PM-1 3½"x4" PHOTOGRAPHIC MICROCOPY TARGET
NBS 1010a ANSI/ISO #2 EQUIVALENT

**Speech-Motor Control and Interhemispheric Relations
in Recovered and Persistent Stuttering**

by

David C. Forster, B.Sc., M.Sc.

A thesis submitted to
the Faculty of Graduate Studies and Research
in partial fulfillment of
the requirements for the degree of

Doctor of Philosophy

Department of Psychology

Carleton University

Ottawa, Ontario

February 29, 1996

© copyright

1996, David C. Forster

National Library
of Canada

Bibliothèque nationale
du Canada

Acquisitions and
Bibliographic Services Branch

Direction des acquisitions et
des services bibliographiques

395 Wellington Street
Ottawa, Ontario
K1A 0N4

395, rue Wellington
Ottawa (Ontario)
K1A 0N4

Your file Votre référence

Our file Notre référence

The author has granted an irrevocable non-exclusive licence allowing the National Library of Canada to reproduce, loan, distribute or sell copies of his/her thesis by any means and in any form or format, making this thesis available to interested persons.

L'auteur a accordé une licence irrévocable et non exclusive permettant à la Bibliothèque nationale du Canada de reproduire, prêter, distribuer ou vendre des copies de sa thèse de quelque manière et sous quelque forme que ce soit pour mettre des exemplaires de cette thèse à la disposition des personnes intéressées.

The author retains ownership of the copyright in his/her thesis. Neither the thesis nor substantial extracts from it may be printed or otherwise reproduced without his/her permission.

L'auteur conserve la propriété du droit d'auteur qui protège sa thèse. Ni la thèse ni des extraits substantiels de celle-ci ne doivent être imprimés ou autrement reproduits sans son autorisation.

ISBN 0-612-13754-6

Canada

Name David C Forster

Dissertation Abstracts International is arranged by broad, general subject categories. Please select the one subject which most nearly describes the content of your dissertation. Enter the corresponding four-digit code in the spaces provided.

Psychology (Psychobiology)

SUBJECT TERM

03-19

U·M·I

SUBJECT CODE

Subject Categories

THE HUMANITIES AND SOCIAL SCIENCES

COMMUNICATIONS AND THE ARTS

Architecture	0729
Art History	0377
Cinema	0900
Dance	0378
Fine Arts	0357
Information Science	0723
Journalism	0391
Library Science	0399
Mass Communications	0708
Music	0413
Speech Communication	0459
Theater	0465

EDUCATION

General	0515
Administration	0514
Adult and Continuing	0516
Agricultural	0517
Art	0273
Bilingual and Multicultural	0282
Business	0688
Community College	0275
Curriculum and Instruction	0727
Early Childhood	0518
Elementary	0524
Finance	0277
Guidance and Counseling	0519
Health	0680
Higher	0745
History of	0520
Home Economics	0278
Industrial	0521
Language and Literature	0279
Mathematics	0280
Music	0522
Philosophy of	0998
Physical	0523

Psychology	0525
Reading	0535
Religious	0527
Sciences	0714
Secondary	0533
Social Sciences	0534
Sociology of	0340
Special	0529
Teacher Training	0530
Technology	0710
Tests and Measurements	0288
Vocational	0747

LANGUAGE, LITERATURE AND LINGUISTICS

Language	
General	0679
Ancient	0289
Linguistics	0290
Modern	0291
Literature	
General	0401
Classical	0294
Comparative	0295
Medieval	0297
Modern	0298
African	0316
American	0591
Asian	0305
Canadian (English)	0352
Canadian (French)	0355
English	0593
Germanic	0311
Latin American	0312
Middle Eastern	0315
Romance	0313
Slavic and East European	0314

PHILOSOPHY, RELIGION AND THEOLOGY

Philosophy	0422
Religion	
General	0318
Biblical Studies	0321
Clergy	0319
History of	0320
Philosophy of	0322
Theology	0469

SOCIAL SCIENCES

American Studies	0323
Anthropology	
Archaeology	0324
Cultural	0326
Physical	0327
Business Administration	
General	0310
Accounting	0272
Banking	0770
Management	0454
Marketing	0338
Canadian Studies	0385
Economics	
General	0501
Agricultural	0503
Commerce-Business	0505
Finance	0508
History	0509
Labor	0510
Theory	0511
Folklore	0358
Geography	0366
Gerontology	0351
History	
General	0578

Ancient	0579
Medieval	0581
Modern	0582
Black	0328
African	0331
Asia, Australia and Oceania	0332
Canadian	0334
European	0335
Latin American	0336
Middle Eastern	0333
United States	0337
History of Science	0585
Law	0398
Political Science	
General	0615
International Law and Relations	0616
Public Administration	0617
Recreation	0814
Social Work	0452
Sociology	
General	0626
Criminology and Penology	0627
Demography	0938
Ethnic and Racial Studies	0631
Individual and Family Studies	0628
Industrial and Labor Relations	0629
Public and Social Welfare	0630
Social Structure and Development	0700
Theory and Methods	0344
Transportation	0709
Urban and Regional Planning	0999
Women's Studies	0453

THE SCIENCES AND ENGINEERING

BIOLOGICAL SCIENCES

Agriculture	
General	0473
Agronomy	0285
Animal Culture and Nutrition	0475
Animal Pathology	0476
Food Science and Technology	0359
Forestry and Wildlife	0478
Plant Culture	0479
Plant Pathology	0480
Plant Physiology	0817
Range Management	0777
Wood Technology	0746
Biology	
General	0306
Anatomy	0287
Biostatistics	0308
Botany	0309
Cell	0379
Ecology	0329
Entomology	0353
Genetics	0369
Limnology	0793
Microbiology	0410
Molecular	0307
Neuroscience	0317
Oceanography	0416
Physiology	0433
Radiation	0821
Veterinary Science	0778
Zoology	0472
Biophysics	
General	0786
Medical	0760

EARTH SCIENCES

Biogeochemistry	0425
Geochemistry	0996

Geodesy	0370
Geology	0372
Geophysics	0373
Hydrology	0388
Mineralogy	0411
Paleobotany	0345
Paleocology	0426
Paleontology	0418
Paleozoology	0985
Palynology	0427
Physical Geography	0368
Physical Oceanography	0415

HEALTH AND ENVIRONMENTAL SCIENCES

Environmental Sciences	0768
Health Sciences	
General	0566
Audiology	0300
Chemotherapy	0992
Dentistry	0567
Education	0350
Hospital Management	0769
Human Development	0758
Immunology	0982
Medicine and Surgery	0564
Mental Health	0347
Nursing	0569
Nutrition	0570
Obstetrics and Gynecology	0380
Occupational Health and Therapy	0354
Ophthalmology	0381
Pathology	0571
Pharmacology	0419
Pharmacy	0572
Physical Therapy	0382
Public Health	0573
Radiology	0574
Recreation	0575

