT for stutterers. Their group of ten young adult stutterers was instructed to phonate /a/ upon hearing each of six pure tone pulses. Three conditions were utilized: a control (normal auditory tone presentation), masking (70 dB of white noise continuous

during tone presentation), and pacing (rhythmic tone presentation). For the control and masking conditions the intervals between stimulus tone presentations were randomly varied whereas in the pacing condition the tones were presented in a continuous 2.5 second on, 2.5 second off pattern, allowing the subject to predict when the next tone presentation would occur. VIT was statistically significantly reduced in the pacing condition over either of the other two, with the masking condition ($P=0.07$) approaching the authors' criterion for statistical significance (0.05) over the control. A Lombard effect also was observed in the masking condition. While the fluency enhancing features of paced phonation may explain the reduction of VIT latency, such an occurrence also could be explained on the basis that the rhythmical nature of the stimulus allowed the stutterers to make phonatory response timing adjustments so that their attempts more closely approximated stimulus onset.

In a companion article Hayden, Adams, and Jordahl (1982) reported the results of a similar approach to VRT measurement. The performance of a control group of nonstutterers was compared to that of a group of adult stutterers using a speech-related reaction time task (oral reading of short sentences) to auditory stimuli under pacing and masking conditions. In this investigation of speech initiation time (SIT) the results for the pacing

condition were essentially the same as found in the earlier study. Both groups had slower SITs during the masking than during the control condition. Stutterers were slower in SIT than nonstutterers for all conditions at a level (0.08) approaching the authors' criterion for statistical significance ($p = 0.05$). It is important to note that in both studies an extremely small number of responses were utilized (six per subject per condition in the VIT study, three per subject per condition in the SIT study) and practice attempts were not allowed.

While Hayden et al. (1982) examined the effect of fluency facilitating conditions on VIT, exactly the opposite approach was utilized by Watson and Alfonso (1982), who examined the effect of situations thought to increase the incidence of dysfluency. The authors interpreted Adams and Reis' (1971) study as indicating that the incidence of stuttering (and thereby possibly LRT latency) should increase as speaking tasks moved from whispered vowels to voiced vowels to phrases, a somewhat questionable assumption in light of the fact that the Adams and Reis findings were based on paragraph length material. Watson and Alfonso utilized both auditory and visual stimuli conditions in examining VRT (which they referred to as laryngeal reaction time: LRT) for whispered and voiced isolated vowels and nonsense (short CVC) syllable phrases. They also analyzed voice onset time

spectrographically within the phrase condition. Each of the eight subjects for the control and for the stuttering group produced 20 responses for each condition following a practice session. All were instructed to phonate upon tone or light offset, with the stimulus onset therefore providing a warning stimulus. In the isolated vowel experiment the only statistically significant difference noted was between the auditory and visual signal, with LRT to the visual signal slower for both groups. No statistically significant differences were observed between whispered and voiced isolated vowel LRT. Both groups displayed statistically significantly slower LRTs for the phrase condition over the isolated vowels; however, a statistically significant difference between the groups was not observed for this measure. Similarly no statistically significant between-group differences were noted for VOT. The authors reported that the use of the warning signal may have contributed to the homogeneity of the two groups' responses.

Summary of VRT Research

In examining the results of voice reaction time research several key findings and factors are observable. First, considerable evidence has accumulated that stutterers as a group are statistically significantly slower in initiating phonation for speech tasks,

especially to auditory stimuli. Second, even when non-statistically significant differences have been reported between groups, the stutterers often generally display slower average voice reaction times, often on the same order of magnitude observed in previous studies reporting significant differences. A possible explanation for this is that studies reporting non-significant results generally utilized considerably fewer VRT trials than studies reporting statistically significant differences. Since great intra-subject variability within VRT measures on the same subject is reported during initial VRT measures (Cross, 1978) this variability would hamper the possibility of achieving significance levels in statistical analysis of the data.

Research concerning VRT to visual stimuli provides few definitive answers. Trends are observable, but little evidence has accumulated that will either disprove or prove multisensory modality differences between stutterers and nonstutterers in VRT response latency. Similarly, while stutterers often displayed a tendency toward slower reaction time in non-speech/non-phonatory activities, few statistically significant results have been reported. Another question remains as to the effect of the age of the respondent on VRT differences observed between the two groups, and the additional possibility that the presence of other speech and language problems may bias the results.

While stutterers as a group are statistically significantly slower in initiating phonation in speech related tasks, when reaction time measures for individual subjects are considered the stutterers' performance overlaps that of the normal speakers. Several of the faster responding stutterers fall well within the performance range of the slower responding nonstutterers, and conversely the performance of many normal speakers falls well within the range of the stuttering group. This is a major stumbling block for the use of VRT research in supporting the claim of aberrant laryngeal functioning as a causal factor in stuttering. Cross (1978) has proposed that the greatly expanded range of variation in the VRT performance of stutterers may provide an explanatory function, in that not knowing what to expect in laryngeal timing may be what throws the timing/sequencing of the speech act off target in stutterers.

A primary failing of VRT measures in supporting causal theories is the enormous range of neural activity encompassed within the traditional reaction time paradigm. These traditional approaches provide no information on the timing of specific neural events between the onset of the stimulus and the first recordable EMG or acoustic activity at the peripheral sites (e.g., the larynx, articulatory structures). Thus, the voice reaction time differences observed between stutterers and

normal speakers may arise within any of several (theoretical) speech-related neural activities such as the sensory transmission from the periphery to the cortex, sensory (stimuli) processing, motor plan retrieval, motor plan transfer, subcortical timing/sequencing adjustments, impaired sensory feedback to subcortical structures, or within the cortico-peripheral transmission. New methods which allow further subdivision of neural activity within the reaction time paradigm are needed.

Research into one of the above components of voice reaction time, that of the auditory (sensory input) system has often indicated poorer retrocochlear auditory function within the stuttering population. Speculation that a disordered auditory system may play a role in stuttering behavior is supported by research data indicating that altering specific parameters of the auditory feedback of speech can result in dysfluency in normal speakers (Lee, 1951; Stromsta, 1959).

Influence of Audition on Speech Production

Most models of speech production consider auditory feedback of speech to play an integral part in the speech production process (e.g., Fairbanks, 1954; Mysak, 1960). Lee (1951) demonstrated that when the auditory feedback of ongoing speech was delayed in reaching the speaker's ear by approximately 200-250 ms, the speech production was

interrupted. These interruptions elicited in some normal speakers via delayed auditory feedback (DAF) consist of repetition of syllables and prolongations of sounds (Lee, 1951; Black, 1951), which are basic behaviors observed in stutterers.

Other alterations of feedback signals besides delayed air conduction feedback (as utilized in DAF) can affect speech patterns of normal speakers. Stromsta (1959) was able to produce an involuntary stoppage of phonation in speakers voicing continuous vowels by providing a feedback sidetone which created bilateral distortion of the produced vowel. Spectrographic analysis of speech produced under the influence of this distorted sidetone revealed some formant changes which resembled those found in stuttering. In a more recent study, Stromsta (1972) found that stutterers had a consistently greater inter-aural phase difference between air and bone conducted sinusoidal signals than did nonstutterers for the seven frequencies that he tested. Values for the stutterers were found to be approximately twice as great as those for the nonstutterers for 150, 300, and 1200 Hz stimuli. The mean inter-aural phase difference for the stutterers at 150 Hz (180 ms) approximates the magnitude of delay utilized in DAF experiments to induce phonatory blockage. Stromsta concluded that this relationship along with his earlier findings offered credence to the theory

that disturbance of laryngeal function effected by an anomalous audition-phonation control system could be a causative factor in stuttering.

If distorted feedback plays a role in stuttering, it would then follow that removing auditory feedback by providing loud masking levels would reduce the severity or eliminate dysfluencies entirely in stutterers. Shane (1955) reduced stuttering in her subjects by as much as 83 to 100 percent while using a 95 dB masking noise when they were speaking. Similar results were reported by Cherry and Sayers (1956) using various pure tones and white noise. Stromsta (1958) reported that the amount of stuttering progressively reduced as the level of the masking noise increased. Maraist and Hutton (1957) similarly found that stuttering decreased as white noise intensity increased. Adams and Moore (1972) noted statistically significantly less stuttering and increased vocal intensity when the speech of stutterers was accompanied by a loud masking noise.

Evaluation of Auditory Function in Stutterers

The previously mentioned research has indicated a possible implication that auditory function might affect fluency. Examination of research results comparing the auditory function of stutterers with that of nonstutterers, however, has indicated few consistent

differences between the two groups except for the area of central auditory processes.

The earliest research into auditory function examined the workings of the peripheral auditory system. For the most part statistically significant differences in pure tone auditory thresholds were not observed between stutterers and nonstutterers. A report of reduced unilateral pure tone threshold sensitivity in stutterers, however, was presented by Tomatis (1954). He theorized that this unilateral, generally right ear, loss forced stutterers to monitor speech with the more auditorily sensitive left ear (ipsilateral to the dominant hemisphere for speech and language). Because of the primacy of crossed pathways the acoustic speech stimuli went initially to the right hemisphere and then had to be shunted back to the left hemisphere for processing. This theoretically resulted in a delay in arrival of the speech signal, creating a condition similar to that produced by delayed auditory feedback when the person was speaking. Later research did not hold this unilateral loss condition in stutterers to be consistent (Van Riper, 1971).

While no differences in peripheral auditory function have been demonstrated between stutterers and nonstutterers, research examining central auditory function, while inconclusive, has consistently produced indications of poorer auditory processing efficiency in

stutterers. Evaluation of relatively basic central auditory processes, such as those required for performance-intensity (speech discrimination testing) function demonstrates little evidence of aberrant auditory function in stutterers. Central tests that require more complex processing, such as under more adverse listening conditions, have demonstrated poorer performance by stutterers. An example of these are the dichotic listening tasks.

In a dichotic listening task paradigm, competing (different) acoustic signals are presented simultaneously to the listener, one signal to each ear. Normal speakers generally have better scores on language-based dichotic listening tasks for the ear that is contralateral to their dominant hemisphere for controlling language. Considerable research is available demonstrating that for normal speakers right ear scores are greater when competing verbal material is fed simultaneously to both ears (Kimura, 1961, 1963, 1967; Carr, 1969). Curry and Gregory (1969) found that 11 of 20 stutterers had higher left ear scores on a dichotic listening task as compared to only five of 20 normal speakers in the control group. Statistically significant smaller differences in scores between left and right ears were found for the stuttering group. There were no differences between groups in performance on non-verbal tasks. Their findings led the

authors to speculate that there is less defined cerebral dominance for language in stutterers. Subsequent research utilizing the basic dichotic listening technique has been less definitive, with generally overall equivocal results (Bloodstein, 1981).

Results of testing with one particular type of dichotic listening task, however, have consistently indicated poorer performance by stutterers. This is the Synthetic Sentence Identification Test (SSI), developed by Speaks and Jerger (1965). Early dichotic test paradigms utilized single nonsense syllables or words as stimuli and competition. The SSI requires the identification of sentence length material against a competing background of conversational speech. Two conditions are utilized: a contralateral competing message (sentence to one ear, competing stimulus to the other) and an ipsilateral competing message (sentence and competing message simultaneously to the same ear). Within each condition various message-to-competition ratios (MCRs) are utilized. Subsequent research with the SSI has indicated that patients with known brainstem lesions perform significantly poorer on the ipsilateral competing message (ICM) condition, while cortically impaired patients demonstrate poorer performance on the contralateral (CCM) subtest (Jerger, 1973; Jerger and Jerger, 1974). Several studies (Hall and Jerger, 1978; Toscher and Rupp, 1978;

Molt and Guilford, 1979; and Larimore and Willeford, 1980) have reported statistically significantly poorer performance by stutterers on the SSI-ICM subtest at the more difficult message-to-competition ratios. No differences are generally reported between stutterers and control groups on the CCM subtest, a test that would appear to pose less demanding processing skills. Hannley and Dorman (1982) reported different results on the SSI-ICM for 20 stutterers who successfully completed a fluency training program. This group of stutterers performed at or above the reported normal listener performance level (Jerger, 1973) on the SSI-ICM. Unfortunately, no control group of nonstutterers was utilized in this study.

Results on other tests of central auditory function have been less definitive. Hall and Jerger (1978) also examined acoustic reflex threshold and amplitude functions. No differences were noted in acoustic reflex threshold levels between stutterers and normal speakers. When acoustic reflex amplitude curves (relating the change in acoustic reflex amplitude to uniform increases in the intensity of the stimulus) were examined for the two groups, stutterers demonstrated a smaller amplitude response at every sensation level above acoustic reflex threshold than did the control group. Hannley and Dorman (1982) investigated acoustic reflex amplitudes and latency

for 19 stutterers and ten normal speakers and reported no differences in either measure between the two groups.

Hall and Jerger (1978) additionally reported a poorer performance by stutterers on the Staggered Spondaic Word Test (SSW) by Katz (1962). The SSW consists of 40 dichotic presentations of bisyllabic words with the final syllable of one word presented simultaneously with the initial syllable of the other word. On this test of temporal lobe auditory function the normal speakers demonstrated a slightly better percentage of correct identification of words presented to the right ear, while the stutterers presented no ear advantage.

Larimore and Willeford (1980) administered nine tests thought to reflect central auditory processes to 13 adult stutterers and two stuttering children. No control group was utilized; differences were judged based on comparison to available normative data. Stutterers scored within normal limits on dichotic sentence identification, masking level difference, acoustic reflex threshold levels, compressed speech identification, and an alternating speech perception test. The stutterers were found to demonstrate poorer than normal performance on the SSI-ICM, filtered speech identification (CVC words low pass filtered at 500 Hz), and binaural fusion of speech (low pass filtered spondaic words to one ear with the same word high pass filtered presented simultaneously to the other

ear). Of these tests, the filtered speech identification is thought to reflect cortical processes; the binaural fusion and SSI-ICM tests are thought to reflect brainstem level auditory processing. Additionally Larimore and Willeford divided their stutterers on the basis of severity of stuttering. The most severe stutterers did not demonstrate a single ear superiority for any of the tasks, while the milder stutterers did manifest single ear superiority on some of the tests.

Performance on dichotic CV monosyllable identification and on masking level difference (MLD) tests was assessed for two groups of stuttering adolescents and a control group by Liebtrau and Daly (1981). The 12 stuttering subjects were divided into an "organic basis" group (three or more positive neuropsychological signs present) and a "functional" group (no compelling evidence of organicity). No statistically significant differences were noted among any of the groups on the dichotic listening task. The masking level difference test consisted of the presentation of a pure tone signal to one ear with a masking noise to the other. In this test baseline threshold measurements for the tone are obtained initially, in which both the tone and the masker are presented in synchronous phase. In the experimental condition, in which the tone and the masker are presented out of phase, normal hearing listeners usually present a

lower threshold for the tone. Shifting the masker out of phase, even though intensity is held constant, results in a release from some of the effects of the masking. The "organic" group of stutterers scored statistically significantly poorer (i.e., smaller reduction of threshold) than either the "functional" group or the normal speakers in the experimental condition. The MLD test is thought to reflect brain stem auditory processes.

The results of the preceding studies on central auditory function in stutterers indicate the possible presence of poorer retrocochlear auditory processing abilities in at least some groups of stutterers. While not definitive, greater evidence has accumulated implicating aberrant brain stem level functioning than cortical, although some studies have reported poorer performance by stutterers on tests thought to reflect cortical processes. A shortcoming of the majority of these central auditory tests is that attentional and motivational factors can significantly affect the results. Since many of the tests require some form of vocal response in stimulus selection and identification, accuracy of responses can be affected by overt and covert stuttering and associated behaviors. Some of the deficiencies could be overcome by tests that examined brainstem and cortical auditory system activity directly, rather than through overt listener physical response.

Such methodology is available through auditory evoked response procedures.

Auditory Evoked Responses

A relatively recently developed electrophysiologic measurement technique allows the obtaining of measurements of auditory system response without requiring an overt physical response on the part of the listener. These measures can be obtained during many subject physiological states. This technique of computer averaging of evoked auditory brainstem and cortical responses allows measurement of neural electrical responses to acoustic stimuli at brainstem, subcortical, and primary, secondary, and associational cortical auditory areas. These responses are normally present but unseen within the ongoing electrical activity of the nervous system, such as when viewed with electroencephalographic (EEG) tracings.

The technique utilizes repeated presentations of an acoustic stimulus with recording of neural electrical responses within a time-locked interval surrounding the stimulus presentation. These multiple recordings of EEG activity are then mathematically averaged via a computer, resulting in the removal of electrical activity extraneous to the response to the stimuli. An example of such an averaged waveform, reflecting the auditory electroencephalic responses (AER), is presented in Figure 1.

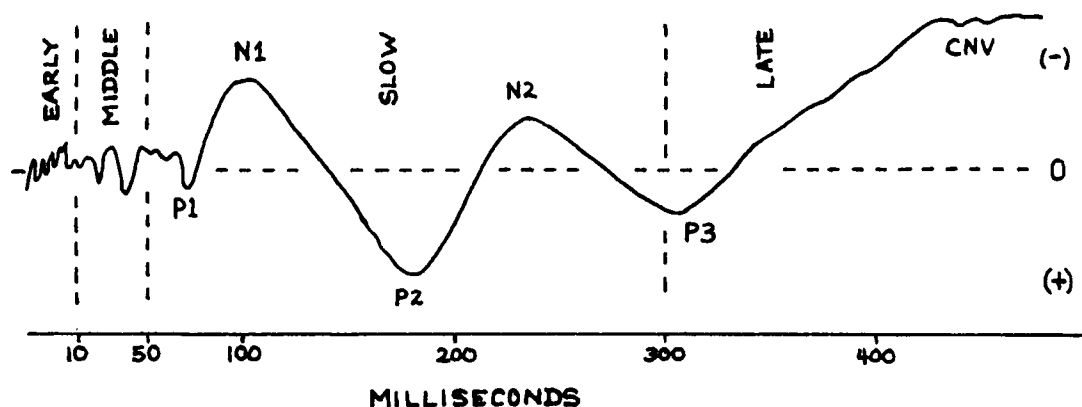


Figure 1. Schematic representation of the four classes of auditory electroencephalic responses.

An adapted version of the terminology used by Davis (1976) will be employed throughout this dissertation to classify the averaged waveforms which are considered to reflect the AER. This adapted set of terms is defined as follows.

Early. Responses occurring within the first one to ten ms following stimulus onset are generally referred to as the early or brain stem electrical responses (BSER) and are thought to reflect activity arising from the eighth cranial nerve and various nucleic sites along the auditory pathway. Six to seven peaks of the waveform can be observed within this period and waves are numbered in order of latency of occurrence (Davis, 1976).

Middle. The components of this auditory evoked electrical response region occur in the time interval of 10 to 50 ms following stimulus onset and are considered as arising from the upper brain stem, subcortical and primary

cortical projection areas. Major peaks of this response interval include a negative peak at approximately 32 ms and a positive peak at around 50 ms (Davis, 1976).

Slow. The slow AER responses occur in the time interval of 50 to 250 ms post-stimulus onset and are considered as evolving from primary cortical projection and secondary associational areas. These are of considerably greater amplitude than the early or middle components. Predominate activity consists of a negative peak occurring about 80 to 100 ms (N1 or N100) and a positive peak (P2 or P200) occurring around 180 to 200 ms after stimulus onset (Tueting, 1978). These slow responses, as with the early and middle responses, are thought to reflect primarily exogenous processing of the incoming signal (Tueting, 1978; McCallum, 1980) although Ritter (1978) reported that N2, a slow response occurring approximately 220-250 ms after stimulus onset, may reflect an early stage of endogenous processing. Donchin (1975) speculated that exogenous processing is involved with identification of specific physical properties of the stimulus such as frequency, intensity, duration, rate, etc. Hillyard and Picton (1978) proposed that N1 may index a cortical process related to an initial selection of a source of information for further processing. Both N1 and P2 seem to be related to stimulus set, i.e., an early stage of selective attention. Endogenous processing

is thought to be largely independent of the physical characteristics of the stimulus and instead depends on the context within which a stimulus is presented and the physiological state of the person tested (Squires and Hecox, 1983).