Speech Pathology	0460
Toxicology	0383
Home Economics	0386

PHYSICAL SCIENCES

Pure Sciences	
Chemistry	
General	0485
Agricultural	0749
Analytical	0486
Biochemistry	0487
Inorganic	0488
Nuclear	0738
Organic	0490
Pharmaceutical	0491
Physical	0494
Polymer	0495
Radiation	0754
Mathematics	0405
Physics	
General	0605
Acoustics	0986
Astronomy and Astrophysics	0606
Atmospheric Science	0608
Atomic	0748
Electronics and Electricity	0607
Elementary Particles and High Energy	0798
Fluid and Plasma	0759
Molecular	0609
Nuclear	0610
Optics	0752
Radiation	0756
Solid State	0611
Statistics	0463
Applied Sciences	
Applied Mechanics	0346
Computer Science	0984

Engineering	
General	0537
Aerospace	0538
Agricultural	0539
Automotive	0540
Biomedical	0541
Chemical	0542
Civil	0543
Electronics and Electrical	0544
Heat and Thermodynamics	0348
Hydraulic	0545
Industrial	0546
Marine	0547
Materials Science	0794
Mechanical	0548
Metallurgy	0743
Mining	0551
Nuclear	0552
Packaging	0549
Petroleum	0765
Sanitary and Municipal	0554
System Science	0790
Geotechnology	0428
Operations Research	0796
Plastics Technology	0795
Textile Technology	0994

PSYCHOLOGY

General	0621
Behavioral	0384
Clinical	0622
Developmental	0620
Experimental	0623
Industrial	0624
Personality	0625
Physiological	0989
Psychobiology	0349
Psychometrics	0632
Social	0451

The undersigned hereby recommend to
the Faculty of Graduate Studies and Research
acceptance of the thesis.

**SPEECH-MOTOR CONTROL AND INTERHEMISPHERIC RELATIONS
IN RECOVERED AND PERSISTENT STUTTERING**

submitted by

David C. Forster, B.Sc., M.Sc.

in partial fulfillment of the requirements for the degree of Doctor of Philosophy

Chair, Department of Psychology

Thesis Supervisor

External Examiner

Carleton University

April 8, 1996

ABSTRACT

The research investigated a dual factor model of the brain mechanisms underlying stuttering and extended it to account for spontaneous recovery from stuttering in children. The model attributes stuttering to the joint effects of a "fragility" in the neural mechanisms that underlie complex sequential motor control and an anomaly of interhemispheric relations. A series of four tasks were performed by 24 adult stutterers, 24 exstutterers and 24 participants who reported never having stuttered. Two of the tasks, a Sequence Reproduction Task and a Bimanual Crank-Turning Task, were intended to probe the one of the factors, the fragility of sequential motor control. Past research has shown adult stutterers to be impaired relative to nonstutterers on the Sequence Reproduction Task, particularly in terms of the time required to initiate tapping of the sequential responses. Other research has produced evidence of a bimanual coordination deficit in adult stutterers. The other two tasks, a Lexical Decision divided visual field task and a Dot Enumeration divided visual field task, were intended to assess the second factor in the model, the anomaly of interhemispheric relations. Previous reports in the literature have described a higher incidence of anomalous visual field asymmetries in stutterers on divided visual field tasks that employed linguistic stimuli.

Stutterers were impaired relative to nonstutterers on the two motor tasks, whereas exstutterers were indistinguishable from the nonstutterers on those tasks. However, both stutterers and exstutterers demonstrated anomalous visual field asymmetries, relative to nonstutterers, on the divided visual field tasks. The results are consistent with past research in support of the dual factor model. The pattern of results was also consistent with an extension to the model which attributes recovery from stuttering to maturation of neural mechanisms involved in the organization of complex sequential responses.

Acknowledgments

First, I would like to extend my very deep gratitude to the most important people involved in this research: the participants. I would also like to thank Carol Bock of the Communication Disorders department of the the Rehabilitation Centre in Ottawa, for her assistance in my search for participants.

Ikuko Webster patiently provided assistance with my statistical analyses on many occasions, and in doing so demonstrated an impressive ability to quickly come to terms with the complex issues and questions brought before her.

Barbara "Eagle-Eye" Westley kindly offered to proof-read both the original proposal, and the final thesis. I am grateful for that, and I am even more grateful for the support and encouragement I have received from her, and from the entire Westley family, over many years.

I am also grateful to Jim Campbell, who came to my assistance as a co-supervisor midway through my work on the thesis, and who saw me through some difficult times.

I would, of course, also like to extend my very deep appreciation to my supervisor, Prof. William Webster, for his guidance and for the patience he showed in maintaining a commitment to my doctoral work under very difficult, long-distance, circumstances.

Finally, I must acknowledge the most important people in my life. I would first like to thank my mother, and my entire family for the support and inspiration they have given me throughout an entire lifetime. This work, and the accomplishment that accompanies it, is in large part a realization of the values they instilled in me.

And last, but by *no* means least, I would like to thank my wife, Dr. Elizabeth Anne Westley, for the support, the patience and the love she has given me throughout the years that I have worked on this project. This is her accomplishment, as much as it is mine.

Dedication

This thesis is dedicated to my brother, Robin Forster. Knowing that there is no "Lorenzo's Oil" for stuttering, it was instead the hope that this work would do Robin proud that made the exercise as salient and as meaningful as it was for me.

Thank-you Robin

TABLE OF CONTENTS

ABSTRACT	ii
Acknowledgments	iii
TABLE OF CONTENTS	v
LIST OF TABLES.....	viii
LIST OF FIGURES.....	x
SPEECH-MOTOR CONTROL AND INTERHEMISPHERIC RELATIONS IN RECOVERED AND PERSISTENT STUTTERING	1
I. Introduction	1
The Syndrome of Stuttering	5
Prevalence and Recovery	9
Assumptions.....	13
II. Evidence of a Neurological Basis to Stuttering: Two Separate Factors	15
Speech-Motor Control in People Who Stutter.....	16
Interhemispheric Relations in People Who Stutter.....	34
III. The Dual Factor Model of Stuttering in Persistent and Transient Childhood Stuttering.....	70
Overview of the Dual Factor Model	70
Separability of the Factors	73
Developmental Implications of the Dual Factor Model	75
Rationale of the Research	87
IV. Summary of the Research and Hypotheses	87
Motor Tasks	87
Divided Visual Field Tasks	91

Summary of Tasks and Expected Results.....	93
METHOD.....	95
Participants	95
Materials	103
Overview of Procedure.....	103
Task 1: Sequence Reproduction Task	104
Apparatus	104
Procedure	105
Task 2: Bimanual Crank-Turning Task.....	107
Apparatus	107
Procedure	108
Tasks 3 and 4: Divided Visual Field Tasks.....	110
Apparatus	110
Procedure	111
RESULTS.....	114
Overview of Analyses.....	114
Sequence Reproduction Task.....	115
Bimanual Crank-Turning	122
Lexical Decision Task	135
Dot Enumeration Task.....	141
DISCUSSION	151
I Generalizing and Extending the Dual Factor Model in Adults.	151
(1). Replication and Generalization of Previous Findings.	152
(2). Separability of the Factors.....	164
(3). Nature of the Anomaly of Interhemispheric Relations	165

II. Recovery from Stuttering.....	168
Extending the Dual Factor Model to Account for Recovery	168
Classification of Exstutterers	172
III. Synthesis: The Dual Factor Model in Persistent and Transient Childhood	
Stuttering.....	175
Summary and Conclusions	182
REFERENCES	184
APPENDICES	222
Appendix A: Perceptions of Stuttering Inventory	222
Appendix B: Edinburgh Handedness Inventory.....	229
Appendix C: Likert Stuttering Scales.	231
Appendix D: Interview Database.....	232
Appendix E: Consent Forms.....	233
Appendix F: Debriefing Form	237
Appendix G. Protocol	239
Appendix H: Tapping Sequences for Sequence Reproduction Task.....	247
Appendix I: Stimulus Set for Lexical Decision Task	248

LIST OF TABLES

Table 1: Mean (and SD) Age and Education, and Percentage with a Positive Family History for Stuttering in Nonstutterers, Stutterers and Exstutterers.....	96
Table 2: Mean (and SD) Onset, Recovery and Likert-Scale Severity in Stutterers and Exstutterers.....	97
Table 3: Mean (and SD) Scores on Struggle, Expectancy and Avoidance Subscales of Perceptions of Stuttering Inventory for Nonstutterers, Stutterers and Exstutterers...	99
Table 4: Group Means (and SD) for the Three Dependent Measures Used in the Analysis of Sequence Reproduction Performance.....	117
Table 5: Group Means (and SD) for the Three Performance Measures Obtained in the Bimanual Crank Turning Task (With Visual Feedback).	126
Table 6: Group Means (and SD) for the Three Performance Measures Obtained in the Bimanual Crank Turning Task (No Visual Feedback).	127
Table 7: Group Means (and SD) as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Lexical Decision Task Collapsed Across Stimulus Onset Asynchrony.....	137
Table 8: Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Lexical Decision Task.	138
Table 9: Total Number of Individuals in the Three Groups with Left Visual Field (LVF) and Right Visual Field (RVF) Advantages on the Lexical Decision Task.....	140
Table 10: Group Means as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Dot Enumeration Task Collapsed Across Stimulus Onset Asynchrony.....	143
Table 11: Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Dot Enumeration Task.....	144

Table 12: Group Means as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Dot Enumeration Task with SOA = 50 ms.....	145
Table 13: Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Dot Enumeration Task with SOA = 50 ms.....	146
Table 14: Outcome of Planned Comparisons (<i>p</i> values) for Motor Tasks. <i>p</i> values are one- tailed for comparisons on the left, two-tailed for comparisons on the right.....	149
Table 15: Outcome of Planned Comparisons (<i>p</i> values) for Divided Visual Field Tasks. <i>p</i> values are one-tailed for comparisons on the left, two-tailed for comparisons on the right.....	150

LIST OF FIGURES

Figure 1. Childhood-version PSI scores as a function of Subscale and Group for exstutterers and stutterers.	100
Figure 2. Adulthood-version PSI scores as a function of Subscale and Group for all three groups.....	102
Figure 3. Mean (and standard error) Response Initiation Time (RIT) in ms as a function of Group and Sequence Length in the Sequence Reproduction Task.....	119
Figure 4. Mean (and standard error) Sequence Execution Time (SET) in ms as a function of Group and Sequence Length in the Sequence Reproduction Task.....	120
Figure 5. Mean probability of a Correct First Sequence (and standard error) as a function of Group and Sequence Length in the Sequence Reproduction Task.	121
Figure 6. Illustration of (A) a typical trace through the track and (B) a representation of the integrated area measure applied to the same trace.	124
Figure 7. Mean Integrated Error area (and standard error) as a function of Group and Lead Hand in the With-visual-feedback condition of the Bimanual Crank-turning Task.	130
Figure 8. Mean Percent Excess Output (and standard error) as a function of Group and Lead Hand in the With-visual-feedback condition of the Bimanual Crank-turning Task.	131
Figure 9. Mean Integrated Error area (and standard error) as a function of Group and Lead Hand in the No-visual-feedback condition of the Bimanual Crank-turning Task.	132
Figure 10. Mean Percent Excess Output (and standard error) as a function of Group and Lead Hand in the No-visual-feedback condition of the Bimanual Crank-turning Task.	133

Figure 11. Mean accuracy (d') (and standard error) as a function of Group and Visual Field in Lexical Decision Task. LVF = Left Visual Field, RVF = Right Visual Field.....	139
Figure 12. Mean (and standard error) visual field asymmetry in accuracy (d') as a function of Group and Stimulus Onset Asynchrony (SOA) in Dot Enumeration Task. LVF = Left Visual Field, RVF = Right Visual Field.....	147
Figure 13. Hypothetical influence of uncertainty about number of elements in sequence on Response Initiation Time in Sequence Reproduction Task.	155
Figure 14. Illustration of the neural amplification system that characterizes the relationship between the fragility of speech-motor control and the anomaly of interhemispheric relations in stuttering.	179
Figure 15. Hierarchical model illustrating the relationship between neuropsychological, psychosocial/affective, and developmental factors in the expression and modulation of stuttering.	181

SPEECH-MOTOR CONTROL AND INTERHEMISPHERIC RELATIONS IN RECOVERED AND PERSISTENT STUTTERING

I. Introduction

Stuttering is a disorder of speech production characterized by excessive and involuntary repetitions and prolongations of speech sounds. Throughout the past 50 years, stuttering has at various times been seen as a neurological disorder (Orton, 1937), a learned disorder (e.g. Sheehan, 1958), and a neurotic or psychosexual disorder (Coriat, 1927). More recently, the view that stuttering has a neurological basis has received renewed support (Andrews, Craig, Feyer, Hoddinott, Howie and Neilson, 1983; Jones, 1966; Moore 1984; Van Riper, 1982), though very little is known about the nature of the brain mechanisms involved. The present research tested a number of hypotheses relating to a neuropsychological model of stuttering. The model being investigated attributes stuttering to the joint effects of an anomalous pattern of interhemispheric relations and a fragility in the neural mechanisms underlying sequential motor control.

Forster and Webster (1991) outlined a model that attempted to identify the neuropsychological mechanisms that underlie stuttering in adults. According to that model, two conditions, both necessary but neither sufficient, interact with one another to give rise to the manifestation of stuttering in adulthood. One of these conditions is a *fragility in the mechanisms that underlie sequential motor control*. The mechanisms responsible for the organization and initiation of complex movement sequences appear to be unusually susceptible to interference from other ongoing neural activity. This could increase the frequency of momentary breakdowns in the necessarily precise timing of the multiple movement components involved in speech activity (Caruso, Abbs and Gracco, 1988; Webster, 1986a).

The second condition involves an *anomaly of interhemispheric relations*. While the nature of this anomaly is unclear, there is neuropsychological, electrophysiological and neuroradiological evidence suggesting that, under certain conditions, it manifests itself functionally in the form of elevated activation of the right cerebral hemisphere. Forster and Webster (1991) suggested that it is the relationship between the anomaly of interhemispheric relations and the otherwise subtle speech-motor fragility that gives rise to the comparably more profound disfluencies that characterize stuttering. Specifically, they argued that the anomaly of interhemispheric relations and the speech motor fragility exert a positive feedback relationship upon each other, leading to a "neural amplification system" capable of exacerbating the very mild speech-motor control deficit. This model will be described in more detail in part III of the Introduction.