Late. These last AER components occur from 300 out to 500 ms post-stimulus onset, consisting of P300 (P3) and the contingent negative variation (CNV). P300 is a large amplitude positive peak occurring approximately 300 ms post-stimulus onset. It is thought to arise from secondary auditory associational areas as well as possibly from other cortical processing sites (similar waveforms are observed in other sensory mediums besides auditory) and is seen in relation to variations in a conditioned stimulus. CNV is a slow sustained negative shift of the waveform occurring out to approximately 500 ms post-stimulus and is observed best in tasks requiring a motoric response to differential stimuli. Unlike the early, middle, and majority of slow components, these late responses are considered as reflecting endogenous processing of the stimulus (Davis, 1976; Hillyard and Picton, 1978). Ritter (1978) speculated that endogenous processing involves such features as decision-making, target selection, information delivery, and match-mismatch recognition.

Latency of the later-occurring AER components, such as N2, P300, and CNV, is affected by the amount of neural processing of the stimulus required in the task. For example, P300 has often been examined in reaction time situations. Both latency and amplitude functions for P300 increase as the amount of decision-making/information processing required increases (Teuting, 1978). There are indications that additional slow and late AER components are observed as additional demands are placed on the neural system. A second negative slow component, N2, has been reported to occur during reaction time tasks. This component, occurring shortly after the appearance of the P2 component, is thought to reflect an early level of endogenous processing. As has been reported for P300, latency of the N2 component, as well as latency of the reaction time response, increases as the amount of neural processing required increases (Ritter, 1978). It appears that the latency of some AER components is sensitive to neural activity levels/neural processing demands.

Amplitude and latency function of the early, middle and slow components of the AER are affected by stimulus intensity, duration, and rate variables. BSER components are generally examined in the context of multiple presentations of short duration acoustic clicks, tone bursts, or pips presented at rapid rates. Because of the relatively low amplitude of the responses (less than 1

microvolt) large numbers of presentations (on the order of 1000) are utilized. Mathematical summation of all electrical activity occurring during the trials allows filtering of neural activity extraneous to the auditory response. Slow components (N1, P2) can be elicited to a wider variety of acoustic stimuli. As with BSER, amplitude of the response increases as stimulus amplitude increases. The N1 response appears to be stable for frequencies up to 2000 Hz and then its amplitude drops abruptly for higher frequency stimuli (Picton, Woods, and Proulx, 1978). Optimum stimulus repetition rates appear to be on the order of two to three seconds and clear waveform tracings can be obtained with as few as 32 presentations (Owen and Matsusaka, 1982). Both early and slow components can be obtained with the subject in many physiological states as long as motoric (especially cephalic) movement is kept to a minimum (Davis, 1976).

Exploration of auditory responses of stutterers with AER techniques has been minimal, due perhaps in part to the scarcity and expense of the recording equipment. Decker, Healey, and Howe (1982) examined latency and amplitude functions of the BSER component to monaural and binaural click stimuli in eight adult male stutterers and a matched control group of nonstutterers. Their study followed prior reports of aberrant brain stem auditory function as evidenced by various central auditory tests

(SSI-ICM, acoustic reflex amplitude function, binaural fusion of speech, masking level differences test). No statistically significant difference in BSER amplitude or latency at click rates of five and 12 per second, presented monaurally and binaurally, was observed between the groups. The stutterers, however, demonstrated statistically significant greater variation for waves III, V, and wave I to V interval latency in monaural listening conditions. Using a waveform subtraction procedure, Decker et al. reported that the variance between groups in wave V latency could be explained in terms of a lack of auditory tract preference in the stutterers examined in that portion of the study. The authors interpreted the results as evidence of brain stem level neurological differences in auditory processing for stutterers.

No evidence of published research on latency or amplitude function for the slow (cortical) AER in stutterers was uncovered during the review of the literature for the present study. A study by Zimmerman and Knott (1974) investigated changes in a late AER (CNV) in an attempt to examine both anxiety effects and hemispheric dominance for speech and language between stutterers and nonstutterers. Two verbal and two non-verbal conditions were utilized. In the non-verbal control condition subjects were instructed to press a key following presentation of an imperative stimulus (light

flash on screen) if it was preceded by the appropriate warning stimulus (high or low pitched tone). A verbal/non-speech condition required subjects to silently read a word flashed on a screen (imperative stimulus) and indicate by a key press if they believed they would stutter on the word if they read it aloud. The second verbal condition involved the subject actually reading the word aloud following presentation of the warning and imperative stimulus. No differences in CNV response amplitude were observed between the groups in the non-verbal conditions. Zimmerman and Knott (1974) examined recordings for fluent utterances only and reported no difference in CNV recordings from a scalp vertex electrode between groups. Recordings from electrodes over Broca's area (motor speech area) revealed a greater left hemisphere CNV amplitude in four of the five normal speakers during verbal conditions. Only two of nine stuttering subjects displayed this left hemisphere asymmetry. The authors speculated that this reflected a difference in inferior frontal lobe activity preceding speech and inferred support for a possible lack of hemispheric dominance for speech in stutterers. They reported the lack of significant differences in vertex amplitudes during speech conditions did not support the presence of different anxiety levels to speech between stutterers and nonstutterers.

Summary

Considerable evidence from voice reaction time research methodology indicates that stutterers, as a group, are significantly slower than normal speakers in initiating phonation to an acoustic cue. A shortcoming of the reaction time research design is that many neurological and physiological events occur within the confines of the design, i.e., between the onset of the signal and the recorded phonatory response. Any or all of these events could conceivably contribute to the slower responses observed for stutterers. Although attempts have been made to explore or control some of the processes occurring in the final stages of the response (e.g., triggering from laryngeal or labial EMG rather than an acoustic phonatory response), little research has delved into the receptive processes within the paradigm. Since other forms of investigation have indicated the possible presence of central auditory processing deficiency in stutterers, research examining auditory processes within the context of a voice reaction time paradigm appears warranted. It would appear that the most accurate way of obtaining such information would be the recording of auditory electroencephalic responses occurring to the reaction time stimulus. Some evidence exists indicating slightly more variability for stutterers within the earliest (brain stem level) AER, however no significant

differences in latency of response were noted. Little research exists on latency functions for later (cortical level) AER in stutterers. Additionally, little evidence exists on the effect of increased neural demands, such as those required for a speech (phonatory) response, on AER latency.

Statement of Purpose

The purpose of the present study was to examine cortical auditory system activity of stutterers as a possible explanation of slower voice reaction time responses often reported for stutterers. The latency of the slow cortical AER responses N1 and P2 to the stimulus tone utilized in eliciting the VRT phonatory response was compared between stutterers and nonstutterers. AER latency to the stimulus tone was determined in two conditions, one in which a phonatory response was required (AER-VR) and one in which subjects listened to the stimulus tone without providing a phonatory response (AER-LO). The listening-only condition was utilized to obtain normative data on the latency of slow cortical AER responses of stutterers and to provide a way for determining if the latency of AER responses was affected by the additional neural requirements of preparing for an immediate phonatory response. The hypotheses examined within this study were as follow.

Hypothesis 1

Because several earlier studies have indicated that stutterers are slower than nonstutterers in voice reaction time tasks, it is hypothesized that the stuttering group in the present study will be slower than nonstutterers in initiating phonation to an auditory stimulus.

Question 1. Is there a statistically significant difference between stutterers and nonstutterers in voice reaction time to a pure tone stimulus?

Hypothesis 2

Since there are many indications in the literature that stutterers may possess a less efficient auditory system than nonstutterers, the second hypothesis of the present study is that stutterers will exhibit auditory electroencephalic responses N1 and P2 to a pure tone stimulus that are slower in latency than those of nonstutterers. This slower response for the stutterers, if present as hypothesized, would likely be observed for both passive listening and phonatory response conditions.

Question 2. Is there a statistically significant difference between stutterers and nonstutterers in N1 response latency to a pure tone stimulus during a listening-only task?

Question 3. Is there a statistically significant difference between stutterers and nonstutterers in P2 response latency to a pure tone stimulus during a listening-only task?

Question 4. Is there a statistically significant difference between stutterers and nonstutterers in N1 response latency to the pure tone stimulus when recorded during a voice reaction time task?

Question 5. Is there a statistically significant difference between stutterers and nonstutterers in P2 response latency to the pure tone stimulus when recorded during a voice reaction time task?

Hypothesis 3

The third hypothesis of the present study is that there will be a relationship between AER latency and voice reaction time. This was developed on the basis that if differences in AER latency measures exist between stutterers and nonstutterers, such differences may reflect the use of different processing patterns or less efficient processing systems between the two groups. Such processing differences may be a component involved in reaction time to auditory stimuli.

Question 6. Are there statistically significant correlations between voice reaction time and AER latency, specifically:

- A. between VRT and N1 latency in the AER-LO condition?
- B. between VRT and P2 latency in the AER-LO condition?
- C. between VRT and N1 latency in the AER-VR condition?
- D. between VRT and P2 latency in the AER-VR condition?

Hypothesis 4

The fourth hypothesis of the present study is that AER latency will be slower in the AER-VR condition than in the AER-LO condition. This was developed from prior research indicating that reaction time often increases when a subject is forced to make a choice or decision before responding. This increase in reaction time is thought to reflect added neural processing and decision making. Similarly, latency of the slow AER component (P300) has been reported to increase as neural demands increase. As the phonatory response utilized in the present study requires precise coordination of multiple respiratory, laryngeal, and articulatory muscles, increased neural activity would be expected when compared to a passive listening condition.

Question 7. Is there a statistically significant difference in AER N1 latency between the listening-only and phonatory response condition?

Question 8. Is there a statistically significant difference in AER P2 latency between the listening-only and phonatory response condition?

Hypothesis 5

The fifth hypothesis of the present study is that stutterers will demonstrate a greater degree of AER latency shift between conditions than will the nonstutterers. If the stutterers demonstrate a less efficient neural system (as previously hypothesized), the increased neural demands of the phonatory response condition should affect the stutterers to a greater degree than the nonstutterers.

Question 9. Is there a statistically significant difference between stutterers and nonstutterers in the amount of N1 latency shift between AER-LO and AER-VR conditions?

Question 10. Is there a statistically significant difference between stutterers and nonstutterers in the amount of P2 latency shift between AER-LO and AER-VR conditions?

CHAPTER II

METHODS

Subjects

Stutterers

Eight adult stutterers and eight adult nonstutterers, matched for age and sex, served as subjects in the study. There were five males and three females in each group. Subjects' ages ranged from 19 to 38 years, with a mean of 27.8 years for the stuttering group and 27.1 for the nonstutterers.

Each subject in the stuttering group was selected on the basis of the following criteria.

1. Each subject had been evaluated and diagnosed by a Speech-Language Pathologist holding American Speech-Language-Hearing Association certification as exhibiting stuttering behavior sufficient in frequency and/or severity to warrant diagnosis as a stutterer.

2. Each subject demonstrated a childhood onset of stuttering as determined in a case history (Appendix B) obtained by the examiner. The onset was not linked to physical trauma to the neurologic system, so far as could be ascertained.

3. Subjects exhibited no pathological structural abnormalities of the speech mechanism as determined through an oral-mechanism examination (Appendix C) by the investigator.

4. Subjects exhibited normal voice quality, pitch, and loudness while counting from one to ten and prolonging phonation of the vowel sound / $\wedge$ / for two seconds.

Subjects exhibited bilateral hearing acuity for air conducted pure tone stimuli within the range of normal, as defined by thresholds not in excess of 20 dB HL (ANSI 1969) at 500, 1000, 2000, and 4000 Hz.

Nonstutterers

Each of the eight subjects in the nonstuttering group was selected on the basis of matching a member of the stuttering group in age and sex. The maximum difference in age for matched pairs was two years. Additional criteria for members of the nonstuttering group are listed below.

1. All were identified through case history (Appendix B) information and examiner evaluation as not possessing at the time the study was conducted nor at any other time a speech or language disorder sufficient in degree to warrant therapeutic intervention.

2. Subjects exhibited no pathological structural abnormalities of the speech mechanism as determined

through an oral-mechanism examination (Appendix C) by the investigator.

3. Subjects exhibited normal voice quality, pitch, and loudness while counting from one to ten and prolonging phonation of the / $\wedge$ / vowel sound for two seconds.

4. All exhibited bilateral hearing acuity for air conducted pure tone stimuli within the range of normal as defined by thresholds not in excess of 20 dB HL at 500, 1000, 2000, and 4000 Hz.

Instrumentation

Auditory Stimulus

Nature of the stimulus. The auditory stimulus presented to elicit a vocal response from the subjects was a 1000 Hz pure tone pulse, one second in duration, with a stimulus rise/fall time of ten ms. Seven sets, each consisting of 20 tones and thus yielding a total of 140 stimulus presentations, were presented to each subject during the voice reaction time (VRT) task. An additional five sets of 20 tones were presented after completion of the VRT task to elicit auditory system responses for the listening-only (LO) task. Fewer sets were utilized in the listening-only condition because no motor response was required from the subjects, thus lessening the number of artifactual (rejected) trials. The listening-only

condition was always presented after the VR condition for two reasons: first, to minimize attentional state differences among subjects during the VRT trials that might arise due to the highly repetitive nature of the stimulus and the relaxed body posture required of the listener; and second, to maximize the meaningfulness of the stimulus in the listening-only condition, thus increasing the subjects' attentional state, in order to enhance AER amplitude.

There was a silent pause between the offset of one tone and the onset of the following tone, with the silent intervals consisting of random assignments of five, six, seven, or eight seconds duration. All stimulus tones were recorded at the same intensity level on one channel of a two channel tape recorder. "Triggering" voltage pulses providing information for computer recording, storage, and analysis were recorded on the second channel. These voltage pulses are described later in this chapter.

The first set of twenty tones (set A) was employed as a practice set to ensure that subjects were able to correctly respond in the voice reaction time (VRT) task. This set was not analyzed with the remainder of the data in computing VRT or auditory evoked response (AER) latency. The remaining sets of tones were designated as sets B, C, D, E, F, and G and were utilized for data gathering during the study. Each set was verbally

identified on the tape preceding the initial tone of each set by the phrase "this is set (letter).". There was a three minute rest period provided between the presentation of each set.

A 1000 Hz pure tone signal, recorded at the same (recording) intensity level as the pure tone stimuli used to elicit the AER and VRT responses, served as a 30 second duration calibration signal at the beginning of the stimulus tape. The recorded tape was examined for stimulus intensity equality and silent interval duration on a graphic level recorder prior to initial administration. Signal-to-noise ratios on the tape averaged approximately (+)40 dB throughout the tape recording. The prepared tape met all conditions previously described.

The nature of the stimulus tape signal was determined on the basis of a review of previous VRT research utilizing a simple motor reaction time paradigm and on previous research investigating optimal signal characteristics for eliciting the N1 and P2 components of the slow auditory electroencephalic response. Previous research results have indicated that simple reaction time is affected by such features as the nature and presence of a ready signal (Henriksen and Holmes, 1973), stimulus modality (Kofeld, 1969), stimulus intensity (Kofeld, 1969), and the number of presentations used (Kofeld, 1969;

Cross, Shadden, and Luper, 1979). Factors affecting recording of the slow AER include stimulus frequency, intensity, duration, and repetition rate (Owen and Matsusaka, 1982).

The traditional simple motor reaction time paradigm makes use of a "ready" or "warning" signal that precedes the actual stimulus signal, thereby increasing the subjects' state of readiness and optimizing reaction time performance. The majority of VRT investigations in stuttering have also made use of such a signal or have instructed the subject to respond to the offset of the stimulus tone, providing in effect a ready signal via the onset of the stimulus tone. The present study, however, did not make use of a ready signal because of the nature of one of the responses under investigation (AER). The possibility existed that the use of a warning signal would result in the elicitation of a preceding auditory evoked potential that would hamper identification of the slow AER. This evoked potential, referred to as the contingent negative variation (CNV) is a negative cerebral potential shift occurring in the interval between a warning stimulus and an imperative or significant stimulus, and thus is elicited through a test design closely resembling the traditional simple reaction time paradigm (Zimmermann and Knott, 1974; Davis, 1976).

While a majority of VRT studies have utilized ready signals in their design there is an indication in the literature that the omission of such a signal should not significantly affect VRT results. A study by Henriksen and Holmes (1973) investigated ready signal effects for three different experimental designs of a simple reaction time task. One group of 20 subjects received a visual ready signal prior to each stimulus presentation, a second group received no ready signal, and a third group experienced both ready signal conditions (presence, absence) in a random order. The authors noted that no statistically significant differences in manual reaction time were present when the ready signal was either present or absent for the entire test. When the subject had received ready signals within a test session slower reactions were observed for the non-ready signal trials. The authors concluded that when the subject had never received a ready signal, the preceding response served as a preparatory cue for the following signal. Cross and Luper (1979) obtained measurements of VRT response to an auditory stimulus presented without using a ready signal. VRT latencies obtained closely resembled those reported for previous VRT studies utilizing some form of ready signal.

Both stimulus modality and stimulus intensity have also been demonstrated as affecting motor reaction time. Auditory stimuli generally elicit a faster response than

cues presented visually, possibly attributable to shorter neural transmission latencies for the auditory tract. Minimum reaction time to auditorily presented tones is observed for medium (60 dB SPL) to high intensity (90 dB) stimuli. Most studies have employed auditory stimulus intensities of 80 to 85 dB SPL to insure minimum stimulus effect on reaction time.

Picton, Woods, and Proulx (1978) investigated factors affecting recording of auditory electrical responses. They indicated that the N1 response satiates at intensities of 60 dB HL (hearing level) and may even decrease as 90 dB HL is approached. For this reason the intensity of the stimulus used in the present study was 50 dB SL (sensation level) which calculated as 65 dB HL for the majority of the subjects, still well within the range for optimum reaction time elicitation. This 50 dB SL for each subject was established by presenting the stimulus at an intensity 50 dB above the subject's previously identified 1000 Hz. threshold. The stimulus used also met criteria regarding stimulus frequency (less than 2000 Hz), duration, and repetition rate reported by Picton et al.

The present study utilized a large number of responses to the stimulus. The reason for this was twofold. First, as was noted in the review of the literature, a practice effect is observed for voice reaction time with the speed of the response generally improving over initial trials.

Cross, Shadden, and Luper (1979) demonstrated that some of the fastest VRT measures were observed by the end of the first 20 trials, with stutterers apparently taking longer than normal speakers to achieve optimum VRT. The second reason for the use of a large number of trials involved the recording of the AER responses. It was recommended by Davis, Mast, Yoshie, and Zerlin (1966) that the minimum number of responses necessary in slow AER research should be 32 or more to insure that definitive characteristics of the waveform are present following signal averaging. The use of 120 responses in the VR condition of the present study insured that adequate numbers would be retained even after artifactual trials were deleted.

Stimulus tape preparation. The instrumentation employed for the auditory tape stimulus preparation is illustrated in Figure 2. A Wavetek model 116 tone generator was utilized to produce a 30 second duration 1000 Hz pure tone calibration signal at a tape recorder volume units (Vu) meter level setting of zero on channel one of the tape.