The relationship between adult stuttering and childhood stuttering has received little attention in the literature on stuttering in general (e.g. Seider, Gladstien and Kidd, 1983; Sheehan and Martyn, 1970; Wingate, 1964a), and less in the literature on the neuropsychology of stuttering (Greiner and Fitzgerald, 1992; Sommers, Brady and Moore, 1975). Estimates suggest that up to 80% of children who stutter may spontaneously stop stuttering before they reach adulthood (Andrews *et al.*, 1983; Sheehan and Martyn, 1970). Furthermore, most estimates suggest that recovery almost always occurs during childhood or adolescence (Andrews, 1984) though a few recoveries probable do occur later in life (Shames and Beams, 1956; Sheehan and Martyn, 1970). Attempts to compare various characteristics of incipient or recovered stuttering to those in whom it persists into adulthood generally support the notion that recovered stuttering and persistent stuttering are related disorders (Joullian, Viswanath and Rosenfield, 1995; Seider *et al.*, 1983; Sheehan and Martyn, 1970; Wingate, 1964a). The question of what underlies this recovery and, more importantly, the failure to recover in persistent stuttering, is important both from a theoretical perspective and for its clinical implications.

The model proposed by Forster and Webster (1991) suggests a number of possible explanations for the apparently spontaneous recovery from stuttering that occurs in the majority of children who stutter. If that model is taken as one's theoretical perspective on childhood stuttering, then recovery suggests that either one or both of the conditions outlined in the model is eventually removed in the majority of cases (those in whom recovery occurs). One might speculate that this recovery is influenced, at least in part, by developmental mechanisms. If one of the two conditions outlined in the dual factor model were met in the course of normal development, then those children in whom the other condition was also met might be pre-disposed to stutter as children, but would recover as the first condition was removed through maturation of the mechanisms involved. Evidence will be reviewed in this Introduction suggesting that, while the anomaly of interhemispheric relations is not likely to be under developmental influence, the relevant speech-motor control mechanisms do seem to undergo maturation in childhood. We may thus speculate that *all* children go through a period in which their speech-motor control mechanisms are "fragile" in a manner similar to that which has been described for adults who stutter. If this were the case, then children who were born with an anomaly of interhemispheric relations would be predisposed to experience a transient period of stuttering in early childhood. That transient period of stuttering would resolve itself in late childhood as the neural mechanisms underlying speech-motor control matured. According to this idea, the stuttering will persist into adulthood *only in those children in which there is some permanent, presumably structural, anomaly in the mechanisms underlying speech-motor control.*

The present research had two main purposes. First, the research involved an attempt to replicate and generalize evidence surrounding the dual factor model as it relates to persistent stuttering in adults. A second purpose was to extend the dual factor model to address the issue of recovery from childhood stuttering.

Four different tasks were chosen, two of which were designed to assess the fragility of sequential motor control and two of which were intended to assess the anomaly of interhemispheric relations. The four tasks were administered to three groups of participants: (1) adults who stutter; (2) adults who stuttered as children but no longer stutter; and (3) a control group with no history of speech disfluency. According to the model of recovery summarized above, both recovered and persistent stutterers¹ should show evidence of an anomaly of interhemispheric relations, while only the persistent stutterers should exhibit evidence of the fragility of sequential motor control.

In addition to the hypotheses outlined above, the research offers an opportunity to provide a further test of the dual factor model as it relates to persistent stuttering in adults. This was accomplished in a number of different ways. First, the research necessitated an attempt to replicate and expand upon evidence previously obtained with persistent adult stutterers. Second, the research afforded an opportunity to address the important issue of whether the two components of the model are neurologically distinct or whether they are different manifestations of a common underlying condition. Third, the research involved an attempt to further explore the anomaly of interhemispheric relations in adults who stutter. Each of these issues will be discussed at greater length in the sections of the Introduction that follow.

¹ The term “stutterer” (and, by extension, “exstutterer” and “nonstutterer”) is justifiably considered to be pejorative by some people who stutter. In this thesis, the term will be avoided wherever linguistic clarity allows. Both “stutterer” and “person who stutters” are defined operationally as a person who considers him- or herself to be someone who stutters. Definitional issues will be discussed further in the following section.

The Introduction is comprised of four major sections. The first section provides an introduction to the syndrome of stuttering. Emphasis will be placed on the issues of definition and epidemiology, as these are the most relevant to the present research. In addition, a number of important assumptions underlying the research will be discussed. These assumptions form the basis of the conceptual framework upon which the research is based.

The second section will review evidence for each of the two components of the dual factor model. The anomaly of interhemispheric relations and the fragility of speech-motor control will be discussed separately in this section, and it will be argued that both conditions are present in adults who stutter.

The third section will discuss the relationship between the two conditions in both persistent adult and transient childhood stuttering. The dual factor model will be described, both in its current state and in terms of the proposed extension of the model to account for recovery in transient childhood stuttering.

The fourth, and final, section of the Introduction will provide an overview of the research and a summary of the hypotheses that were tested by the research.

The Syndrome of Stuttering

Stuttering is most commonly defined simply as a disorder of speech production characterized by involuntary repetitions and prolongations of sounds (Andrews *et al.*, 1983). While this definition has served adequately in many contexts, agreement on a formal definition of the disorder has been more difficult. The first standard definition, provided by Wingate (1964b), described three basic features of stuttering. The first of these was "a disruption in the fluency of verbal expression, characterized by involuntary, audible or silent, repetitions or prolongations" in speech, which are frequent, marked in character, and not readily controllable. The second characteristic of stuttering is the

presence of accessory activities involving the speech apparatus, the facial muscles and other body parts that accompany speech disfluencies. According to Wingate (1964b) these activities give the appearance of a "speech-related struggle." The third characteristic is the presence of a negative emotional state, ranging from tension and anxiety to fear, embarrassment and frustration.

More recently, Andrews *et al.* (1983), in a major review of research on stuttering, highlighted the disagreement about a definition of stuttering but summarized the situation by saying that "there is a consensus that repetitions and prolongations are necessary and sufficient for the diagnosis" of stuttering (p. 227). This definition, particularly the presence of the word "sufficient" and the lack of the word "involuntary" provoked a great deal of controversy (Bloodstein, 1990; Ingham, 1990; Perkins, 1983; 1990a; 1990b; Smith, 1990; Wingate, 1984; Martin, 1986). Perkins (1983; 1986; 1990a) has suggested that the crucial element of any valid definition of stuttering is the term "involuntary," referring specifically to "the temporary overt or covert loss of control of the ability to move forward fluently in the execution of linguistically formulated speech" (Perkins, 1984, p. 431). While disfluencies also occur frequently in the speech of "fluent" speakers, and these disfluencies are not voluntary by any definition, Perkins (1984) argues that these disfluencies occur as a normal part of the flow of fluent speech and that they constitute neither a loss of control nor a blockage in the flow of speech.

Another key element of Perkins' (1984) definition, which has profound clinical and theoretical implications is the notion that disfluencies may be covert; thus the reliable identification of stuttered speech by an independent observer is rendered nearly impossible. This view is clearly supported by the data of Martin and Haroldson (1986), who demonstrated that independent observers typically identify more instances of stuttering in the speech of a stutterer than does the stutterer himself. This could be due to the fact that the stutterer and the observer have different definitions of what an instance of stuttering is.

The observer likely listens for repetitions and prolongations of sound, while the stutterer is likely sensing moments of loss of control of their speech, and ignoring disfluencies that occur without this sensation. In fact, a recent report suggests that even the stutterer himself may have difficulty identifying his own moments of stuttering reliably when listening to recordings of his own speech (Moore and Perkins, 1990). These findings cast doubt on the validity of any operational definition that depends on the reliable identification of moments of stuttering by independent observers.

If we accept Perkins' (1984) definition of stuttering, then it becomes clear that we would be best to define stuttering operationally as whatever the person who stutters defines as stuttering. Similarly, we should define a person who stutters (or stutterer) as one who considers himself or herself to be a person who stutters (or stutterer). Perhaps, as Perkins (1983) stated it, focusing on objective reliability to the exclusion of validity makes us "not much better than the kindergarten children who vote to determine whether the hamster is pregnant" (p.248). Bloodstein (1981) would appear to agree with that sentiment:

If we want to be guided by a more "objective" definition we must not ask questions about "stuttering" but about repetitions, prolongations, broken words, speech rate, and the like and must be content with answers that are not about stuttering, but about repetitions, prolongations and so forth. (Bloodstein, 1981, p. 9)

The loss of control to which Perkins (1984) referred may vary greatly in its manifestations, including undetectable "covert" disfluencies, overt repetitions and prolongations of sounds, and severe blockages that can last for seconds or even minutes in the most extreme cases. People who stutter describe these occurrences by saying they feel "stuck," that they simply cannot get started because something is interfering with airflow or phonation (Van Riper, 1982). It is during blockages that the struggle to which Wingate (1964b) referred is most visible. Tremor, overflow movements, clenching of teeth,

blinking, facial contortions and even limb movements may be associated with stuttering (Van Riper, 1982; Wingate, 1964b).

The type of struggle described above is clearly a negative emotional experience. While the disorder manifests itself in the speech system as dysfluency, in the person, it is manifested as fear and anxiety. The fear may be directed outward. It can be a fear of a particular phoneme, or word, or it can be a fear of a situation. However, the most pervasive fear may be one that is directed within. This is the fear of a loss of control (Perkins, 1983), or "verbal impotence" (Van Riper, 1982). The ability to predict which words or phonemes will trigger a disfluency and the ability to sense an impending loss of control can affect the speech of the person who stutters in another way: avoidance. Avoiding words or sounds must inevitably force the speaker to substitute synonyms, to circumlocute, or to avoid speaking situations altogether (Bloodstein, 1981). At this level, stuttering does not simply affect speech fluency but impairs communication and social interaction. Van Riper (1982) and Carlisle (1985) have discussed the dread that may become associated with speaking situations in people who stutter. This dread may lead to an individual that is psychosocially paralyzed -- one that is unable, or at least unwilling to engage in certain social activities (Carlisle, 1985; Van Riper, 1982; Webster, 1989).

There is another aspect of this dread which may ultimately be critical to a full understanding of the mechanisms that underlie stuttering. Some authors (e.g. Bloodstein, 1984; Sheehan, 1958) have considered stuttering to be a learned disorder. While it is not the intention here to review the literature on stuttering as a learned behaviour, there is one learned aspect to the overall syndrome that is both self-evident and of theoretical interest to the present discussion. The fear surrounding specific situations and speech sounds that have become associated with stuttering in an individual might give rise to a conditioned emotional response. These sounds or situations may, after repeated pairings with the pain and embarrassment of stuttering, ultimately end up acting as noxious conditioned stimuli

themselves regardless of the speaker's degree of fluency at any given moment in time. As will be discussed later, there is evidence that negative emotions activate specific brain structures, and may activate the right hemisphere preferentially. This provides a means by which the psychosocial and neurological aspects of stuttering could be related. Stuttering may thus be seen as a complex multidimensional syndrome, rather than simply a disorder of speech-motor control, a learned disorder or an emotional disorder.

The World Health Organization (1980) classified an impairment as the anatomical, physiological or psychological loss or reduction of structure and/or function in a tissue, organ or functional system affected by a disease. A disability was defined as the effect of the impairment on the normative functional adaptation of the person, representing the objectification of impairments in the everyday activities of the individual. Finally, a handicap is seen as the disadvantage conveyed by the disability on the life adjustment of the person in his or her social environment (see The Council of Europe, 1989). The syndrome of stuttering fits well into this framework. The impairment in stuttering is the disruption of normal speech-motor fluency. The functional manifestation of this impairment is interference with speech fluency, resulting in a difficulty with interpersonal communication; this is the disability of stuttering. This disability may lead to a social handicap if the person withdraws from any aspect of social contact as a result of his or her disability.

Prevalence and Recovery

The most widely accepted estimates suggest that the lifetime risk of stuttering is around 4-5% (Andrews and Harris, 1964; Andrews *et al.*, 1983; Bloodstein, 1981; Cooper, 1972; Porfert and Rosenfield, 1978). The prevalence of stuttering in the population is believed to be roughly 1% (Andrews and Harris, 1983). The large discrepancy between the lifetime risk and the prevalence of stuttering is related to the fact that a large percentage of those who are ever diagnosed with stuttering appear to

spontaneously stop stuttering (Andrews and Harris, 1964; Cooper, 1972; Cooper and Wells, 1974; Panelli, McFarlane, and Shiply, 1978; Porfert and Rosenfield, 1978; Sheehan and Martyn, 1966; 1970). Although the issue of recovery has considerable implications for the diagnosis, classification and treatment of stuttering, as well as for our understanding of the mechanisms that underlie it, little research has focused on the factors that determine the life history of stuttering

The figures cited above are estimates. Given the lack of agreement on an appropriate definition for stuttering, it is perhaps not surprising that there is also a lack of consensus on issues so basic as the approximate incidence, prevalence, lifetime risk and the rate (or even existence) of spontaneous recovery from stuttering. Figures for lifetime risk range from 0.93% (Cooper and Wells, 1974) to 10% (Dickson, 1971). Recovery rates range from 44% (Cooper, 1972) to 80% (Andrews and Harris, 1964; Panelli, McFarlane and Shipley, 1978; Sheehan and Martyn 1966; 1970) - ignoring, of course, those who dispute that spontaneous recovery takes place at all (e.g. Ingham, 1983).

Among those who agree that spontaneous recovery from stuttering does take place, there is a surprising degree of satisfaction with the fact that a figure of 80% has been frequently obtained (e.g. Andrews *et al.*, 1983; Sheehan and Martyn, 1970; Panelli *et al.*, 1978), in spite of wildly divergent paths taken to reach this figure. Andrews and Harris (1964) found that most recovery takes place before adolescence, and virtually all recoveries take place before age 16. On the other hand, Sheehan (1979) notes that while 80% of his subjects had reported recovery, 51.5% recovered after the age of 15. Meanwhile, Panelli *et al.* (1978) observed 80% recovery in children between ages 2 and 7. The largest sample size was used by Sheehan and Martyn (1970), but Andrews and Harris (1964) and Panelli *et al.* (1978) both used a more rigorous longitudinal design.