A Wavetek model 116 tone generator provided the 1000 Hz sinusoid used for both trigger and stimulus signal with the frequency accuracy determined using a Data Precision model 5740 frequency counter. The timing of the trigger signal for the computer was regulated by a Grason-Stadler

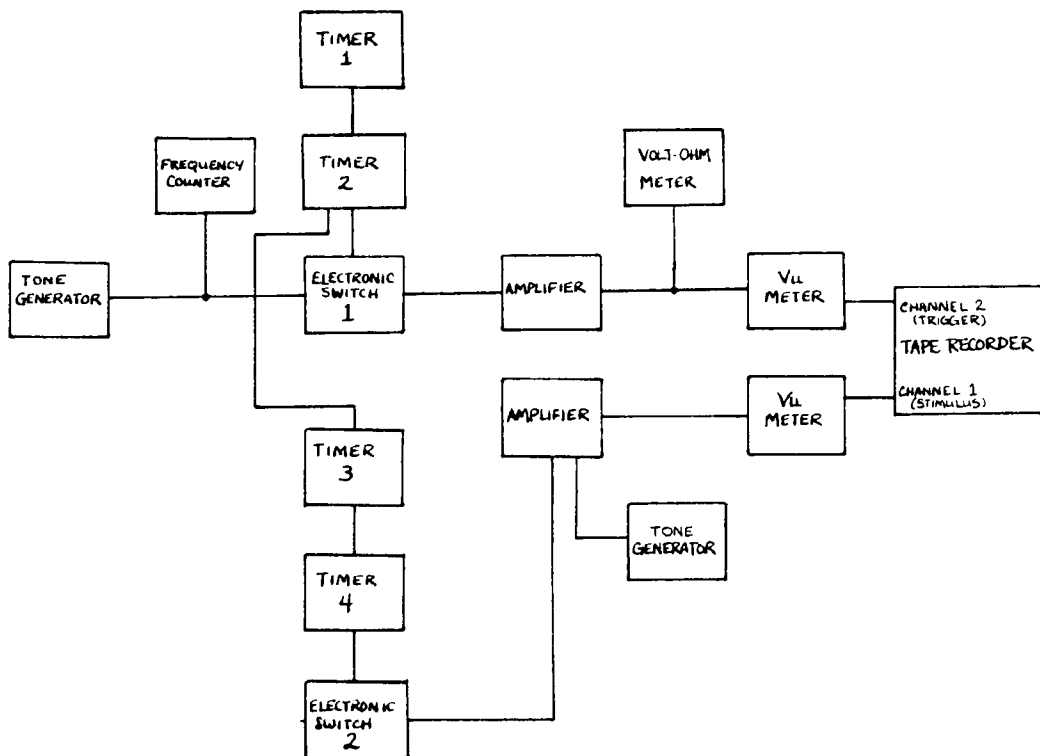


Figure 2. Block diagram of the instrumentation employed in recording the stimulus tape.

(G-S) model 1287B electronic switch (#1). Timer #1 (G-S model 1216A) turned on electronic switch #1 (set to yield a signal rise time of ten ms) allowing passage of the trigger signal. Timer #2 turned off the electronic switch after 250 ms. The signal then went to a G-S model 1288 amplifier for intensity adjustment to a 0.5 volt level at a tape recorder Vu level of zero (the voltage level required to trigger the computer). The signal was recorded on channel two of an Akai model R576 stereo cassette tape recorder.

Timers #3 and #4 regulated electronic switch #2 to provide the stimulus tone. Timer #3 was connected in series to timer #2 and allowed passage of the pure tone signal through the electronic switch 750 ms after trigger signal offset (one second after trigger signal onset). Timer #4 turned off switch #2 after a one second stimulus tone duration. The pure tone stimulus signal from electronic switch #2 passed through a G-S model 1288 amplifier and the tape recorder Vu meter to yield a Vu meter intensity indication of zero. This signal, the auditory stimulus, was recorded on channel one of the tape recorder. The silent intervals of five to eight seconds were placed by the examiner manually selecting time intervals on timer #1, with interval time selected from a random numbers table.

During experimental sessions channel two of the tape (trigger signal) was routed to the analog-to-digital converter of a Digital Corporation PDP-11 computer. Channel one (stimulus signal) was delivered to an amplifier (G-S model 1705 audiometer) and then to headphones. The timing pattern of the trigger and stimulus signals are illustrated in Figure 3. Tone and silent interval duration for the entire tape was checked on a Bruel and Kjaer graphic level recorder, as was stimulus intensity uniformity and noise levels during silent intervals.

Determination of Phonatory Response

The presence of a phonatory (voice) response was determined through placement of a contact microphone on the surface of the skin at the approximate level of the cricoid cartilage. The location was determined through tactile manipulation by the examiner.

The custom built amplifier for the microphone was set to two-thirds of maximum gain and locked. Threshold level for the phonatory response on the contact microphone was determined through trial settings as the level which did not yield a response to swallowing or gross laryngeal movements yet produced a recordable voltage to phonatory responses.

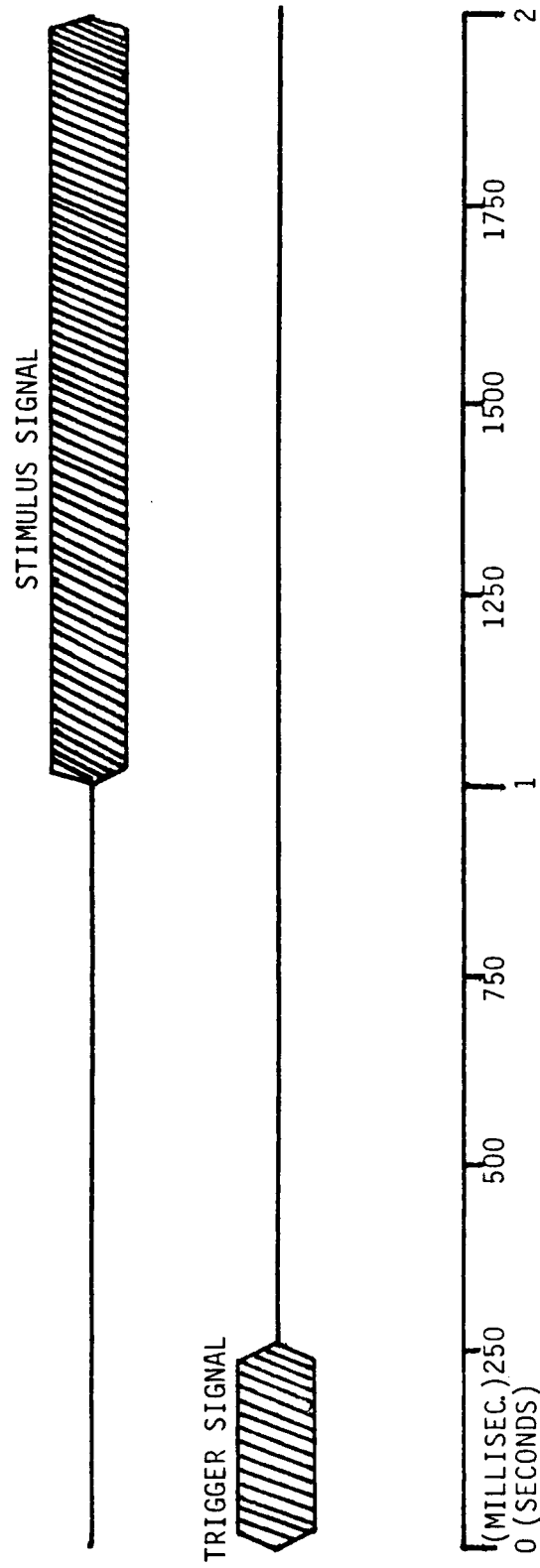


Figure 3. Illustration of the timing sequence of computer trigger tone and auditory stimulus tone utilized in recording the stimulus tape.

Calibration of Instrumentation

Stimulus intensity was controlled via presentation through a G-S model 1705 audiometer. Adjustment of stimulus intensity was accomplished through adjustment of the audiometer stimulus presentation level dial based on each subject's threshold level for a 1000 Hz tone. Calibration of equipment was checked periodically (initially, mid-study, and at termination) during the study with a Bruel and Kjaer (B & K) model 4147 flat plate coupler, B & K model 4152 artificial ear, B & K model 4144 condenser microphone, and B & K model 2203 sound level meter. No variation in stimulus intensity level was observed during calibration checks. Intensity of the trigger signal was checked using a volt-ohm-meter. The calibration instrumentation arrangement is shown in Figure 4.

Procedures

Electrode Placement

Figure 5 illustrates the instrumentation arrangement for the test session. The electrodes and contact microphone were attached to the subject prior to entering the test room. All non-scalp electrode sites were prepared with an acetone scrub and mild abrasion prior to electrode attachment. Scalp electrodes were placed via an electrode cap (resembling a swimming cap in appearance)

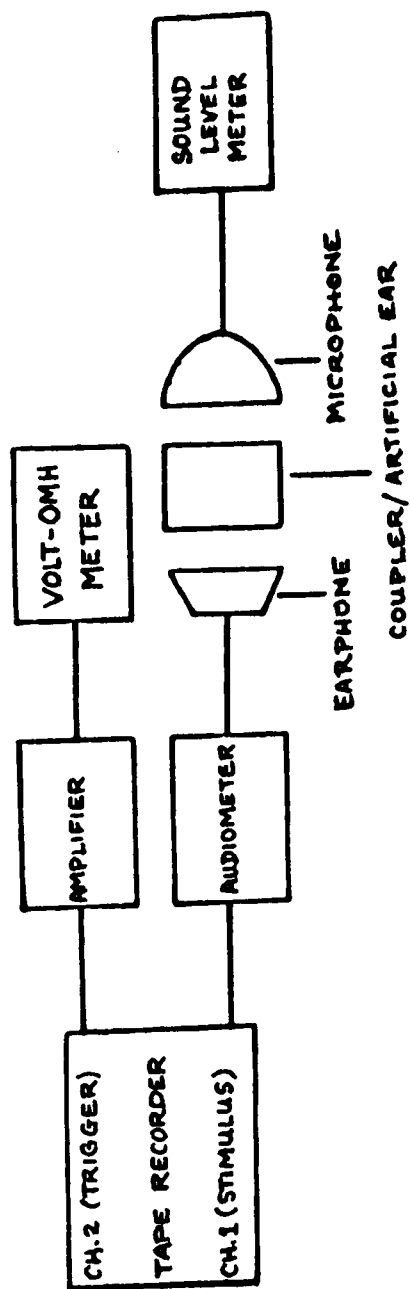


Figure 4. Instrumentation arrangement for intensity calibration of the auditory stimulus tone and the trigger pulse.

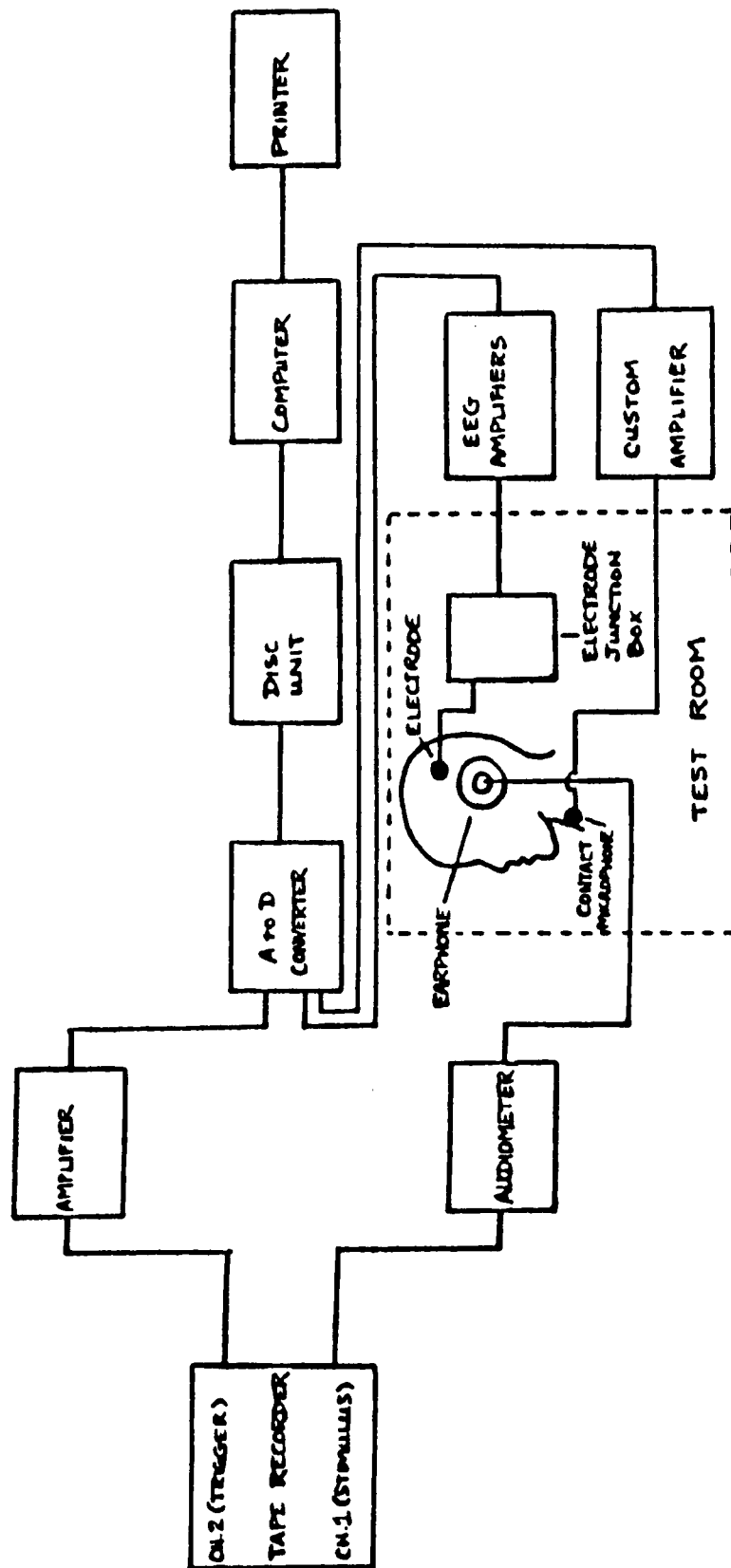


Figure 5. Instrumentation for recording voice reaction time and auditory electroencephalic responses.

designed to agree with the International Federation 10-20 electrode placement system guidelines (Jasper, 1983). Electrolytic gel was inserted into the electrode via a hypodermic syringe once the cap had been placed on the subject. Signals to be analyzed for the present study were recorded from site Cz (vertex). Additional recordings for later analysis were recorded from sites C3, C4, P3, and P4. Data reported in the present study only consist of what was recorded from site Cz. Data from sites C3, C4, P3, and P4 are not included in the results of the present study. Two electrodes were placed on the subject's face to record the electrooculogram (EOG) for artifact detection. This involved placement of one electrode at the lower canthus of the right eye which was referred to another electrode placed above the nasion. All scalp electrodes were referenced to linked ear electrodes. Once the electrodes were applied, their electrical resistance was measured on a Grass electrode impedance meter. Only resistance levels of less than 5000 ohms were accepted. Electrodes not meeting this criterion were reapplied. Contact microphone placement procedures have been previously described. Electrode placement is illustrated in Figure 6.

Testing was conducted in an electrically shielded room with ambient noise levels below 45 dB SPL for the octave bands 31.5 to 16000 Hz, inclusive, as determined on a B &

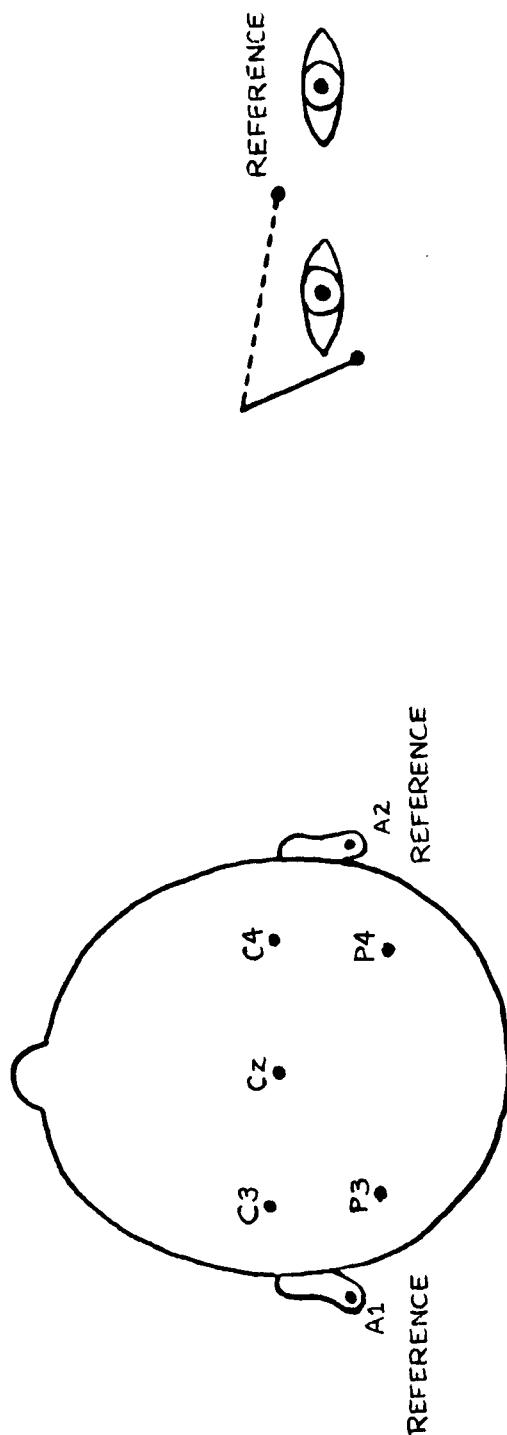


Figure 6. Illustration of electrode placement for recording auditory electroencephalic responses from sites Cz, C3, C4, P3, P4, and for monitoring eye movement.

K type 2203 sound level meter coupled to a B & K type 1613 octave filter set. Subjects were seated in a padded reclining chair set in a semi-upright position. No instrumentation other than electrode connections was present in the test room.

Instructions to Subjects

The consent form, providing instructions and information for subjects, is contained in Appendix A. Subjects were informed that the purpose of the first experimental task was to determine how fast they could initiate phonation of the neutral vowel sound / $\wedge$ / upon hearing the onset of the stimulus tone. This particular vocal response was selected as its production would require a minimum of articulatory movement from a neutral oral position. The importance of maintaining a neutral relaxed body posture during testing and avoidance of any movement other than phonation was stressed. The subjects were informed that there would be an interval immediately after each tone presentation where muscular immobility was not necessary, and that tones would be presented in sets of 20 (yielding a three to four minute test interval) with a three minute rest period between each set. To reduce the likelihood of electromyographic (EMG) signals from facial musculature movements contaminating the data, a bite block, made of a five

millimeter thick closed cell foam pad, was placed between the subject's upper and lower teeth in the region of the first and second molars. This resulted in slightly open jaw posture and reduced jaw movement during phonation. Subjects were instructed to maintain a fixed downward gaze, focusing on the region of the foot pad of the chair.