Ingham (1983) has argued that the figures of 80% are too high, and highlights a number of methodological problems with the studies on spontaneous recovery. Ingham

(1983) suggests that the figure should be in the 30-50% range, though it is unclear how this figure is derived. He casts doubt on the notion that any recovery is spontaneous, arguing that it connotes some kind of "mystical intervention." (p. 114). More specifically, he argues that,

...the role of therapy in recovery studies, whether self-managed or clinician managed, appears to have received only cursory consideration. Obviously, the very notion of 'spontaneous recovery' becomes meaningless if recovery is associated with treatment, regardless of who directs it. (p. 121-122)

The argument that the role of therapy in recovery should receive more than cursory consideration is certainly a valid one. Nevertheless, most speech pathologists consider therapy to be a means of management, rather than a cure for the disorder. While it is clear that treatments for stuttering can lead to significant improvement in fluency (e.g. Andrews *et al.*, 1983; Andrews, Guitar and Howie, 1980; R.L. Webster, 1980; Woolf, 1967), it is also clear that relapse following treatment is a common experience (Boberg *et al.*, 1979; Mowrer, 1975).

The definition of stuttering provided by Perkins (1984) may be relevant to this discussion. Most of the studies of recovery from stuttering have employed retrospective designs, relying on the respondents' memories, and, perhaps most importantly, their own definition of stuttering. There is clearly no therapeutic means available today that could offer the promise of eliminating the tendency to lose control of one's speech. Until such an intervention is found, it seems that the likelihood that any recovery from stuttering was due to a therapeutic intervention is rather slim. When a person states that they once stuttered, but that they no longer do, it seems fair to assume that they no longer lose control of the automatic or conscious control of speech.

The assumption in this thesis will be that most cases of reported recovery from stuttering are ultimately based in constitutional factors rather than therapeutic intervention.

whether clinician-managed or self-managed. Some interesting, and perhaps relevant, statistics provide some support for this notion. First, the likelihood of recovery is clearly associated with the severity of stuttering. For example, Sheehan and Martyn (1970) found that 87%, 74% and 50% of mild, moderate and severe stutterers had reported recovery, respectively. This is in spite of the fact that, respectively, only 23%, 50% and 50% of the same groups had received therapy. In addition, a substantially higher proportion of those who had received no therapy (88%) had recovered, as compared to those who had received therapy (71%). We need not conclude from this finding that therapy is in fact an impediment to recovery. It is important to note that the severe cases of stuttering are the ones most likely to undergo therapy. We may, however, conclude that, even if therapy provides a means to manage stuttering, it is not related to an increased likelihood to recover. We may also infer from these data that the fact that an individual who recovers from stuttering has received any kind of therapy certainly does not necessarily imply that the therapy caused the recovery.

The notion of spontaneous recovery does not need to imply any sort of "mystical intervention." Since stuttering appears to be (as will be discussed below) an incompletely penetrant genetic disorder, its expression is clearly influenced by environmental factors. If we accept that stuttering has a neurobiological basis, then its expression could be modified by the well-documented changes that take place in the central nervous system over the course of development. In particular, it has for some time been well known that the brain mechanisms underlying motor control (Connolly and Stratton, 1968; Denkla, 1973; 1974; Fagard and Pezé, 1992; Wolff, Gunnoe and Cohen, 1983), speech and language (Basser, 1962; Kolb and Fantie, 1989), and interhemispheric communication (Jeeves, Silver and Milne, 1988; O'Leary, 1978; Salami, 1978) undergo development throughout early childhood. While any relationship between development, normal or disrupted, and

stuttering is hypothetical at this point, there is certainly no basis for the position that such a relationship is impossible.

Assumptions

The most basic assumption underlying the research is the assumption that stuttering is primarily a disorder of speech fluency. Related to this assumption is the belief that the psychosocial aspects of the stuttering syndrome are secondary to the impairment of speech fluency.

A second assumption is that the impairment in speech fluency has an organic basis. First, stuttering runs in families. The prevalence of stuttering among first degree relatives of stutterers is near 15%, which is greater than three times the rate in the general population (Andrews *et al.*, 1983). Furthermore, stuttering not only runs in families, but is related in a very specific way to the sex of both the proband and the proband's relatives (Kidd, 1984; Kidd, Heimbuch and Records, 1981). At the highest risk are male relatives of females who stutter, and at the lowest risk are female relatives of males who stutter (Kidd *et al.*, 1981; Kidd, Kidd and Records, 1978). The familial patterns that have been observed in families that contain stutterers are consistent with a sex-modified vertical transmission of susceptibility to stuttering (Kidd *et al.*, 1981). While this does not necessarily prove a genetic mode of inheritance, the consistent familial patterns that have emerged are consistent with a number of single-gene or polygenic genetic models, but are *not* consistent with most cultural or environmental explanations. Finally, the best evidence in support of an organic basis to stuttering is the genetic evidence from twin studies: monozygotic twins have a considerably higher concordance for stuttering than dizygotic twins (Howie, 1981; Andrews, Morris-Yates, Howie and Martin, 1991). These data, in combination with the familial patterns described above, provide very strong evidence of a genetic basis for stuttering. While any of the above patterns might be explainable by a sufficiently ambitious

environmental or cultural explanation, the notion that a genetically-based, though incompletely penetrant, organic mechanism underlies stuttering is much more consistent with the range of currently available evidence.

The third assumption underlying the research is that the organic mechanisms responsible for stuttering reside in the central nervous system or, more precisely, the brain. As will be reviewed throughout this paper, there is a large body of evidence suggesting that people who stutter differ from fluent speakers on a number of neurological or neuropsychological characteristics that appear to reflect constitutional differences, albeit subtle ones. This includes behavioural evidence of anomalous interhemispheric relations (e.g. Travis, 1978; Moore, 1984; Webster, 1988) and impairments in speech-motor programming (Caruso, Abbs and Gracco, 1988; Zimmerman, 1980), neuroradiological evidence (e.g. Pool, Devous, Freeman, Watson and Finitzo, 1991; Watson, Pool, Devous, Freeman and Finitzo, 1992; Watson, Freeman, Devous, Chapman, Finitzo and Pool, 1994; Wood *et al.*, 1980), and electrophysiological evidence (Moore, 1984; 1986; Pool, Finitzo and Freeman, 1988).

A fourth assumption underlying the research is that there is a subset of children who stutter who will undergo spontaneous recovery at some point before they reach adulthood. It is important to distinguish between recovery, which implies that the individual no longer experiences genuine loss of control of speech, and management or coping. The latter may involve any number of coping mechanisms, ranging from beneficial ones (i.e. those that form the basis of therapeutic interventions) to maladaptive ones (i.e. avoidance of words or speaking situations).

Finally, the model of recovery being investigated here is based upon the tentative assumption that transient childhood stuttering is related, neurologically, to persistent stuttering in adults. There is currently no empirical basis for the alternative possibility that transient childhood stuttering is unrelated to persistent stuttering in adults. The assumption

is difficult to test directly in children, however, since there is presently no reliable means of predicting which children who stutter will continue to stutter into adulthood. One means of testing the assumption is to examine the family histories of recovered and persistent stutterers. For example, Sheehan and Martyn (1970) reported that the familial incidence of stuttering did not differ between recovered stutterers and persistent stutterers, but was higher in those groups than in normal controls. Seider *et al.*, (1983) reported a higher frequency (7%) of recovered stutterers among the first-degree relatives of persistent stutterers than has been reported in the general population (approximately 3%). Both these findings support the hypothesis that recovered and persistent stuttering are related disorders. Furthermore, Wingate (1964a) collected information on a broad range of issues related to the speech and the background of 50 recovered stutterers, and reported that "the information supplied by these recovered stutterers is similar to the findings reported in the literature as typical of stutterers."