Stimulus Presentation

The VRT task, presented first to all subjects, began with a set of 20 stimulus tones presented through earphones as a practice set. All subjects demonstrated by proper production that the task was understood and all completed the practice session satisfactorily. The examiner then started the test presentation. Upon completion of the 140 tone practice and test presentations the throat microphone and bite block were removed and the listening-only task was begun. For this procedure the subject was instructed to maintain the relaxed body posture and downward gaze while simply listening to five more sets of tones. The practice set was deleted so that each subject received 100 tone presentations. No physical response was requested during this portion of the test. Set presentation was in the same manner as for the VRT task.

All auditory stimuli were presented through Telephonics TDH-39 headphones with MX41AR auricular

cushions. The examiner observed the subject frequently throughout the test session to check for extraneous or excessive movement. Subjects were reminded after every other set to maintain a relaxed posture and to minimize movement. Artifacts were also monitored via EOG tracings (artifact channel) and computer reporting of successful presentations. The EOG channel to the computer resulted in automatic discarding of any trial with extraneous eye movement or excessive facial EMG activity. This artifact threshold level (30 millivolts) was established during pre-study pilot trials. It appeared to be high enough in sensitivity so that visible eye movements were detected, yet low enough in sensitivity that an excessive number of trials were not rejected when no visible eye activity was present.

Both the VRT and the listening-only task were completed during the same test session for each subject. The approximate time required for each session, including audiometric screening, electrode attachment, testing, and electrode removal was two hours. Approximately one hour of this time was spent in direct task administration.

Computer Analysis Of Data

Computer Data Recording

The onset of the trigger signal of the stimulus tape started the storage of data on the computer. The

computer, a Digital Corporation model PDP-11, was programmed to sample each channel once every 7.5 ms in the order of EOG, P3, P4, C3, C4, Cz, and microphone channel. Due to the sequencing of the trigger and stimulus the computer began recording data one second prior to stimulus tone onset. The onset of phonation as detected by the contact microphone resulted in the cessation of data storage for that trial unit. Once all trials were completed the computer produced a summated average of all non-discarded (EOG artifact-free) trials.

The constant one second ratio between trigger pulse onset and stimulus onset was necessary for VRT identification, as VRT was determined on the basis of total time elapsed (trigger onset to phonation onset) minus one second. VRT measurements for each non-discarded trial were stored and a printout of all VRT values, mean VRT, and range was provided by the computer.

Wave Latency Measurement

Computer averaging of the recorded EEG waveforms for all non-artifacted trials yielded a summated waveform with mean amplitude values given for every 7.5 ms recording interval. An example of such a summated waveform is shown in Figure 7. For the listening-only condition N1 was defined as the time the maximum negative amplitude value occurred in the period zero to 150 ms after stimulus

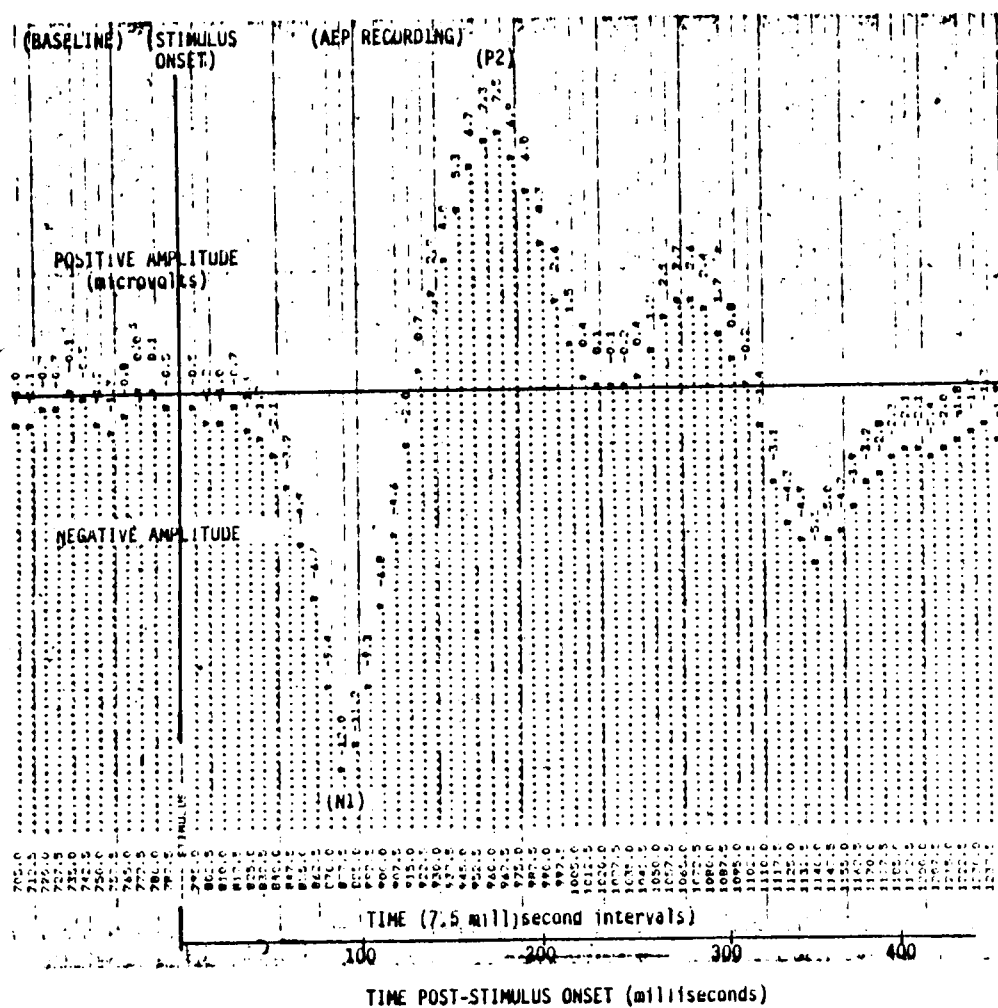


Figure 7. Illustration of summated waveform recorded from site Cz (vertex) during the listening-only condition (nonstuttering subject).

onset. P2 was defined as the time the maximum positive amplitude value occurred in the period 150 to 250 ms after stimulus onset. When the same maximum amplitude value held for two or more 7.5 ms recording intervals latency was recorded as the arithmetic mean of the time intervals involved. Because of the apparent presence of non-discarded facial EMG artifacted trials encountered in some subjects during the VRT task, a modification was made to the P2 identification criteria for the VR condition. The waveforms recorded for several subjects during the VR (phonation) condition showed a pattern of gradually increasing positive polarity shift following N1, continuing to 400 ms post stimulus onset. These were also the subjects who demonstrated the greatest number of artifacted (discarded) trials. As such a pattern did not allow accurate identification of P2 using the original criterion, it was decided to change criteria. Following a discussion with several dissertation committee members the criterion for P2 latency identification during the VR condition was changed to the first positive peak occurring after 150 ms (rather than the greatest). Peak was defined as a positive amplitude value at least one microvolt greater than the averaged value for the 22.5 ms interval immediately preceding and following it. When peak amplitude held the same value for two or more intervals an arithmetic mean was calculated for the value.

Reliability Evaluation

Reliability of Latency Measures

All recorded AER waveforms were reviewed independently by three audiologists certified by the American Speech-Language-Hearing Association who were familiar with auditory evoked response measures (the investigator and two audiologists employed by the University of Utah). Latency values for N1 and P2 for each subject in each condition were calculated independently by each reviewer using the aforementioned criterion. Subjects' names and group classification were removed from the documents prior to review. Inter-judge agreement for N1 and P2 latency values was 100 percent following the redefinition of identification criteria for wave P2 latency in the VR condition. Intra-judge reliability was not examined due to the unanimous agreement on latency measures by all three reviewers.

No test-retest reliability measures were obtained in the present study. This was an oversight by the primary investigator. Some speculation as to the reliability of the AER measures can be made via examination of standard deviation values for the various wave/condition situations (contained in Table 1, page 83) and by comparing the present results to published data.

values were relatively small (9.82 ms to 14.06 ms) ranging from six to twelve percent of group mean values. While the standard deviation value during the P2-VR condition for the stuttering group (37.20 ms) was almost twice as large as that for the normal speakers (20.81 ms), this value still was only 17 percent of the group mean. The difficulties in wave P2 latency identification in this condition were discussed previously. Overall, the relatively small standard deviation values and the similarity of the two groups' means provides indirect support for the reliability of the measures. N1 and P2 values obtained in the present study are similar to normative data reported by Davis (1976) and Polich and Starr (1983).

Reliability of VRT Measures

VRT latency values were calculated by the same computer which recorded the data in the present study. The VRT values reported are the mathematical average of all nonartifacted trials. Since no human judgement was required in ascertaining latency values, no judges were utilized in deriving mean VRT values for each subject. As a check on reliability, the principal investigator recalculated mean VRT, based on computer recorded values for all stimulus presentations in which nonartifacted responses were recorded, for three subjects. Mean VRT for

recalculated mean VRT, based on computer recorded values for all stimulus presentations in which nonartifacted responses were recorded, for three subjects. Mean VRT for these three subjects was the same as calculated via the computer program.

As was noted for AER measures, no subjects were retested in the present study. The agreement of VRT measures obtained in the present study with VRT values obtained in previous studies is discussed in Chapter IV (page 106).

CHAPTER III

RESULTS

The hypotheses of the present study were that stutterers would evidence longer measures of voice reaction time, longer cortical auditory electroencephalic response latencies, and that the addition of a phonatory response requirement would result in a greater increase in auditory response latency for the stutterers than for the nonstuttering group. To test these hypotheses a series of single factor (between group) analyses of variance (ANOVA) were computed. The factors examined were VRT latency, N1 latency and P2 latency in the listening-only condition, N1 latency and P2 latency in the VR condition, and the degree of latency shift for each slow AER component between listening-only and VR conditions. Pearson Product-Moment Correlation Coefficients were calculated to examine the relationship between auditory function (as evidenced by AER latency measures) and voice reaction time.

All analyses utilized mean voice reaction values for each subject, calculated by mathematically averaging all non-artifacted VRT trials for each subject. Similarly, AER latency measures correspond to mean values, as they result from computer summation of all non-artifacted trials. Therefore, mathematical means given for each

group are actually mean of means computations. They are referred to as simply "mean" values in the present study for ease of comprehension. VRT and slow AER component latency measures are expressed as milliseconds post-stimulus onset. Latency shift (the change in slow AER component latency between listening-only and VR conditions) is expressed as the difference in milliseconds between the two conditions (+ or -), referenced to the listening-only condition. Although AER recordings were made from several cortical sites, the present study deals only with latency recordings from site Cz.

The acceptance level for statistical significance of measures was probability measurements of 0.05 or better. Mean of means VRT values, range of means, and standard deviations for each subject are presented in Appendix D. Appendix E contains the mean AER values and latency shift amounts across conditions for each subject. The results are presented according to the series of experimental questions in Chapter I.

Voice Reaction Time

Experimental Question 1

Is there a statistically significant difference between stutterers and nonstutterers in mean voice reaction time to a pure tone stimulus?

Voice reaction time measures obtained during individual trials for the nonstuttering subjects ranged from 222.50 ms to 747.50 ms. Individual trial voice reaction times for the stuttering subjects ranged from 222.50 to 980.00 ms. Mean VRT latency for each subject is contained in Appendix D and is illustrated in Figure 8. The mean, range, and standard deviation of voice reaction time for the two groups are contained in Table 1. Mean of means voice reaction time values were 338.58 ms (with a range of 276.62 to 440.62 ms and a standard deviation of 73.18 ms) for the nonstutterers and 426.49 ms (range 320.16 to 543.72 ms and standard deviation of 102.95 ms) for the stuttering group.

A single factor analysis of variance was utilized to investigate the difference between the groups in voice reaction time and is summarized in Table 2. The results reveal a statistically significant difference ($F=6.08$, $p=0.04$) between the groups in mean voice reaction time with the stutterers averaging as a group 87.91 ms slower than the nonstutterers in initiating phonation to an auditory stimulus.

While the difference in mean VRT between groups was statistically significant, there was a considerable amount of overlap in reaction time values between the two groups. This overlap is illustrated in Figure 8. Five of the stutterers had mean VRT values within the range of the

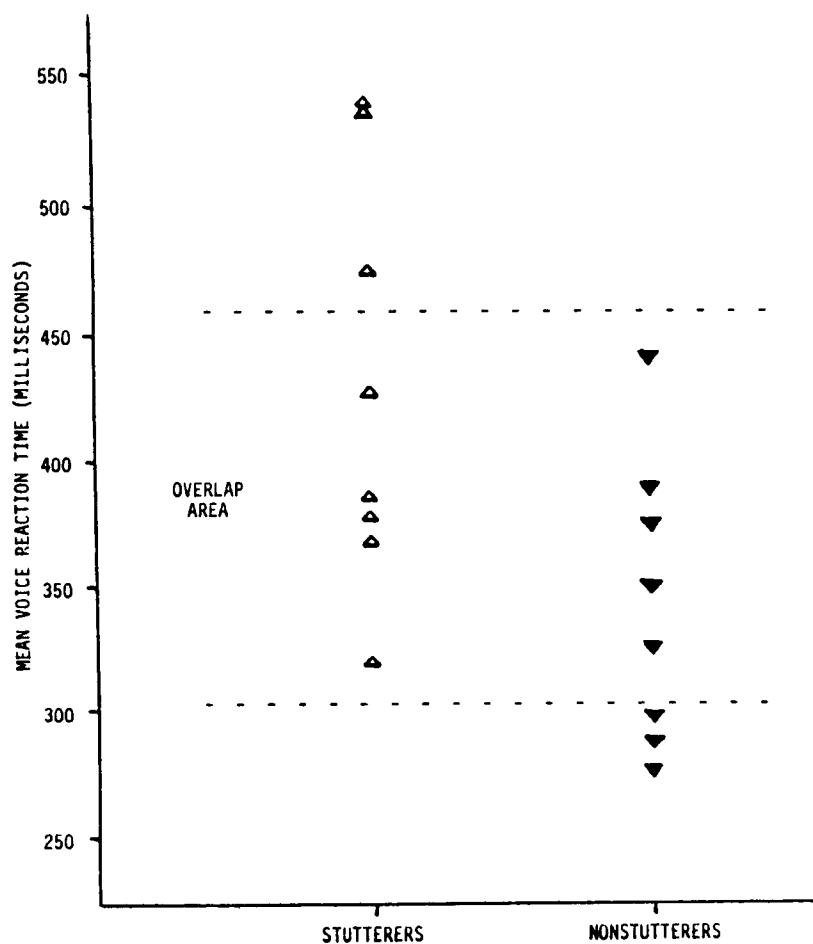


Figure 8. Scatter plot illustrating the overlap of mean voice reaction time values for individual stutterers and nonstutterers.

TABLE 1

MEAN, RANGE, AND STANDARD DEVIATION VALUES IN MILLISECONDS
FOR STUTTERERS AND NONSTUTTERERS ON VOICE REACTION
TIME (VRT), N1 AND P2 LATENCY FOR LISTENING-ONLY
(LO) AND VOICE REACTION (VR) CONDITIONS,
AND N1 AND P2 LATENCY SHIFT (LS)

GROUP/ FACTOR	NUMBER	MEAN (OF MEANS)	RANGE	STANDARD DEVIATION
STUTTERERS				
VRT	8	426.49	222.50-980.00	102.95
N1-LO	8	87.50	72.50-102.50	9.82
P2-LO	8	174.21	155.00-188.75	11.76
N1-VR	8	94.26	73.40-104.90	11.01
P2-VR	8	217.94	173.70-277.40	37.20
N1-LS	8	6.76	-3.90- 21.20	7.73
P2-LS	8	43.73	-7.55-107.40	36.68
NONSTUTTERERS				
VRT	8	338.58	222.50-747.50	73.18
N1-LO	8	98.28	87.50-110.00	9.49
P2-LO	8	183.13	166.25-200.00	11.34
N1-VR	8	104.08	86.50-113.80	14.06
P2-VR	8	213.31	187.50-258.80	20.81
N1-LS	8	5.80	-4.20- 25.10	10.18
P2-LS	8	30.18	-5.00- 73.80	23.62

TABLE 2

SUMMARY OF SINGLE FACTOR ANALYSIS OF VARIANCE BETWEEN STUTTERERS AND NONSTUTTERERS FOR THE VARIABLES VOICE REACTION TIME (VRT), N1 AND P2 LATENCY IN THE LISTENING-ONLY (LO) AND VOICE REACTION (VR) CONDITIONS, AND N1 AND P2 LATENCY SHIFT

VARIABLE	MEAN SQUARES BETWEEN GROUPS	MEAN SQUARES ERROR	F-RATIO	PROBABILITY
VRT	29842.53	4911.13	6.08	0.048*
N1-LO	464.94	93.29	4.98	0.042*
P2-LO	317.29	133.47	2.38	0.145
N1-VR	214.62	159.53	1.35	0.265
P2-VR	86.03	908.95	0.09	0.763
N1-LS	3.66	97.66	0.03	0.856
P2-LS	768.68	951.66	0.81	0.384

* Indicates statistically significant at the 0.05 level of confidence.

slower responding nonstutterers. The fastest responding stutterer had the fourth fastest mean latency overall, responding faster than five of the normal speakers. The slowest responding nonstutterer had a mean latency value slower than the mean response time of five of the stutterers. The overlap between the two groups is similar to that reported by previous studies (Cross and Luper, 1979) and is discussed in Chapter IV of the present study.

Slow AER Latency, AER-LO Condition

Experimental Question 2

Is there a statistically significant difference between stutterers and nonstutterers in N1 response latency to a pure tone stimulus during a listening-only task?

Mean latency of the AER N1 component in the listening-only condition was 87.50 ms for the stuttering group (with a standard deviation of 9.82 ms over a range of 72.50 to 102.50 ms). Mean N1 latency of the nonstuttering group was 98.28 ms with a standard deviation of 9.49 ms over a range of 87.50 to 110.00 ms.

The mean, range and standard deviation values for N1 latency in the listening-only condition are displayed in Table 1. The results of a single factor analysis of variance (as displayed in Table 2) indicates that the stuttering group exhibited a statistically significant

($F=4.98$, $p=0.04$) shorter N1 latency to a pure tone stimulus than the nonstutters.

Individual subjects' AER latencies recorded during the LO task are summarized in Appendix E and are illustrated in Figure 9. As was previously noted for VRT measures, there was a large amount of overlap in N1 latency values between the two groups. Three subjects in the stuttering group had N1 latency values shorter than any of the nonstutterers and three normal speaking subjects had N1 latencies longer than those of any of the stutterers.

Experimental Question 3

Is there a significant difference between stutterers and nonstutterers in P2 response latency to a pure tone stimulus during a listening-only task?

Stutterers also exhibited shorter mean P2 latency, with an average latency value of 174.21 ms (with a range of 155.00 to 188.75 ms and a standard deviation of 11.76 ms) as opposed to 183.13 ms (range of 166.25 to 200.00 ms and standard deviation of 11.34 ms) for the nonstutterers. As Table 2 summarizes, the results of the analysis of variance of the listening-only condition P2 latencies between stutterers and normal speakers ($F=2.38$, $p=0.14$) do not satisfy the probability level criterion selected for this study.