II. Evidence of a Neurological Basis to Stuttering: Two Separate Factors

The notion that two separate neurological anomalies contribute causally to stuttering is central to the dual factor model. The evidence for each of these two anomalies comes from entirely separate traditions of stuttering research, and each has typically been studied and discussed separately from the other. On the one hand, there has been a long tradition of studying speech-motor control in stuttering, and many prominent speech-language pathologists have concluded that a deficit in the sequencing or timing of speech movements is at the core of the disorder (Andrews *et al.*, 1983; Caruso, Abbs and Gracco, 1988; Travis, 1982; Van Riper, 1982; Zimmerman, 1980; 1984). On the other hand, there has also been a strong tradition in speech-language pathology and neuropsychology of studying hemispheric specialization and interhemispheric relations in people who stutter, and there is an entirely separate class of theories which have attempted to account for stuttering in terms

of those constructs. It will be argued here that there is merit to both positions, and that a broader account of the literature on stuttering necessitates an effort to explore possible relationships between these two different anomalies. In the remainder of this section, each of the two anomalies described above will be discussed separately. The section that follows will describe the dual factor model in terms of the relationship between the two anomalies and other factors which influence stuttering.

Speech-Motor Control² in People Who Stutter

Evidence of Fragility of Sequential Motor Control

A number of authors have suggested that a basic feature of stuttering is a difficulty in generating the complex sequences of movements inherent in speech motor control. Kent (1984) suggested that "a primary difference between stutterers and nonstutterers lies in their capacity to generate temporal programs, or time structures of action" (p.283). MacKay and MacDonald (1984) called stuttering a "sequencing and timing disorder." Van Riper (1982) also considered stuttering to be, at its root, "a disorder of timing," and suggested that "when a person stutters on a word, there is a temporal disruption of the simultaneous and successive programming of muscular movements required to produce one of the word's integrated sounds..." (p. 415).

Webster (1985; 1986a; 1989) demonstrated that the sequencing deficit in people who stutter may not be limited to the speech domain. Typically, studies of motor coordination in stuttering have been limited to analyses of speech and speech-motor control

² The term "speech-motor" control is not intended to imply that the mechanisms involved are limited to the speech domain. On the contrary, the motor anomalies in stuttering to be discussed below are not limited to speech. They involve processes, such as the organization of sequential responses, that are critical to both speech and nonspeech movements.

(e.g. Caruso, Abbs and Gracco, 1988; Rastatter and Dell, 1987; Watson and Alfonso, 1987; Zimmerman, 1980; 1984). However, as early as 1957, Seth reported that stutterers differed from nonstutterers on a motor task (finger-tapping) that did not involve speech. It is important to note that people who stutter do not demonstrate any evidence of gross motor coordination deficits outside the speech domain. Cox (1982) compared stutterers to fluent controls on a number of standardized neuropsychological tests. In this study, the stutterers actually had substantially higher finger-tapping rates than fluent controls for both the dominant and nondominant hands. This task, while being relatively sensitive to gross brain damage, reveals little about the complex sequential motor control mechanisms involved in speech, since it involves a single repetitive movement with no sequential component.

Webster (1985) had stutterers and nonstutterers perform two finger tapping tasks: a repetitive index finger-tapping task, and repetitive sequential finger-tapping task. Both groups tapped more quickly on both tasks with their right hand. The stutterers tapped as quickly as the nonstutterers on both tasks. However, while the total number of errors on the repetitive sequential finger-tapping task was small for both groups, the stutterers made approximately twice as many errors as nonstutterers. These results were taken to indicate that stuttering is not associated with a general problem with the sequencing and timing of motor behaviours. The higher error rate in the stutterers was tentatively attributed to an age covariate which, when entered into the analyses, eliminated the group difference with respect to errors. However, the results of a later study suggest that the difference in error rates may have been related to a genuine group difference. Webster (1986a) had stutterers and fluent speakers perform a finger tapping task similar to the one described above. However, in this experiment, response times to initiate the response sequences were also assessed. The sequences were presented visually on a display panel, and the participants were instructed to begin tapping as quickly as possible after presentation of the last element

in the sequence. Once again, overall finger-tapping rates did not differ between the two groups. However, the stutterers performed fewer correct sequences over the duration of the trial, made substantially more errors on the first sequence, and had slower overall response onset times. Webster (1986a) suggested that the mechanisms responsible for the organization and initiation of complex movement sequences may be unusually susceptible to interference from other ongoing neural activity, possibly from the contralateral (right) hemisphere, in people who stutter. Webster (1989) later obtained similar results in a similar sequence reproduction task in which subjects were encouraged to take as much time as necessary to initiate the novel response sequences. The stutterers still had a much higher probability of an error in the first sequence, and performed fewer overall correct sequences than the nonstutterers. These results suggest that the speeded nature of Webster's (1986a) Sequence Reproduction Task was not necessary to demonstrate the difficulty with the initiation of sequences. Rather, the novel nature of the responses appeared to be a more critical factor.

The mechanisms underlying speech-motor control in people who stutter are not grossly deficient. As demonstrated by Webster (1985; 1986a) people who stutter can generate complex manual sequential responses as rapidly as fluent speakers. They do, however, generate more errors, and have greater difficulty initiating novel sequences, as manifested both in the accuracy and the onset latency of those sequences. Both of these findings are consistent with the observable characteristics of stuttering. They are also consistent with the findings of Zimmerman (1980) and Caruso *et al.* (1988), who both noted abnormalities in the fluent speech in stutterers. Zimmerman (1980) examined the movements and timing of the lips and jaws during the fluent utterances of the syllables /pap/, /mam/, and /bab/. in stutterers and fluent speakers. The stutterers' utterances had a number of anomalies including longer onset latencies, slower voice onsets and slower movement transition times. They also demonstrated greater variability in the timing of lip

and jaw movements. This latter finding is consistent with the findings of Caruso *et al.* (1988), who recorded upper lip, lower lip and jaw movements during the /p/ sound in the nonsense word "sapapple." Once again, only fluent utterances were studied. Fluent speakers consistently demonstrated an invariant "upper lip - lower lip - jaw" ordering, while stutterers had highly variable sequencing of those same sounds. Caruso *et al.* (1988) studied the movement velocities and amplitudes as well as the sequencing of the various component movements. The anomaly they noted was specific to the sequencing component; movement velocities and amplitudes were similar for the two groups. This specificity of the anomaly in stutterers' fluent speech was considered to be suggestive of a neural impairment at a specific level of the speech-motor control hierarchy. Since the anomalies were noted in fluent utterances, Caruso *et al.* (1988) hypothesized that they might reflect an "inherently unstable situation, perhaps indicative of underlying neuromotor control problems" in people who stutter.