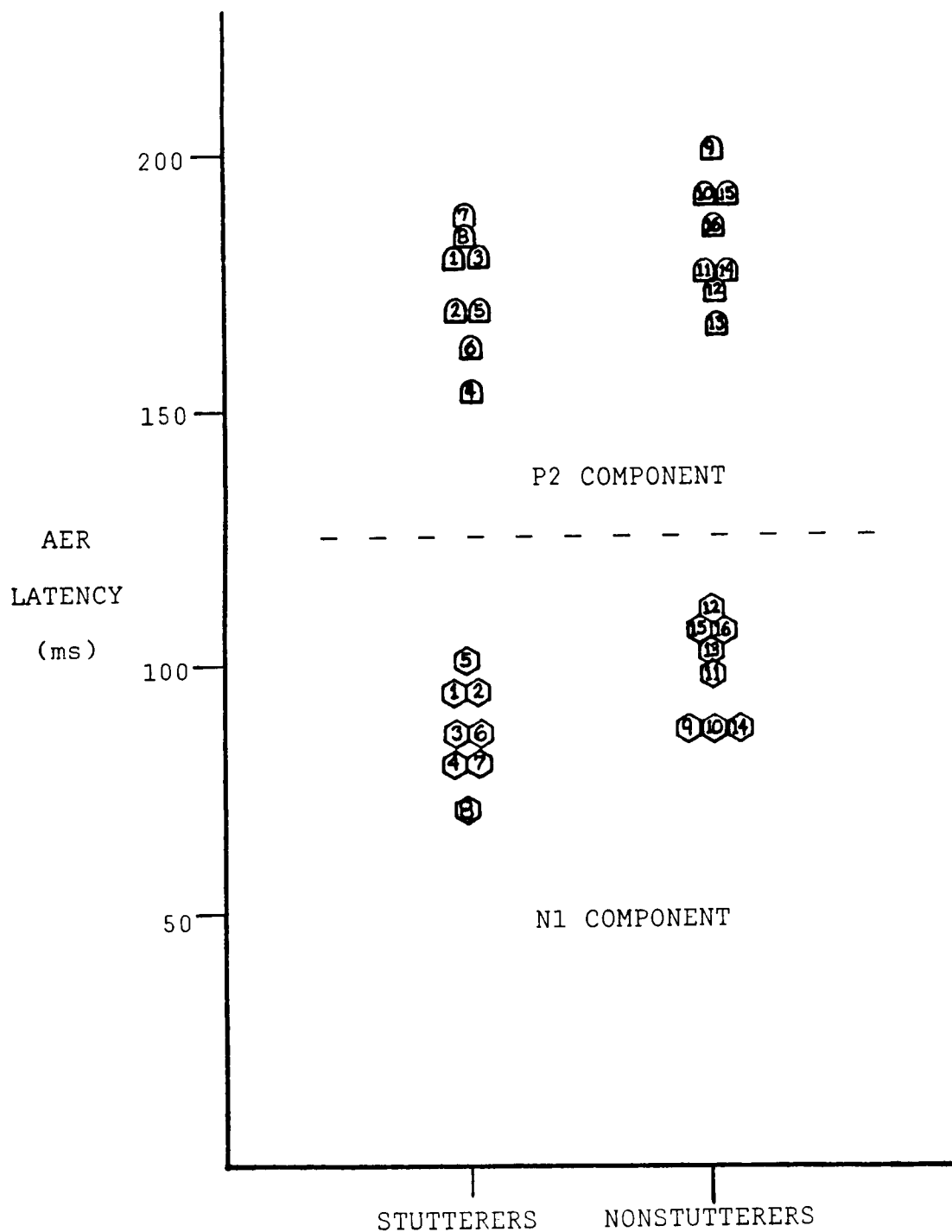


Figure 9. Scatter plot illustrating latency functions for N1 and P2 responses by individual subjects during the listening-only condition.

Figure 9 graphically displays the latency measures for N1 and P2 for the two groups. As was observed for the N1 component, overlap in latency measures also is apparent for P2. The stuttering group had the two shortest latencies overall, and three nonstutterers had longer P2 latency values than any of the stuttering group.

To summarize the AER listening-only condition results, stutterers had the shortest group mean AER components recorded during the listening-only condition, and several normal speakers had longer latency measures than the slowest responding stutterer. While stutterers were significantly faster in N1 latency, the lack of a significant difference in P2 latency and the large extent of overlap of AER measures between the groups would indicate little usefulness of slow AER results in differentiating between stutterers and normal speakers, at least when obtained during a listening-only task. The implications of the faster auditory evoked potential responses obtained from the stuttering group are discussed in Chapter IV of the present study.

Slow AER Latency, AER-VR Condition

Experimental Question 4

Is there a statistically significant difference between stutterers and nonstutterers in N1 response

latency to the pure tone stimulus when recorded during a voice reaction time task?

The mean, range and standard deviation values for the N1 and P2 components of the slow AER as recorded during a VRT task are displayed in Table 1 (page 75). The stutterers again exhibited a shorter mean N1 latency as a group than the nonstutterers (94.26 and 104.08 ms respectively). Figure 10 illustrates the range of individual subjects' N1 and P2 latencies during the voice reaction time task. Individual latency measures for the stutterers ranged from 73.40 ms to 104.90 ms, with a standard deviation of 11.01 ms. Individual latency measures for the nonstutterers ranged from 95.20 to 113.80 ms, with a standard deviation of 14.06 ms. Analysis of variance procedures, however, did not indicate the difference between groups to be statistically significant, with an F-ratio of 1.35 yielding a probability level of 0.26.

Experimental Question 5

Is there a statistically significant difference between stutterers and nonstutterers in P2 response latency to the pure tone stimulus when recorded during a voice reaction time task?

The P2-VR recordings were the only AER recordings in which the nonstuttering group displayed a shorter mean

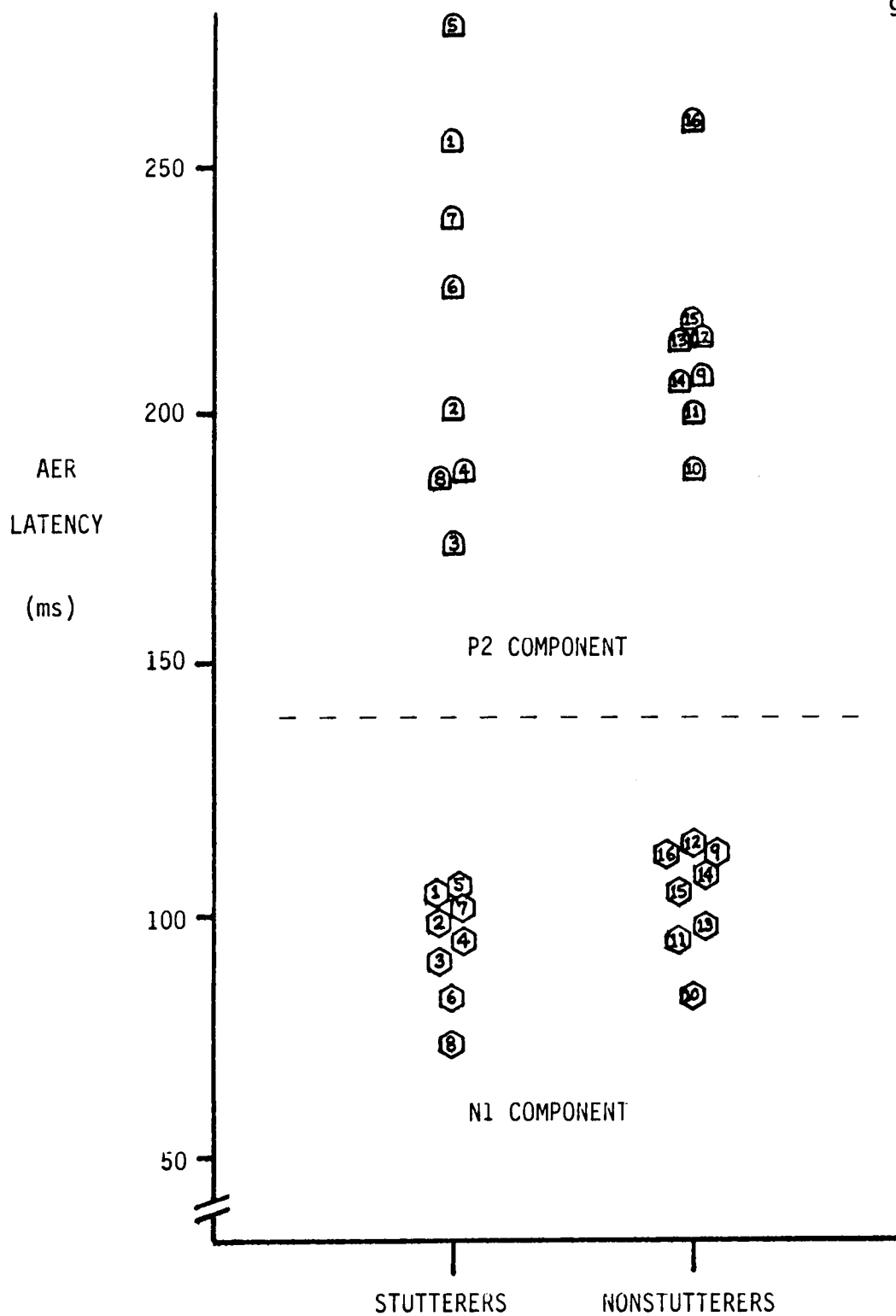


Figure 10. Scatter plot illustrating latency functions for N1 and P2 responses by individual subjects during the voice reaction condition.

latency than the stuttering group. The P2 latencies for the nonstutterers in the VR condition ranged from 187.50 to 258.80 ms with a mean latency of 213.31 ms and a standard deviation of 20.81 ms. The stuttering subjects had a mean P2 latency of 217.94 ms for responses ranging from 173.70 to 277.40 ms and a standard deviation of 37.20 ms. While this was the only condition that appeared to meet the original hypothesis that stutterers would have slower AER latencies than nonstutterers, the difference did not test as statistically significant ($F=0.09$, $p=0.76$).

Standard deviation values for both groups in the P2-VR condition were larger than those obtained for the other AER measures, indicating considerably more inter-subject variability in the P2-VR recordings. The range of latencies for the nonstutterers in the P2-VR condition (71.30 ms) was more than double the range in the P2-LO condition (33.75 ms). An even larger difference was present for the stuttering subjects, with a P2-VR latency range spanning 103.70 ms, slightly more than three times the P2 latency range in the listening-only condition (33.75 ms). This large increase in range of response for both groups during the VR condition may be due in part to difficulty in the identification of P2, as was previously mentioned in Chapter II of the present study. If the measures do reflect actual changes in P2 latency in the VR condition, the large range may reflect the use of

different processing strategies or patterns by individual speakers. These possibilities are discussed in Chapter IV of the present study.

Again there was a large degree of overlap in P2 latency measures between the groups (as illustrated in Figure 10). Three of the stuttering subjects had latency measures shorter than those of the fastest responding nonstutterer. This was the only condition, however, in which the longest AER was also given by a stuttering subject (277.40 ms).

VRT/AER Relationships

Experimental Question 6

Are there statistically significant correlations for voice reaction time and AER latency specifically:

- A. VRT with N1 latency in the AER-LO condition?
- B. VRT with P2 latency in the AER-LO condition?
- C. VRT with N1 latency in the AER-VR condition?
- D. VRT with P2 latency in the AER-VR condition?

A series of Pearson Product-Moment Correlation Coefficients were calculated to examine the relationship between voice reaction time and auditory function as measured by slow AER component latency. Table 3 presents the correlation coefficient values of VRT with N1 or P2 latency in both conditions (listening-only or voice reaction) for all subjects combined and for each group

TABLE 3

CORRELATION COEFFICIENTS FOR THE VARIABLES VOICE REACTION TIME (VRT) AND N1 AND P2 LATENCY FOR THE LISTENING-ONLY (LO) AND VOICE REACTION (VR) CONDITIONS. COEFFICIENTS ARE PROVIDED FOR ALL SUBJECTS COMBINED AND SUBJECTS BY GROUP

RELATIONSHIP	ALL SUBJECTS COMBINED	STUTTERERS	NONSTUTTERERS
VRT with N1-LO	-0.10	-0.17	0.59 *
VRT with P2-LO	-0.26	0.04	-0.27
VRT with N1-VR	-0.01	-0.15	0.28
VRT with P2-VR	0.39 *	0.22	0.65 *

* Indicates statistically significant at 0.05 level of confidence.

separately. When relations for VRT and AER latency were analyzed for all subjects, a statistically significant correlation was found only for VRT and P2 latency in the AER-VR condition ($r=0.39$). When this relationship was examined for each group, a statistically significant correlation ($r=0.65$) was present for the nonstutterers. The correlation coefficient for the stuttering group in the AER-VR condition was 0.22, which was not statistically significant.

One other statistically significant correlation was observed for the normal speaking group. While a statistically significant overall correlation was not present, the correlation among the nonstutterers between VRT and N1 in the listening-only condition ($r=0.59$) was significant.

Examination of individual subjects' VRT and AER latencies (contained in Appendices D and E and illustrated in Figure 11 for the stutterers and Figure 12 for the nonstutterers) reveals this general lack of a consistent relationship between VRT latency and the various AER latencies. The stutterer with the fastest phonatory onset time displayed AER latencies that were longer than those of the majority of the other stutterers. Similarly the stuttering subject with the slowest phonatory onset time had the slowest AER latency time among the stutterers for only one condition (P2 in the listening-only condition).

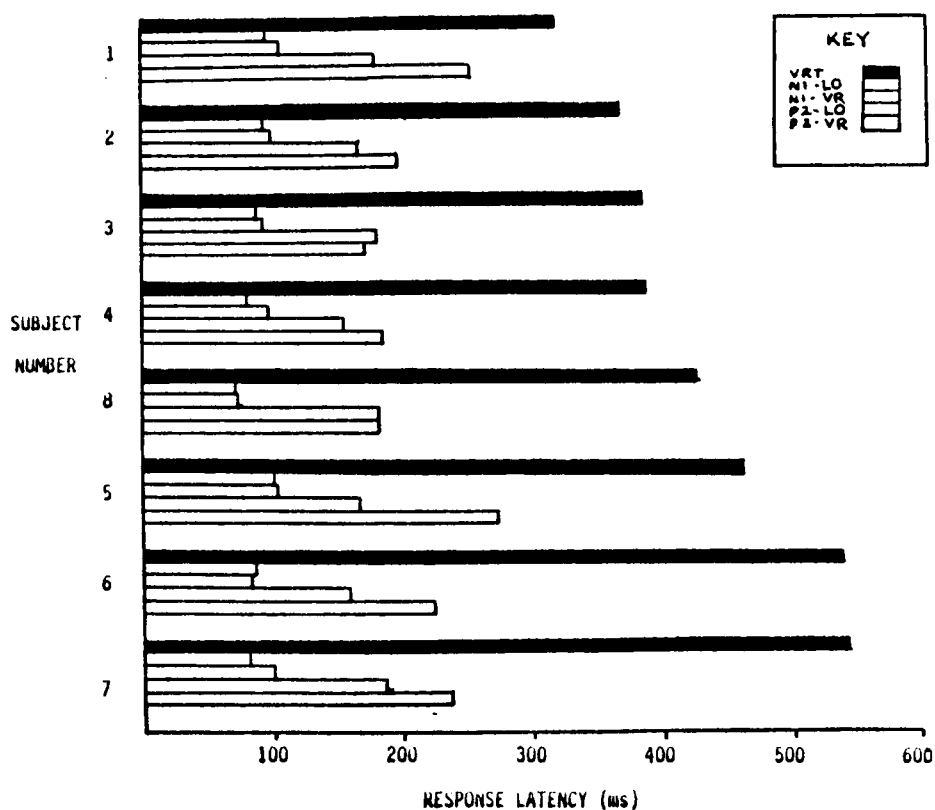


Figure 11. Illustration of voice reaction time and auditory electroencephalic response relationships for individual stuttering subjects during listening-only and voice reaction conditions.

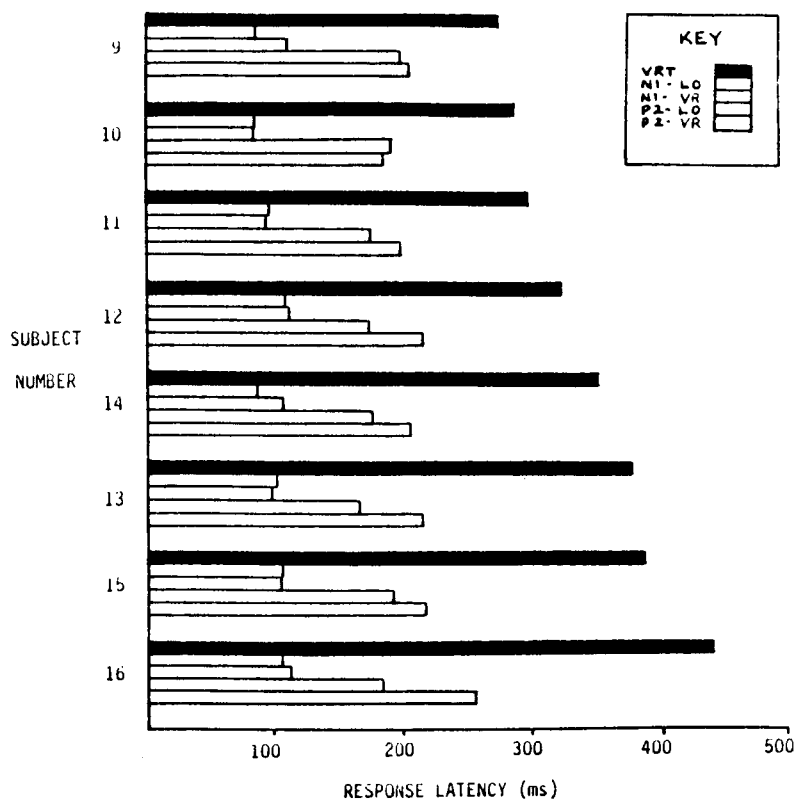


Figure 12. Illustration of voice reaction time and auditory electroencephalic response relationships for individual stuttering subjects during listening-only and voice reaction conditions.

The pattern was similar for the nonstuttering subjects, although higher correlations were observed for VRT and N1 in the listening-only condition and for VRT and P2 in the VR condition. Apparently little relationship exists between voice reaction time and a subject's initially-occurring cortical level auditory evoked responses. This appears true whether the auditory evoked responses were recorded during the voice reaction time task or not. This is discussed in detail in Chapter IV of the present study.

AER Latency Shift Between Conditions

Experimental Question 7

Is there a statistically significant difference in AER N1 latency between the listening-only and phonatory response condition?

There have been reports from other types of studies exploring reaction time and AER latencies indicating that latencies increase as additional neural demands are placed on the subject (Ritter, 1978). The present study examined AER latencies in phonatory and non-phonatory conditions to determine if the addition of a phonatory response increased AER latency. Appendix E contains the various latency measures for each subject as well as the amount of latency shift between the listening-only and VR conditions. These latency shift values were obtained by

subtracting the listening-only measure from the VRT measure. Therefore positive latency shift values indicate that AER's occurred later in the phonatory condition than in the non-phonatory condition, while negative values would indicate that responses were slower in the non-phonatory than in the phonatory condition.

Mean N1-L0 latency values ranged from 72.50 ms to 110.00 ms when examined for all sixteen subjects (stuttering and non-stuttering groups combined). The mean of means overall N1-L0 latency was 92.89 ms, with a standard deviation of 10.86 ms. Mean N1-VR latency values for the sixteen subjects ranged from 73.40 ms to 113.80 ms with a mean of means of 99.18 ms and a standard deviation of 11.30. A one way analysis of variance utilized in examining the difference between N1-L0 and N1-VR latency values for all sixteen subjects yielded an F-ratio of 2.57, which was not statistically significant ($P=0.15$). The addition of a rapid phonatory response requirement did not appreciably increase the latency of the N1 auditory response component.

Some explanations for this lack of a statistically significant difference becomes more apparent when the N1 latency shift data (Appendix E, NLS column) are examined. While four subjects had N1 latency increases of greater than fifteen milliseconds in the N1-VR condition (two stutterers and two nonstutterers), ten subjects had

latency shifts of less than five milliseconds (with five of those subjects actually exhibiting a N1-VR latency that was slightly faster than their N1-L0 latency).

Experimental Question 8

Is there a statistically significant difference in AER P2 latency between the listening-only and phonatory response conditions?

Mean of Means P2-L0 latency for the sixteen subjects in the study was 178.67 ms. Mean latency values ranged from 155.00 to 200.00 ms, with a standard deviation of 12.07 ms. When the phonatory response requirement was added (P2-VR condition) the mean of means latency was 215.63 ms. Individual subjects' mean latency ranged from 173.70 ms to 258.80 ms, with a standard deviation of 29.22 ms among the subjects. A one-way analysis of variance yielded an F statistic of 21.85, which was significant at the 0.01 level of confidence.

Examination of the amount of latency shift (Appendix E, PLS column) reveals that twelve of the sixteen subjects exhibited an increased P2 latency of fifteen or more milliseconds in the phonatory response condition. In fact, nine of the sixteen had latency shifts of thirty milliseconds or more (six stutterers and three nonstutterers).

It appears that the addition of a rapid phonatory response requirement did significantly increase the latency of the P2 component of the auditory encephalographic response as hypothesized. It did not affect the earlier occurring N1 component. Possible reasons for this discrepancy are discussed in Chapter IV.

AER Latency Shift Between Groups

The fifth hypothesis of the present study was contingent on the outcome of the seventh and eighth experimental questions, i.e., that the addition of the rapid phonatory response requirement would increase the AER response N1 and P2 latency over that observed in the listening-only condition. Hypothesis 5 predicted that if such a latency increase was observed, it would be greater for the stuttering group than for the nonstuttering group. As was reported, no statistically significant difference between listening-only and voice reaction conditions was observed in N1 latency measures. A statistically significant difference was observed in P2 latency measures. As no N1 latency difference was observed, statistical analysis to answer question 9 (between-group differences) is not reported.

Experimental Question 9

Is there a statistically significant difference between stutterers and nonstutterers in the amount of N1 latency shift between AER-LO and AER-VR conditions?

Since no statistically significant difference in N1 latency between the two conditions was present when both groups' scores were considered, no between-group statistical comparisons are reported.

Experimental Question 10

Is there a statistically significant difference between stutterers and nonstutterers in the amount of P2 latency shift between AER-LO and AER-VR conditions?

While no overall difference in N1 latency between conditions was observed, the converse was true for wave P2. The difference in P2 latency between listening-only and phonatory response conditions was statistically significant. A between-groups statistical analysis was next computed to determine if such an effect was more prominent for the stutterers than for the nonstutterers, thus supporting Hypothesis 5.

P2 latency shift for the stutterers ranged from -7.55 ms to 107.40 ms, yielding a mean P2 shift of 43.73 ms with a standard deviation of 38.06 ms. The mean P2 latency shift for the normal speakers was 30.18 ms, with a range of -5.00 ms to 73.80 ms and a standard deviation of 24.60

ms. An F-ratio of 0.81 ($p=0.38$) was computed in a one way analysis of variance. This lack of a statistically significant between-group difference may be an effect of the extremely large variability in response (standard deviations were almost as large as group means) or may indicate that stutterers and nonstutterers are equally affected by phonatory programming demands. These possibilities are discussed in Chapter IV.

CHAPTER IV

DISCUSSION

The theoretical basis for the present study was that slow and/or inefficient auditory processing could contribute to the slower phonatory initiation time to acoustic stimuli that has previously been reported for stutterers. Auditory processing in the present study was examined in respect to latency measures of early cortical level responses to auditory stimuli (slow AER waves N1 and P2). Phonatory initiation time to an acoustic stimulus was examined within the context of a VRT paradigm. It also was theorized that the addition of an immediate phonatory response requirement to the acoustic stimulus might affect the way in which the signal was processed, and that this might appear as a longer latency for N1 and P2 responses in the AER-VR condition than in the listening-only condition. The expectations of the investigator were that stutterers would exhibit a greater increase in AER latency for the phonatory condition than would nonstutterers.

The hypotheses tested in the present study were developed from the above concepts. These hypotheses were . as follow:

1. Because several earlier studies indicated that stutterers are slower than nonstutterers in voice reaction time tasks, it was hypothesized that the stuttering group in the present study would be slower than nonstutterers in initiating phonation to an auditory stimulus.
2. Since there are many indications in the literature that stutterers may possess a less efficient auditory system than nonstutterers, the second hypothesis of the present study was that stutterers would exhibit auditory encephalographic responses N1 and P2 to a pure tone stimulus that were slower in latency than those of nonstutterers.
3. The third hypothesis of the present study was that there would be a relationship between AER latency and voice reaction time. This was developed on the basis that if differences in AER latency measures exist between stutterers and nonstutterers, such differences may reflect the use of different processing patterns or less efficient processing systems between the two groups. Such processing differences may be a component involved in reaction time to auditory stimuli.

4. The fourth hypothesis of the present study was that AER latency would be slower in the AER-VR condition than in the AER-LO condition. This was developed from prior research indicating that reaction time often increases when a subject is forced to make a choice or decision before responding. This increase in reaction time is thought to reflect added neural processing and decision making. As the phonatory response utilized in the present study requires precise coordination of multiple respiratory, laryngeal, and articulatory muscles, increased neural activity would be expected when compared to a passive listening condition.
5. The fifth hypothesis of the present study was that stutterers would demonstrate a greater degree of AER latency shift between conditions than nonstutterers. If the stutterers demonstrate a less efficient neural system (as previously hypothesized), the increased neural demands of the phonatory response condition should affect the stutterers to a greater degree than the nonstutterers.

The interpretation and discussion of the results of the present study are presented in regard to each of the above hypotheses.

Hypothesis 1: Voice Reaction Time Differences

Difference Between Stutterers and Nonstutterers

The initial hypothesis of the present study was that stutterers would be significantly slower than nonstutterers in initiating phonation to an auditory stimulus. This hypothesis was tested in experimental question 1 and appears to be a valid hypothesis. Results of the present study indicate that the stuttering group had a mean voice reaction time statistically significantly longer than that of the normal speakers, with an 87 millisecond difference between groups. Such findings are in good agreement with previous VRT studies that reported stutterers to be significantly slower in VRT (Hayden, 1975; Adams and Hayden, 1976; Starkweather, Hirschmann, and Tannebaum, 1976; McFarlane and Prins, 1978; Cross, Shadden, and Luper, 1979; Cross and Luper, 1979; and Reich, Till, and Goldsmith, 1981). The magnitude of the difference observed between stutterers and nonstutterers in the present study is also close to that reported by several of the aforementioned studies that utilized adult subjects (Starkweather, Hirschmann, and Tannenbaum, 1976; Cross, Shadden, and Luper, 1979; and Reich, Till, and Goldsmith, 1981).

Variability of VRT Measures

This study was similar to many of the previous reaction time studies that reported statistically significant differences between stutterers and nonstutterers in that a large number of VRT trials were used (e.g., Cross, Shadden, and Luper, 1979; Cross and Luper, 1979). Many of the studies that failed to find statistically significant differences between stutterers and normal speakers utilized a small number of trials (Murphy and Baumgartner, 1981; Venkatigiri, 1981) and some did not use initial trial sets to allow the reaction time to stabilize. With the high amount of variability reported within subjects for voice reaction time (Cross, Shadden, and Luper, 1979; Cross and Luper, 1979), use of small numbers of trials would have increased the effects of such variability on influencing statistical analysis of differences. An indication of the high degree of variability in reaction time measures within individuals is present in the current study. Both groups, stutterers and normal speakers, displayed a considerable amount of variability. Standard deviations for individual subjects' voice reaction time measures ranged from 33 to 81 ms for the nonstuttering group and from 38 to 133 ms for the stutterers. Five of the stutterers had VRT standard deviation measures larger than 60 ms; only one of the nonstutterers had a standard deviation exceeding this

level. Cross (1978) suggested that such variability in the ability to initiate phonation may play a role in stuttering behavior. The presence of such inconsistent phonatory initiation activity in the normal speech production pattern could interfere with learning the coordinated motor pattern necessary for fluent speech.

This view that increased variability may hamper accurate coordination of speech motor activity agrees with earlier hypotheses on the nature of stuttering, such as that of Van Riper (1971) and Perkins, Rudas, Johnson, and Bell (1976). Van Riper (1971), following an extensive review of research concerning stuttering, suggested that stuttering should be considered a disorder of timing. He theorized that when a person stuttered on a word there was a temporal disruption of the simultaneous and successive programming of muscular movements required to produce one of the word's integrated sounds. If speaking requires the production of motoric sequences that must be precisely timed, uncertainty in phonatory initiation timing could interrupt the simultaneous and successive motor coordination necessary for fluent speech, resulting in a stuttered production. Similarly, Perkins, Rudas, Johnson, and Bell (1976) theorized that stuttering may result from a discoordination of the processes of respiration, phonation, and articulation. The high degree of variability in stutterers' phonatory initiation time noted

in the present and previous VRT studies may be a source of disordinating activity.

Increased VRT Measures in the Present Study

The present study agreed with previous reports on the magnitude of VRT differences between stutterers and normal speakers, and with previous reports of high VRT variability. The mean reaction time measures obtained in the present study, however, were considerably longer than those reported by others. One factor that may have contributed to this was that no warning or ready stimulus was utilized. This would not necessarily explain all of the discrepancy however, as VRT latencies were longer than those reported by other studies in which warning signals were not utilized (e.g., Cross and Luper, 1979). Another source that also may have contributed to the longer VRT measures was the subjects' state during reaction time procedures. The subjects in the present study were instructed to maintain a relaxed, neutral body posture, attempting to reduce voluntary muscular activity to the best of their ability. Such procedures may have inhibited subjects from tensing laryngeal and oral structures to facilitate vocal reaction time. The use of the relaxed body posture and downward gaze with relaxed eye musculature may also have encouraged a slightly lower attentive state.

Overlap in VRT Measures Between Groups

Another aspect of the present study's VRT results that merits discussion is the overlap of VRT values between the stutterers and nonstutterers (Figure 8, page 82). When all mean reaction time values are considered, the three fastest were recorded for nonstutterers. The fastest responding stutterer had the fourth fastest overall mean VRT, and the VRTs of the next four fastest responding stutterers fell within the range recorded by the remaining five normal speakers. The mean reaction times of the remaining three stutterers were slower than those for any of the nonstutterers. While three nonstutterers had the fastest mean reaction times overall, and three stutterers had the slowest times overall, the bulk of mean reaction time values for the two groups were intermingled.

Relationship Between VRT Measures and Stuttering Severity

Reaction time measures did not clearly delineate the two groups. Cross and Luper (1979) suggested that the overlap found between the groups in their study may have been related to stuttering severity. The more fluent speaking stutterers may present voice reaction times that closely approximate those of normal speakers. To determine if this was a valid explanation for the overlap observed in the present study, three Speech-Language Pathologists certified as clinically competent by the American Speech-Language-Hearing Association were asked to

independently rate, post-hoc, each of the stutterers on a stuttering severity scale comprised of levels one to five. A rating of one was indicative of a very fluent speech pattern, and five was used for very severe communication problems. All three Speech Pathologists were familiar with the speaking pattern of each of the stutterers within a wide variety of speaking situations. The three judges were in unanimous agreement on severity ratings for five of the eight stutterers. For the other three subjects, two of the three judges were in agreement, and these were the severity values utilized in statistical analysis. For each of the three subjects, where the agreement was not unanimous, the dissenting judge's severity rating differed only by a single level from the majority rating. A Pearson Product-Moment correlational procedure performed post hoc to examine the relationship between stuttering severity and VRT yielded a correlation coefficient of 0.277, a much weaker statistical relationship than expected.

This lack of agreement between level of fluency and VRT latency may have resulted from the fact that a majority of the stuttering subjects were enrolled in a fluency shaping stuttering therapy program at the time the study was conducted. Several of the stutterers who previously would have been ranked as exhibiting a severe communication impairment were displaying highly fluent

speech patterns following therapy. All were rated on the basis of their present speaking pattern. The result then was that a few of the stutterers with relatively long VRT measures were rated as highly fluent. It would be interesting to follow the maintenance of fluency in these subjects over a longer period to determine if such physiological measures as VRT may have a predictive ability towards therapy success.

Hypothesis 2: Slow Auditory Electroencephalic
Response Latency

The hypothesis tested by experimental questions 2, 3, 4, and 5 in the present study was that stutterers would demonstrate longer AER latencies than nonstutterers in both listening-only and phonatory response conditions. This hypothesis was developed in light of prior research indicating the presence of central auditory processing deficits in stutterers (Curry and Gregory, 1969; Hall and Jerger, 1978; Molt and Guilford, 1979) and the indications that voice reaction time differences between stutterers and nonstutterers were more pronounced for acoustic than other modality stimuli (McFarlane and Prins, 1978). The results of the present study, however, do not support the hypothesis that stutterers would have significantly longer N1 and P2 AER latencies. In fact, the converse appears to be at least partially true: the stuttering group

exhibited a statistically significant shorter AER latency in one of the conditions (AER component N1 in the listening-only condition). Mean of means N1-L0 latency for the stutterers was 87.50 ms, which was 10.78 ms shorter than the mean of means N1-L0 latency for the nonstutterers (98.28 ms). There were no other statistically significant differences between the groups for either the N1 or P2 component for either condition.

In two of the three other wave/condition situations examined, the differences in group mean values were similar to those observed during the N1-L0 situation, but the differences were not statistically significant. The stuttering group had shorter mean of means latencies than the nonstutterers for wave P2 in the listening-only condition, and wave N1 in the voice reaction condition. Magnitudes of these differences in means were very similar, with a difference of 8.92 ms in the P2-L0 condition and 9.82 ms in the N1-VR condition, very close to the difference observed for the N1-L0 condition (10.78 ms). The only condition in which the nonstutterers displayed a shorter mean of means latency than the stutterers was for the P2 component in the phonatory response condition, with the nonstutterers 4.63 ms shorter in latency. It is important to again mention, however, that the only statistically significant difference was for the N1 component in the listening-only condition.

The lack of a statistically significant difference in mean AER latency for three of the four conditions/ components examined indicates that there are not major differences between stutterers and nonstutterers in the AER components examined in the present study. These results will be discussed relative to the N1-L0 discrepancy, the lack of statistically significant differences and the problems encountered in recording and identifying P2-VR.

N1-L0 Discrepancy

Contrary to the original hypothesis of the present study, the stuttering group had a significantly shorter mean N1-L0 latency than the nonstutterers. There are several possible explanations for this. One explanation is that it may reflect hyperexcitability of the central nervous system. Brutten and Shoemaker (1967) cite research by Frank indicating that a constitutional predisposition to excessive excitation of the central nervous system results in a tendency towards anxiety, hypersensitivity to the environment, hyperactivity, and hesitancy. Brutten and Shoemaker report that some of these symptoms are observed in stutterers, as also is autonomic nervous system overreactivity, and these features, combined with conditionability, may be an important factor in fluency failure. The significantly

shorter N1-L0 latency for the stuttering group may reflect a tendency towards hyperexcitability or overreactivity of the nervous system. Small, De Meyer, and Kendall (1969) reported shorter auditory and visual evoked potential latencies in autistic children, who are hyperresponsive to stimuli. Martineau, Garreau, Barthelemy, and Le Lord (1980) reported an increased latency (normalization) of visual and auditory evoked potentials in autistic children treated with oral doses of vitamin B6 and magnesium, a treatment which often improves an autistic child's responses to stimuli.

The significantly shorter N1 latency for the stutterers was not observed in the phonatory response condition. While this would seemingly remove support for the hyperexcitability hypothesis since one might expect the overreactivity to be present in both conditions, an alternate explanation is also available. The significant difference may not be present for the N1-P2 condition because stutterers (and possibly the nonstutterers as well) may approach the rapid phonatory response condition with a different neural set or processing strategy. Some support for this position is present in the lack of intra-subject consistency in wave N1 latency between the two conditions. Information on AER latency for each individual subject is available in Appendix E. Examination of these data reveals that although subject

eight (stutterer) had the shortest N1 latency in the stuttering group for both listening-only and voice reaction conditions, this consistency was not present for the majority of the stuttering subjects. For example, subject seven had the second shortest N1-L0 latency, but had the sixth shortest N1-VR latency.

The lack of consistency in N1 latency between conditions may reflect the use of a different neural set or strategy. This premise is also supported by other AER data. Four of the nonstutterers had N1-VR latencies that were shorter than the latency obtained in the N1-L0 condition. While these differences in latency were small, generally on the order of one to four milliseconds, no such pattern was seen in the stuttering group. Only one stutterer had a shorter N1-VR than N1-L0 latency (subject seven). This may be an indication of the use of different processing strategies by the two groups.

Inconsistency within individual subjects' N1 latency measures and differences between the two groups in latency shift patterns have been cited in the present study as evidence of possible usage of different processing patterns. A third indication of possible neural differences among subjects was also observed among the members of each group. Two subjects in each group had N1 latency shifts of 20 ms or more for the VR condition while the majority of subjects in each group had latency shifts

of less than five ms. This again may reflect the use of different processing strategies or the presence of less efficient neurological systems among group members. The greatest consistency of wave form latency between conditions in the stuttering group was seen in the subjects with the longer latencies. Subjects one, two, and eight had the three longest N1 latencies in the listening-only condition, and these three subjects accounted for three of the four longest latencies in the voice reaction condition. Those whose neurological system was slow to respond in one condition generally were also slower to respond in the other condition. Those stutterers who were quick to respond in one condition were not necessarily as fast at responding in the second condition.

Obviously there are very few clear patterns in these data. There does appear to be enough consistency, however, to suggest that either the two groups, or more likely some members within each group may utilize different processing strategies or demonstrate varying levels of neurological efficiency.

There is a third possible explanation for why the N1-L0 results did not agree with the original hypothesis. The original hypothesis was predicated on reports from other studies that examined auditory processing in stutterers and nonstutterers, with the stutterers

generally performing significantly poorer on these central auditory tests. There is a possibility that the present study examined a very different type of processing from that normally examined by traditional central auditory tests. This possibility will be explored further in the next section of this chapter, dealing with the lack of observable differences between the groups in AER latency.

Lack of Significant Differences in AER Latencies

As was previously mentioned, the original hypothesis of the present study was that stutterers would display longer AER latencies than nonstutterers. The N1-L0 discrepancy from this has been previously discussed. There was another finding that was contrary to the hypothesis, and that was the lack of a statistically significant difference between stutterers and nonstutterers in AER latency for three of the four wave/condition situations examined. Two possible explanations for this are the large degree of overlap between the two groups for each wave/condition and the possibility that the present study's methodology examined a different type of auditory processing than had been measured in previous studies.

The first of these two points that will be discussed is the overlap between the groups. The AER wave latency in each condition for individual subjects is contained in Appendix E. A summary of those data follows.

N1-L0 recordings. The three shortest N1 latencies recorded were for stuttering subjects. The three longest N1 latencies were observed in nonstutterers. The latencies of the remaining ten subjects (stutterers and nonstutterers) were intermingled.

P2-L0 recordings. The two shortest P2 latencies in the phonatory response condition were produced by stuttering subjects. The largest discrepancy in group latency ranking was observed in this condition, for the five longest N1 latencies were produced by the nonstuttering group. The latencies of the six remaining stutterers were interspersed among those recorded for the four fastest responding nonstutters.

P2-VR recordings. As in all the other conditions, the shortest latencies were observed in stutterers (three). The voice reaction condition was also the only condition in which the longest AER latency was recorded for a stutterer. The latencies of the nonstuttering subjects were intermingled among those of the stutterers.

It is apparent that responses for the two groups greatly overlapped for both AER components and in both experimental conditions. Stutterers as a group did not differ considerably from nonstutterers in latency of the AER N1 and P2 components. The frequent intermingling of latency values between the two groups probably accounts

for the lack of statistically significant differences for three of the four wave/condition situations observed. Measurement of N1 and P2 latency does not delineate between the majority of stutterers and nonstutterers, including the N1-L0 condition, for even in that condition the majority of latency measurements for nonstutterers and stutterers fell within a similar range.

What such a conclusion masks however, is that some of the stutterers did respond differently from at least some of the nonstutterers. Some of the stuttering subjects were noticeably faster than the majority of the nonstutterers for at least one AER component in both conditions. For example, stutterer number eight had the shortest N1 latency of all 16 subjects in both conditions. Stutterer number four had a N1 latency shorter than any nonstutterer in the listening-only condition and was faster to respond than all but two nonstutterers in the phonatory response condition. Stutterer number four also had a shorter P2 latency than that observed for any of the nonstutterers in both conditions.

Such a pattern as described above would appear to differentiate some of the stutterers from the nonstutterers. Yet the large degree of overlap in subjects' performances between the two groups meant that statistically significant differences between the groups

generally were not seen. This large degree of overlap in measures between stutterers and nonstutterers has been observed in many other studies that utilize neurological and/or physiological measurements. A factor involved in this may be that the assumption of homogeneity of neurological and physiological responses in the stuttering group is an error, for wide differences in performance among the stutterers for each wave/condition situation were observed, as was a wide variance in VRT latency. It may be that very different etiologies may exist among the stutterers, with some being primarily physiologically/neurologically-based, others primarily psychologically-based, and still others may have developed stuttering through learned aberrant speaking patterns or learned emotional responses to speech. If such is the case, then one might not expect a group of stutterers to perform significantly better or worse than a group of nonstutterers on measures examining any one of the above areas. Indeed, it would be more appropriate to discuss the results of such studies as the present one in terms of the responses of individual subjects rather than using parametric statistical treatments, since the assumption of a normal distribution of the independent variable/object of interest may be incorrect.

The other explanation mentioned for the lack of support for the second hypothesis of the present study

(between-group differences in AER latencies) was that the AER components examined in the present study may reflect different levels of processing than had been evaluated in prior auditory research.

Central auditory tests, such as the Staggered Spondaic Word Test (SSW) developed by Katz (1962) and the Synthetic Sentence Identification test battery (SSI) developed by Speaks and Jerger (1965) employ linguistic stimuli, and are thought to reflect high-order cortical level processing of such features as phonemic, phonologic, semantic, and syntactic variables. The measures employed in the present study involved early cortical level processing of a pure tone (nonlinguistic) signal.

Both N1 and P2 are thought to reflect exogenous processing of the signal, i.e., selective attention to specific characteristics of the stimulus (Donchin, 1975; Hillyard and Picton, 1978; Tueting, 1978, McCallum, 1980). Both the N1 and P2 components are seen in response to specific physical parameters of the signal, such as frequency, intensity, duration and presentation rate (Picton, Woods, and Proulx, 1978). While both index important processing necessary in recognition of an incoming stimulus as the proper one to respond to, they may provide little indication of the efficiency or intactness of the later occurring higher-order processing, such as examined by the language-based central auditory

tests. Observations of this later occurring processing may be done more appropriately via examination of the endogenous AER responses such as P300, the sustained negative potential, and the contingent negative variation (Donchin, 1975).

Limitations of the experimental measures were recognized at the outset of the present study. The AER latency measures were selected in recognition that they could be utilized to examine auditory processing of the stimulus signal utilized in many VRT studies, and that this could be accomplished during actual voice reaction time measures, an option offered by no other method of examining auditory function. It was hoped that if abnormal auditory processing of the stimulus was a factor in the slower voice reaction times often reported for stutterers, this aberrant processing pattern would be observable in N1 and P2 latency measures.

There were not, however, statistically significant differences between the groups for either wave in either condition (except for the N1-L0 situation). The results of the present study as examined thus far do not support the contention that the use of different or inefficient auditory processing patterns by the stutterers is a major factor in their slower group voice reaction time measures, at least when group wave measures and group VRT measures are examined. There may be, however, an exception to this

conclusion - the P2-VR results can be interpreted several ways.

P2-VR Recording Difficulties

The N1-L0 discrepancy from the original hypothesis (discussed previously on page 104) and the lack of support in the AER wave/condition measures for that hypothesis have been discussed above. There is a third area relative to the AER measures that also warrants discussion, for it also may have affected the outcome of the study. This area is that of the difficulties encountered in recording and identifying the P2 wave component during the VR condition.

As was previously reported, there was not a statistically significant difference between stutterers and nonstutterers in wave P2 latency recorded during the voice reaction condition. This was however, the only AER wave/condition situation in which the nonstutterers had a shorter mean group latency than the stutterers (4.63 ms shorter). There is slight support for questioning either or both of the above results, for this was the one wave/condition situation in which actual wave peak latency for some of the subjects was unclear.

The reason for the difficulty in the identification of the P2-VR latency was that the AER response was occurring very close to the phonatory response. In some subjects

electromyographic activity associated with the phonatory response was occurring at approximately the same time as the P2 response, masking clear identification of the auditory component. Both stuttering and nonstuttering subjects had individual reaction time trials in which phonation occurred at 222 ms after stimulus onset, which is extremely close to the average occurrence of the P2 component (approximately 200 ms).

This was apparently not a factor for the majority of the subjects, for a clear P2 amplitude peak was observable, and the resulting waveform resembled the P2 waveform recorded in the listening-only condition. For seven of the subjects, however, a clear peak amplitude of P2 was not observable. The seven subjects instead exhibited a slow gradual positive shifting of the electrical potential which continued climbing in amplitude out to approximately 400 ms post-stimulus onset. The original criteria for identification of the P2 latency was the latency of the greatest positive peak occurring after 150 ms post-stimulus onset. As P2 could not be identified using this criteria for seven of the subjects, the criteria was changed to reporting the first positive peak (rather than the greatest) occurring after 150 ms post-stimulus onset. Peak was defined as a positive amplitude value at least one microvolt greater than the

averaged value for the 22.5 ms interval (three samplings of the electroencephalic brain activity) immediately preceding and following it. When peak amplitude held at the same value for two or more intervals an arithmetic mean was calculated for the value.

All of the summated waveforms were reviewed independently by three audiologists who were familiar with auditory evoked response measures. Using the revised criteria all three reviewers agreed on the same P2 latency measure for each of the seven subjects. While the identification of a latency value for wave P2 was reliable, the validity of that measure is questionable. In two subjects the waveform peak identified as P2 was a prominent peak in the gradually positive-climbing waveform. The waveform peak identified as P2 for the remaining five subjects was only the first of several minor peaks in the waveform shift. The waveform morphology after 100 ms for these five subjects did not resemble the recordings made of them during the listening-only condition, nor did it resemble previously reported waveforms for the P2 component. The latency value reported for P2 in the phonatory response condition may be inaccurate for approximately one-third of the subjects.

This factor becomes especially pertinent since five of the seven subjects exhibiting abnormal P2-VR waveform

morphology were stutterers. This different morphology may indicate the presence of a much higher level of muscle action potential activity towards the phonatory task for the stuttering group. Indeed, there were more trials rejected due to recording artifacts for the stutterers than for the nonstutterers (mean number of rejected trials was 30.88 for the stutterers and 15.75 for the nonstutterers). Another explanation, however, would be that the stutterers displayed a different auditory response pattern than the majority of nonstutterers during the phonatory response condition. What was identified as P2 latency and statistically compared to the nonstutterers' measures may have not been the P2 component at all. The P2 component may have been absent completely for some of the stutterers during the phonatory response condition, or it may have occurred at a different time than was identified and was simply masked out by the higher level of EMG activity in the stutterers. There is support for the contention that P2 latencies may be different than recorded in the results of the examination of VRT/AER relationships (Hypothesis 3) and latency shift between conditions (Hypothesis 4).

Hypothesis 3: VRT/AER Relationships

The third hypothesis of the present study was directed at the relationship between AER latency and voice reaction

time. The hypothesis was that if differences in AER latencies were observed between the groups, these differences may reflect the use of aberrant auditory processing patterns and therefore would be related to the voice reaction time latency. As discussed above, few differences in AER component latency were observed between stutterers and nonstutterers. Since the hypothesis was predicated on the existence of differences, it might then be expected that there would not be robust relationships between VRT and AER latencies. This was the case in the present study. The statistical relationship was examined between VRT and latency of each of the two AER components for the two conditions. The relationship was examined both for all subjects combined and individually for each group. Only three statistically significant correlations were observed. These are discussed below.

VRT with N1-L0 Latency

A statistically significant correlation between voice reaction time and N1 latency was observed only for the nonstuttering group, and only in the listening-only condition. This also, however, was the only condition in which a statistically significant difference in AER latency was observed between the groups, with the stutterers shorter than the nonstutterers in N1 latency. The nonstutterers correlation coefficient was 0.59,

indicating only a weak relationship between VRT and N1 latency. As N1 latency increased among the nonstuttering subjects, their VRT latency also increased. This relationship was in agreement with the original hypothesis. This was, however, only for the nonstutterers. The correlation coefficient for the stutterers was -0.17 , indicating that voice reaction time measures decreased as N1 latency increased.

VRT with P2-VR Latency

A statistically significant correlation between VRT and P2 latency was observed during the phonatory response condition. The overall correlation (all subjects combined) was 0.39 which, although it tested out as statistically significant, indicated only a very weak positive relationship. When this relationship was further examined to determine if it was present for both groups, it appeared the relationship was clearest for the nonstutterers. The correlation coefficient for VRT and P2 latency for the stutterers was 0.22 , which was not statistically significant. For the nonstutterers the r -value was 0.65 , which was statistically significant. As with VRT and N1-L0 for the stutterers, this relationship was positive as hypothesized (voice reaction time increased as a function of P2 latency increase) but was not noticeably robust.

There is a second important factor present in the VRT/P2-VR relationship and that was the lack of this relationship for the stuttering group. As was discussed earlier it was difficult to determine the actual P2 peak latency during the VR condition, especially for the stutterers. It was proposed that P2 latency may have been incorrectly identified for five of the eight stutterers. If this was the case and P2 latency either occurs at a different time or is not present, one would not expect to see a clear relationship. Since no major problems in P2 identification were present for the nonstutterers, and a clear relationship was observed, some indirect support is present for the contention that P2 latency for stutterers during speaking situations may be more divergent from that of the nonstutterers than was reported in the present study. Some stutterers may be doing something different in auditory processing during the phonatory response condition. There is further support for this contention when the amount of latency shift for wave P2 between the two conditions is examined

Hypothesis 4: Increase in AER Latency Between Conditions

The fourth hypothesis of the present study was that the addition of a phonatory response requirement would increase the N1 and P2 response latency over what was observed in the listening-only condition. This hypothesis

was predicated on prior reports in the literature that latencies of various AER components increase as additional neural demands are required from the subject. These generally have been the endogenous AER components such as P300 and the contingent negative variation (Donchin, 1975; Ritter, 1978; McCallum, 1980).

In the present study the change in AER latency was derived by subtracting the latency in the listening-only condition from the latency in the voice reaction condition. This value was referred to as latency shift and was expressed in milliseconds. Latency shift values were expressed as either negative numbers (shorter VR condition latency than LO condition latency) or positive numbers (longer VR condition latency than LO condition latency). The derived latency shift values for the individual subjects are contained in Appendix E.

N1 Latency Shift Between Conditions

A one way analysis of variance between N1-LO latencies and N1-VR latencies for all 16 subjects did not result in a statistically significant F-ratio. The difference in means between the two conditions was 6.29 ms (N1-LO mean of means was 92.89 ms and N1-VR mean of means was 99.18 ms). The N1 values recorded for individual subjects generally were very similar between the two conditions. This can be seen by examination of Figures 11 and 12

(pages 95 and 96) and/or examination of latency shift values contained in Appendix E. More than one half of the subjects (62 percent) had latency shifts of less than five ms. Five of those subjects had negative latency shift values (N1-VR latency was shorter than N1-L0 latency).

The N1 latency shift data do not support the hypothesis that the increased neural processing required for production of an immediate phonatory response will increase AER latency beyond that recorded in a task requiring no physical response. It is highly probable that the type of processing that N1 is thought to reflect (stimulus parameters) was simple enough in the present paradigm that it may not have been affected by the presence of other neural demands. The subjects' only requirements were the recognition of a simple pure tone (single-frequency signal). If N1 indexes intensity or frequency characteristics, recognition of the stimulus signal from the low ambient noise level would not require very sophisticated processing. Quite likely such a low order of processing complexity would be unaffected by preparatory phonatory activity.

P2 Latency Shift Between Conditions

Unlike the N1 condition, a statistically significant difference was present between mean P2 latency for the listening-only and the phonatory response condition. Mean

P2-L0 latency for the sixteen subjects was 178.67 ms and mean P2-VR latency was 215.63 ms, a difference of 36.96 ms. Seventy-five percent of the subjects had a positive latency shift of at least 15 milliseconds in the phonatory response condition. Six stutterers and three nonstutterers had latency shifts of 30 milliseconds or more.

P2 latencies were statistically significantly longer in the voice reaction condition than in the listening-only condition. As the only difference in response requirements between the two conditions was that of rapid phonation of / $\wedge$ / following stimulus onset, it would appear that extra attention to the signal (as it was meaningful) or the extra neural activity required for rapid phonation would account for that latency increase. There is a third explanation, the difficulty in identifying P2-VR latency. Each of these is discussed below.

Attention to Stimulus Signal. It is still not understood what level or specific processing feature the P2 waveform indexes. It is thought to index processing of one or more physical features of the signal, such as intensity, frequency, duration, rate, etc. As the listening requirements in the VR condition were relatively simple (recognition of a high amplitude single-frequency

signal against a low intensity ambient noise background) it would appear that little additional processing activity would be required over that of the listening-only condition. It would likely not be on the order of 30 milliseconds or more, which was the latency shift observed for nine of the sixteen subjects. Something other than added attention to the stimulus may account for the significant latency shift observed.

Neural/Activity for Phonation. A second explanation for the significant latency shift observed between the LO and VR conditions relates to the additional neural activity required for the phonatory task. In the VR condition the subjects were attempting to coordinate multiple respiratory, laryngeal, and articulatory structures for rapid phonation while still maintaining a relaxed physiological state during this preparation. This calls for rather complex levels of neural activity and interaction. Vigilance is also important in the VRT task and is unimportant in the LO task.

Additionally, the majority of this activity will be beginning or underway shortly after the initial recognition of stimulus signal onset. Since on a cortical level this is thought to be reflected by middle AER components and/or initial slow (N1) components, it places the bulk of the phonatory activity arising very close to

the average P2 response latency (around 175 to 200 ms). Since some of the stuttering and nonstuttering subjects had individual voice reaction time measures as early as 222 ms post-stimulus onset, that implies that the bulk of the neural activity for phonation occurred in the interval approximately between 100 ms (cortical identification of signal) to 220 ms (phonation onset) this would explain why no significant increase in latency was seen for the N1 component while a significant increase was observed for the P2 component. The results of the present study appear to support the contention that the addition of phonatory response requirements increases the latency of auditory processing activity.

P2 Identification Difficulty. As has been previously discussed, the P2-VR condition was the only wave/condition situation in which there was difficulty in identifying and determining the latency of an AER component. There is a question concerning if P2 was appropriately identified in the present study, primarily for the stuttering group.

A review of waveform morphology by the researcher for the seven affected subjects indicated that only the earliest occurring positive peak (after 150 ms) was selected as P2 for each of these subjects. Such a procedure should shorten, rather than falsely inflate, P2 values. This appears to be true, for only four of the

seven subjects involved had P2-VR latency increases greater than 30 ms, and this is less than one half of the total number of subjects with latency shifts of that magnitude (nine subjects). When examined in such a manner it would appear the P2-VR confusion did not provide a considerable contribution to the significant P2 latency shift observed.

The second way that the possible confusion in P2-VR identification may have affected the results of the present study is that auditory responses may differ in waveform morphology during phonatory trials and P2 may not be present for all subjects. It appeared to be clearly present for six of the eight nonstutterers; their waveform morphology was very similar in both conditions. Five of the eight stutterers had VR condition AER waveforms that did not resemble the P2 component waveform morphology obtained during their LO condition recording. If the P2 component had been misidentified in the VRT recordings, it therefore would have affected the latency measures of stutterers more than those of nonstutterers. This could have resulted in a difference between the two groups in latency measures. As will be discussed in the section of this chapter dealing with the final hypothesis, such a discrepancy between the groups was not present. It appears that the results of the present study support the contention that the addition of a phonatory response

requirement significantly increased auditory processing time, at least as measured by the later occurring exogenous AER component.

Hypothesis 5: AER Latency Shift Between Groups

The fifth and final hypothesis of the present study was that if a difference in latency was observed between the conditions due to the addition of a phonatory response, this shift in latency would be greater for the stuttering than for the nonstuttering group. This hypothesis was tested statistically for each of the two AER components and is discussed below.

N1 Latency Shift Between Groups

No statistically significant difference was found between the two conditions when the latency values for all subjects were combined together. A similar result was obtained when latency shift measures between the two groups were analyzed. There was not a significant difference between stutterers and nonstutterers in the amount of latency shift observed for the N1 component.

This result was not unexpected due to the lack of a significant difference in latency when the members of the two groups were combined. N1 latency shift measures for the two groups were very similar, with a difference in group means of less than one millisecond. Possible reasons for the lack of an increase in N1 latency measures

when the phonatory response was added were given in the previous section.

P2 Latency Shift Between Groups

There was a statistically significant difference between the two experimental conditions in P2 latency. The addition of a phonatory task did increase the latency of the P2 component. The results, however, do not support the fifth hypothesis, that such a latency shift would be more noticeable for the stutterers than for the nonstutterers. Mean P2 latency shift for the stutterers was 43.76 ms, and for the nonstutterers was 30.18 ms. The difference in means was not statistically significant. This may have been due to the large variability in latency measures for both groups (standard deviations were almost as large as group means) and also due in part to the difficulty in identifying the P2 component latency value for some of the subjects.

As was discussed earlier, there is some question if the values recorded for five stuttering subjects and two nonstuttering subjects are true and accurate measures of their P2 latency. It was mentioned in the previous section that due to the criteria used, the recorded values would more likely be underestimations of P2 latency rather than overestimations. There is a possibility that the mean P2-VR latencies for some of the stuttering group

might be even greater than recorded, and thus the difference in mean latency shift between the groups reported for the present study may be inaccurate. This, however, cannot be determined in the present study, and so the conclusion is that while an increase in P2 latency was observed when the phonatory task was added, there was no difference between the groups in the amount of this increase. Stutterers responded similarly to nonstutterers.

CHAPTER V

SUMMARY AND CONCLUSIONS

Summary

The purpose of the present study was to examine cortical auditory system activity of stutterers as a possible explanation of slower voice reaction time responses previously reported for stutterers. Mean latencies of the auditory electroencephalic responses (AER) N100 (N1) and P200 (P2) and mean voice reaction time (VRT) to an auditory stimulus were investigated for adult stutterers and normal speakers. Two conditions were utilized. One condition (AER-VR) involved recording AERs during a voice reaction time task. In this condition each subject phonated a previously specified speech sound (/^/) as quickly as possible upon hearing the onset of a 1000 Hertz (Hz) frequency tone. AERs were recorded for the 1000 Hz. stimulus tone. In the second condition, listening- only (AER-LO), AERs were recorded while the subjects listened to the same 1000 Hz. stimulus tone without producing a phonatory response. The subjects participating were eight stutterers and eight nonstutterers, with the two groups matched for age and sex. All subjects presented normal hearing sensitivity for pure tone auditory stimuli and exhibited no abnormal

neurological symptoms. All members of the stuttering group presented a childhood onset of stuttering.

Five hypotheses relating voice reaction time and auditory processing as measured by auditory electroencephalic response latency were tested in the present study. Single factor analysis of variance (ANOVA) computations were utilized to examine differences between stutterers and normal speakers for each of the factors VRT, AER N1 latency in the AER-LO and the AER-VR conditions, and AER P2 latency in each of the two response conditions. The difference in AER latency between the two conditions (referred to as latency shift) and the difference between stutterers and nonstutterers in latency shift was also examined through single factor ANOVA computations to determine the effect of a phonatory response on AER latency. Pearson Product-Moment Correlation Coefficients were calculated to examine the relationship between VRT and AER latency. A summary of these statistical treatments relative to the five hypotheses is presented below.

Hypothesis 1

The stuttering group will be slower than the nonstuttering group in initiating phonation to an auditory stimulus.

The stutterers in the present study were statistically significantly slower than the nonstutterers in voice reaction time, supporting the hypothesis. Additionally, the relationship between voice reaction time and stuttering severity was examined post-hoc. There was not a statistically significant relationship between VRT and severity of stuttering.

Hypothesis 2

Stutterers possess a less efficient auditory system.

As evidence of this, stutterers will exhibit longer N1 and P2 AER latencies to a pure tone stimulus in both AER-LO and AER-VR conditions.

The stuttering group in the present study did not exhibit significantly longer AER latencies in either condition. There was only one condition in which a statistically significant difference was present between groups. This was for the AER N1 response in the listening-only condition, and in this condition the stutterers had significantly shorter N1 responses than nonstutterers. The results of the present study do not support the hypothesis that stutterers have less efficient auditory processing, at least as indicated by N1 and P2 latency measures.

Hypothesis 3

There will be a relationship between auditory processing and voice reaction time. Subjects with later occurring N1 and P2 responses will display longer VRT than subjects with earlier occurring AER components.

There was only one statistically significant correlation when the VRT measures and the various AER measures for all subjects were compared. This was for VRT with the P2 component during the VR condition. The correlation coefficient was 0.39, indicating that the relationship was not robust. When the measures were compared by groups there were no statistically significant correlations for the stuttering group. The results of the present study do not support the hypothesis that voice reaction time is related to auditory processing efficiency.

Hypothesis 4

Speech is a neurologically demanding task. The addition of a phonatory response requirement will increase auditory processing time as indicated by an increase in N1 and P2 latencies when compared to those recorded in a passive listening situation.

There was not a statistically significant difference in N1 latency between the two conditions. The addition of the phonatory response requirement did not affect N1

latency. There was a statistically significant increase in P2 latency in the VR condition over that in the LO condition. The majority of preparatory phonatory activity in a VRT task would likely be occurring in the time interval after normal N1 occurrence and around the time of normal P2 occurrence. This result supports the hypothesis that auditory processing may be affected as the amount of neural demands increase.

Hypothesis 5

Stutterers may possess a less efficient neurological system than nonstutterers. The effect of the additional neural demands of phonation will have a greater effect on the stutterers than on the nonstutterers.

There were no statistically significant differences between stutterers and nonstutterers in the amount of latency shift observed between the listening-only and phonatory response conditions. The results of the present study do not support the hypothesis that stutterers are affected more than nonstutterers by the presence of added neural demands.

Conclusions

The results of the present study indicate that stutterers require a significantly longer time than

nonstutterers to initiate phonation to an auditory stimulus. As stutterers displayed a significantly shorter N1 response latency to an auditory stimulus in the listening-only condition, and no significant difference was observed between stutterers and nonstutterers for the P2 response latency in the listening-only condition, nor for either AER component in the voice reaction condition, it would appear that slower initial cortical-level auditory processing of the stimulus by stutterers would not be a feasible explanation for the VRT discrepancy. This conclusion is further supported by other results from the present study. No relationship between voice reaction time and the AER latencies in either condition was observed for the stuttering subjects. While the addition of a phonatory task did increase the latency of one of the AER components (P2) as hypothesized, there was not a significant difference between stutterers and nonstutterers in the amount of this increase. Relative to the type of auditory system activity examined, stutterers responded much the same as nonstutterers.

The results of the present study suggest that the slower VRT response in the stuttering group observed in the present study and in previous studies apparently arises from factors other than the auditory system. Such factors may be deficiencies or errors in motor program retrieval, motor programming, central-to-peripheral

efferent motor transmission, or in abnormal laryngeal position or activity. Additionally such nonneurophysiologically-based factors such as learned compensatory laryngeal patterns, hesitancy, or psychological factors may contribute to the VRT discrepancy between the groups.

While the results do not support the original contention that the observed difference in voice reaction time between the groups is attributable to inefficient auditory processing, one other significant finding in the present study does have implications for the role of central auditory impairments in stuttering. As was hypothesized, the additional demands of the rapid phonatory production in the VR condition significantly increased the latency of the P2 AER component. This hypothesis was developed from prior reports that indicated that the latency of some of the endogenous AER components increased as neural processing demands increased (Ritter, 1978; Teuting, 1978). While no difference between stutterers and nonstutterers in the amount of P2 latency shift was observed in the present study, a relatively simple processing task and a relatively simple phonatory task was involved. There are indications in the literature that stutterers may possess a less efficient neurological system (e.g., the central auditory processing research such as that of Curry and Gregory, 1969; Hall and

Jerger, 1978), or utilize a different neurological pattern (e.g., Moore and Haynes, 1980). If this is true, the shift in neural processing/neural efficiency observed in the present study may change dramatically when the demands of maintaining a normal conversational rate of speech are present. The stutterers with a less efficient or inadequate processing system would be affected to a much greater degree, and a breakdown in the ongoing flow of speech would not be unexpected. Therefore the significant shift in processing observed in the present study becomes relatively important. Apparently even relatively simple phonatory tasks place observable demands on the neural system. The demands of conversational speech may be excessive for some stuttering subjects.

Implications for Further Research

Auditory Processing and Stuttering

Further examination of the auditory system in stutterers is warranted. While the present study did not find stutterers to exhibit statistically significant longer auditory evoked responses than nonstutterers, this does not necessarily conflict with other results indicating poorer performance by stutterers on central auditory tests. The latter utilize more complex, generally verbal, stimuli which theoretically tax the neurologic system more than the pure tone stimulus

utilized in the present study. Examination of the later occurring endogenous AER responses, such as P300, to complex stimuli may give a more accurate indication of auditory system integrity as well as provide additional information on overall neurologic system efficiency.

Voice Reaction Time and Stuttering

Further exploration into voice reaction time measures of stutterers is also warranted. While the consistent indication of statistically significant differences between stutterers and normal speakers on voice reaction time is scientifically interesting, the consistent overlap of the two groups on VRT measures limits application of the information. Studies that identify subgroups of stutterers by voice reaction time latency and then correlate other physiological measures with performance of the groups may help to establish that etiological factors predisposing people to stuttering behavior may be different for different subjects. Indeed, a replication of the present study, utilizing only stuttering subjects with VRT slower than the group mean may yield more conclusive results concerning auditory processing and stuttering. Secondly, additional exploration of physiological and neurophysiological processes other than those of the auditory system may provide further information on the cause of the difference in phonatory

reaction time between stutterers and nonstutterers. Such exploration may also yield information relative to the presence of multiple deficits that could further delineate subgroups of stutterers and have implications on therapeutic techniques.

Differential Diagnosis of Etiological Components

Similarly, the lack of a significant correlation within the stuttering group on voice reaction time and severity of stuttering in the present study has implications for assessing therapy candidates and therapy techniques. Guitar and Bass (1978) indicated that factors such as initial severity of stuttering and the attitude of the stutterer are important for predicting success in therapy. The correlations were not perfect - one can't predict from severity measure alone. Results from the present study indirectly support this conclusion, for a few of the stutterers who were actually severe stutterers prior to therapy were experiencing a high level of fluency due to recent therapy when assessed for the study. The other factors involved in success in therapy may include the nature of the predisposing factors. The use of measures such as voice reaction time may delineate subgroups that demonstrate greater success in therapy not from lesser degrees of initial severity but rather from the existence of normal physiologic responses. Examples

of such measures include electromyography (Freeman and Ushijima, 1975), nasal videoendoscopic viewing of vocal fold activity (Conture, McCall and Brewer, 1977), and respiratory patterns (Watson and Alphonso, 1982). Indeed, the use of such measures may also provide an indication of optimal therapy techniques to utilize for each subgroup. Monitoring of performance on such measures may also provide an index of therapy success.

Neurological Demands and Stuttering

The indication from the present study that the application of additional neural demands slows neurologic response time is especially pertinent to the stuttering population. Much further exploration is needed in this area. The processing and motor tasks involved in the present study were relatively simple, and even allowed for some degree of preprogramming of responses. If such simple tasks increase neurologic response latency, the effect of extremely complex tasks such as conversational level speech production along with concomitant demands as a listener at times may overwhelm the neurologic systems of some stutterers. Monitoring of various neurologic responses such as the endogenous AER P300 as carefully selected hierarchies of demands, especially speech-related demands, are placed on subjects may provide valuable information delineating differences between stutterers and

nonstutterers. Such a technique could establish the complexity level at which the subject could no longer maintain fluent speech. This information would have definite therapeutic value.

Techniques such as described above would be especially beneficial in dealing with childhood stuttering. Bloodstein (1981) has indicated that tension and fragmentation of speech occurs when a child exceeds his/her linguistic, emotional, or physiologic capabilities. If critical levels for one or more of these areas could be established, and through child and family training be avoided, stuttering behaviors may never develop fully.

Response Variability and Stuttering

The issue of excessive variability in the physiologic responses of stutterers should be further explored. A recent work by Zimmerman (1980) has indicated that articulatory responses in stutterers may exceed normal acceptable range of motion and trigger abnormal reflex activity. Excessive response variability may act in the same manner, eliciting abnormal patterns or disturbing motor programming for coordinated ongoing activity. Investigation into the effect of altering response requirements such as simplifying and then increasing the complexity may demonstrate changes in the degree of variability. If such is the case, much beneficial therapeutic information would be gained.

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APPENDICES

APPENDIX A

STATEMENT OF INFORMED CONSENT

Informed Consent

You have been requested to take part in a research project through the Department of Audiology and Speech Pathology at the University of Tennessee. The purpose of this experiment is to measure how quickly you can react to the onset of a tone by producing the sound /ʌ/ ("uh"). Your brain's electrical activity will be measured during this task, very similar to the way that a doctor does an electroencephalogram test. It is necessary to place electrical receivers, called electrodes, on your head and face to record your brain's activity. To do this, the spot where the electrode is to be placed will be cleaned and the electrode will be temporarily applied with electrode paste. This provides a gentle bonding, and the electrode can be easily removed at any time with light pressure. The electrodes will be connected to a machine that records the electrical activity. There is no danger of electrical shock to you.

You will hear a tone, signaling you to respond, through a set of headphones. A contact microphone, to record your production of /ʌ/, will be placed against your neck. It is necessary that you remain very still during the testing, moving nothing except the parts of your body used in speaking. To aid you in keeping your jaw from moving excessively a small plastic disc, called a bite block, will be placed between your back teeth on one side.

The test session will last for about one hour. Of this, approximately 20 minutes will be needed for attaching the electrodes, 25 minutes for testing, and 15 minutes to remove the electrodes and all traces of them. If at any time during the test session you wish to cease your participation, simply indicate so to the examiner and the testing will be halted immediately. You may withdraw from this project at any time without penalty.

There are no known risks to you from the experiment, and your name will not be used in any discussion or publication of the results. The study is designed to investigate brain activity preceding speech in normal speakers and those with speech disorders. You will receive little or no benefit personally from this project. However it may provide information important in helping people with speech disorders.

If you have any questions please call or speak to:

Larry Molt or Dr. Harold L. Luper
Department of Audiology and Speech Pathology, The University of Tennessee
Knoxville, Tn. 37996-0740
Phone: (615) 974-5010 or 974-5019

I have read and understood the above information and agree to participate in the above described project.

Signature: _____ Date: _____

Please sign both consent forms. One will be collected by the examiner. Please retain the other one for your information. Thank You.

APPENDIX B

CASE HISTORY QUESTIONS

- I. Have you ever been diagnosed or considered yourself as a stutterer?

IF YES, ANSWER BOTH SECTIONS. IF NO, GO TO SECT. II.

1. If so, what person/profession made the original diagnosis?
2. At what age was this diagnosis made?
3. Do you remember what form/severity the stuttering took at that time?
4. Was any speculation made as to the cause?
5. Age therapy initiated/length of treatment.
6. Was therapy successful? Have you remained fluent?

II. Birth/Developmental History:

1. Any abnormalities in mother's pregnancy?
2. Any birth defects?
3. Ever seriously injured or severe shock?
4. Major operations?
5. Education completed/age?
6. Any developmental delays?
7. Any Physical disabilities?
8. Ever diagnosed/treated for mental/
9. psychological disorder?
10. Ever diagnosed/treated for any neurological/ seizure disorder?
11. Is anyone in your family considered to be a stutterer? Relationship?

APPENDIX C

ORAL MECHANISM EVALUATION FORM

ORAL MECHANISM EXAMINATION
For The Adult Neurologically Impaired
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	Instructions	Expectations	Appearance	Movement	Other
1. Facial/General					
Observe					
eyes					
forehead					
nasolabial fold					
expression					
drooling					
swallowing					
Test					
forehead wrinkling					
eye closing					
eyebrow raising					
swallowing (optional)					
2. Jaw					
Observe					
habitual posture					
Test					
repeated mouth					
opening/closing					
3. Lips					
Observe					
structure					
Test					
bilateral retraction					
(smile)					
unilateral retraction					
protrusion					
lip pressing					
4. Teeth and Gums					
Observe					
dentures					
teeth					
gums					
Test					
bite					
5. Tongue (crucial)					
Observe					
structure					
size					
fasciculation					
Test					
protrusion					
alternate/pro-					
trusion retraction					
lateralisation					
elevation					
alternate eleva-					
tion/depression					
6. Hard Palate, Laryngeal					
and Velopharyngeal					
Mechanisms					
Observe					
hard palate					
velum					
Test					
velar elevation					
on /a/					
sustained /a/					
loudness of sustained /a/					
pitch level of					
sustained /a/					
cheek puffing					
7. Diadochokinetic Rates					
Test					
rapid repetition /pa/					
/ta/					
/ka/					
/pataka/					
tap-tapper-tapping					
juice-juicy-juicier					
sit-city-citizenship					
8. Articulation/Voice					
Obtain brief speech					
sample and					
transcribe					
Observe					
prosody					
intelligibility					
pitch					
loudness					
voice quality					

APPENDIX D

TABLE D-1

MEAN VOICE REACTION TIME (VRT) VALUES, AND RANGE AND
STANDARD DEVIATION VALUES FOR INDIVIDUAL SUBJECTS
IN THE PRESENT STUDY

SUBJECT NUMBER	GROUP	MEAN VRT (ms)	RANGE (ms)	STANDARD DEVIATION
1	STUTTERER	320.16	252-432	38.4
2	STUTTERER	369.20	230-680	52.9
3	STUTTERER	383.65	252-597	64.8
4	STUTTERER	388.84	267-597	45.4
5	STUTTERER	464.86	222-627	62.1
6	STUTTERER	540.58	252-912	97.8
7	STUTTERER	543.72	342-822	80.1
8	STUTTERER	427.56	245-980	133.4
9	NONSTUTTERER	276.62	222-410	40.2
10	NONSTUTTERER	289.03	230-402	33.0
11	NONSTUTTERER	299.29	222-470	39.3
12	NONSTUTTERER	323.84	260-500	36.8
13	NONSTUTTERER	378.29	237-537	54.4
14	NONSTUTTERER	352.39	245-500	52.0
15	NONSTUTTERER	388.90	222-747	80.9
16	NONSTUTTERER	440.62	327-590	52.0

APPENDIX E

TABLE E-1

MEAN VALUES (IN MILLISECONDS) OF THE AUDITORY ELECTROEN-
CEPHALIC RESPONSES N1 AND P2 AND AMOUNT OF LATENCY SHIFT
(LS) BETWEEN LISTENING-ONLY (LO) AND VOICE REACTION (VR)
CONDITIONS FOR EACH SUBJECT IN THE PRESENT STUDY

SUBJECT	N1-LO	P2-LO	N1-VR	P2-VR	N1-LS	P2-LS
STUTTERERS						
1	95.00	181.25	104.30	254.30	9.30	73.05
2	95.00	170.00	99.20	200.40	4.20	30.40
3	87.50	181.25	91.20	173.70	3.70	-7.55
4	80.00	155.00	96.30	186.30	16.30	31.30
5	102.50	170.00	104.90	277.40	2.40	107.40
6	87.50	162.50	83.60	225.60	-3.90	63.10
7	80.00	188.75	101.20	239.95	21.20	51.20
8	72.50	185.00	73.40	185.90	0.90	0.90
NONSTUTTERERS						
9	87.50	200.00	112.60	206.35	25.10	6.35
10	87.50	192.50	86.50	187.50	-1.00	-5.00
11	98.75	177.50	95.20	200.45	-3.55	22.95
12	110.00	173.75	113.80	215.00	3.80	41.25
13	102.50	166.25	98.30	214.55	-4.20	48.30
14	87.50	177.50	108.30	205.80	20.80	28.30
15	106.25	192.50	105.50	218.00	-0.75	25.50
16	106.25	185.00	112.50	258.80	6.25	73.80

VITA

Lawrence Fraser Molt was born in St. Petersburg, Florida on March 13, 1953. He attended elementary and junior high school in that city and graduated from St. Petersburg Senior High School in June 1971. The following August he entered St. Petersburg Junior College, majoring in Psychology. He received an Associate of Arts degree, cum laude, in May 1973. Following a bicycle tour of Great Britian and western Europe, he enrolled in the combined undergraduate/graduate program in Speech-Language Pathology and Audiology at the University of South Florida (Tampa). He was named as a Dean's Outstanding Scholar for the 1973-74 academic year, and graduated with a Master's degree in June 1977. He was the first graduate from that program to complete a dual major in both Speech-Language Pathology and Audiology.

Lawrence Molt served clinical fellowship years for the two fields in the Brevard County Public School System (Titusville, Florida), first as a Speech-Language Pathologist for the hearing-impaired program (1977-78) and later as an Educational Audiologist (1978-79). He received the Certificate of Clinical Competency from the American Speech-Language-Hearing Association in Speech-Language Pathology in August 1978 and in Audiology in August 1979.

Lawrence entered the doctoral program in Speech and Hearing Science at the University of Tennessee (Knoxville) in September 1979. His major areas of study were speech science, neuroanatomy/physiology, dysfluency, and neuropathologies of speech and language. While at the University of Tennessee he served as a Speech-Language Pathologist and Clinical Supervisor for the Neuropathology Services Program. He received the Doctor of Philosophy degree in March 1985.

Lawrence became an instructor at the University of Utah (Salt Lake City) in January 1983. He is currently an Assistant Professor of Speech Science at that institution. He married Alexandra Dean Rowland in January 1977, and they have two children, Kristen Alexandra and Douglas Ryan.