Webster (1986b) obtained evidence consistent with the hypothesis that the sequential response mechanisms in people who stutter are particularly susceptible to interference from ongoing activity in the contralateral hemisphere. Subjects performed the repetitive sequential finger-tapping task with one hand concurrently with a stimulus-contingent knob-turning task in the other hand, and found that the secondary task performed by the contralateral limb interfered with the tapping task more in the stutterers than in the nonstutterers. Once again, this is compatible with the idea that the sequential response mechanisms in people who stutter are particularly susceptible to interference from activity in the contralateral hemisphere. Also consistent with that notion were the results of a study that involved concurrent bimanual handwriting (Webster, 1988). Subjects were asked to copy lists of numbers with their two hands simultaneously, and the letters produced by the two hands were compared. Stutterers' letters were more poorly formed and, perhaps most significantly, included more mirror-image letter reversals with their left

hand than nonstutterers. Similar results were reported by Greiner, Fitzgerald and Cooke (1986). The presence of mirror-reversals is strongly suggestive of interhemispheric overflow or interference. Webster (1988) suggested that these results supported the "ungated callosal function" hypothesis; the idea that each hemisphere is particularly susceptible to interference via the corpus callosum from homologous regions in the contralateral hemisphere.

However, neither study (Webster, 1986b; 1988) specifically compared the effects of ipsilateral versus contralateral concurrent activity. One difficulty in making such a comparison is the fact that two concurrent manual activities will necessarily involve contralateral cerebral hemispheres because of the crossed connectivity of our nervous system. A second motor task that could offer a comparison between ipsilateral versus contralateral concurrent activity would be necessary, however, in order to test the hypothesis that the sequential response mechanisms are particularly susceptible to contralateral hemispheric activity in people who stutter.

An experiment by Forster and Webster (1991) allowed such a comparison. A repetitive sequential finger-tapping task was performed by one hand, while a stimulus-contingent foot-responding task was performed by one foot. By comparing ipsilateral and contralateral limb pairs, this task allowed a comparison of intra- versus inter-hemispheric interference on the sequential response mechanisms. This comparison is based in part on the functional cerebral space model of Kinsbourne and Hicks (1978). The functional cerebral space model hypothesizes that concurrent cortical activities will interfere with each other to a degree that is related to the "functional cerebral distance" between the processing centres for the two functions. Functions that are mediated by cortical regions that are physically close to one another will interfere with each other to a greater extent than functions that are mediated by more distant regions. Kinsbourne and Hicks (1978) demonstrated that speech (localized in the left hemisphere) interferes with a right-handed

unimanual activity to a greater extent than left-handed activity. Similarly, Forster and Webster (1991) hypothesized that ipsilateral hand-foot activity would lead to greater interference than activities by contralateral hand-foot pairs. In addition, the degree of interference by contralateral limb pairs would involve a callosal gating component, whereas ipsilateral limb pairs would not. If people who stutter have a selective callosal gating deficit, they would be expected to exhibit selectively greater interference, as compared to fluent speakers, under the contralateral limb pairs conditions.

The results of this study did not confirm the ungated callosal function hypothesis (Forster and Webster, 1991). It was found that, while ipsilateral limb combinations did lead to greater interference than contralateral limb combinations in both groups (in support of the functional cerebral space hypothesis of Kinsbourne and Hicks, 1978), stutterers did not experience proportionally greater interference than nonstutterers. In fact, stutterers did not demonstrate greater interference under either condition than nonstutterers. This is in contrast to the findings of Webster (1986b; 1988). There are a number of possible explanations for this. First, the susceptibility to interference apparent in the earlier studies may have been more general, rather than involving a selective callosal gating deficit. It might have been the case that the concurrent foot-responding task involved neural systems that were so anatomically distant from the sequential response mechanisms that they did not lead to neural interference as such. The interference that was observed in both groups could still be explained by a limited resource model (e.g. Kahneman, 1973).

A second possible explanation is that the interference that was demonstrated by Webster (1986b; 1988) could be more specific rather than more generalized. Since the earlier studies involved concurrent bimanual activity, the two channels interfering with one another were connected directly via the corpus callosum, rather than by the diffuse spread of activation through the cerebral cortex as described by the functional cerebral space hypothesis (Kinsbourne and Hicks, 1978). This is presumably a fundamentally different

kind of interference, possibly related to specific neural mechanisms underlying bimanual coordination. One advantage of this explanation is that it accounts for the lack of elevated interference in stutterers in the study by Forster and Webster (1991). Since that study did not involve bimanual activity, one would not expect stutterers to demonstrate higher overall levels of interference

Summary: The term "fragility" has been used because it conveys the two principle elements of the proposed speech motor deficit in people who stutter. The mechanisms for sequential motor control are not "damaged" or lacking in people who stutter. Rather, they are, according to the hypothesis, unusually susceptible to interference, leading to an increased tendency to experience momentary breakdowns in the fluent sequencing of complex sequential motor processes. Evidence has been reviewed which illustrates an increased tendency to produce errors in manual sequential processes, difficulty initiating complex manual sequences, and anomalous speech-motor sequencing in the fluent speech of people who stutter. Additionally, evidence has been presented which demonstrates greater interference in people who stutter in bimanual concurrent task performance but not in concurrent hand-foot activity. It was suggested that the elevated interference observed by Webster (1986b; 1988) may be related to the mechanisms specific to bimanual coordination rather than to either a generalized susceptibility to interference or a general callosal gating deficit.

The following section will discuss a possible neural basis for the fragility of sequential motor control.

Speech-Motor Control and the Supplementary Motor Area

A number of investigators (Caruso *et al.*, 1987; McClean, 1990; Pool, Freeman and Finitzo, 1988; Pool *et al.*, 1991; Van Lieshout, Hulstijn and Peters, 1996; Watson *et al.*, 1994; Webster, 1988) have independently suggested that stuttering may be associated with some structural anomaly involving the supplementary motor area (SMA). The evidence

implicating the SMA in stuttering will be discussed in this section. Prior to that discussion there will be a review of evidence relating to the SMA's role in a variety of functions.

The SMA is a region of the cerebral cortex that is situated on the mesial surface of the frontal lobes, anterior to the primary motor area (M1) and above the cingulate cortex. The SMA corresponds roughly to the cytoarchitectonic region defined as Brodmann's area 6. While the SMA is arranged in a crude somatotopic manner (Fried *et al.*, 1991), electrical stimulation evokes complex movements such as orienting of the body or closing of the hand (Fried *et al.*, 1991; Penfield and Rasmussen, 1950). The SMA receives extensive subcortical projections from the basal ganglia (particularly the globus pallidus) via the thalamus (Schell and Strick, 1984; Tokuno, Kimura and Tanji, 1992). In addition, the SMA has extensive reciprocal corticocortical projections with the anterior premotor area, M1, and posterior parietal cortices (Goldberg, 1985; Tanji, 1994).

Functions of the Supplementary Motor Area (SMA)

The supplementary motor area is believed to be involved in the programming of complex movement sequences (Goldberg, 1985; Grafton, 1994; Rao *et al.*, 1993; Roland, Skinhøj, Larsen and Lassen, 1977; Shibasaki *et al.*, 1993; Tanji, 1994). Simple motor tasks, such as maintaining compression of a spring between the thumb and forefinger are associated with an increase in blood flow activity in the contralateral primary motor and somatosensory cortices, but not in the SMA (Roland *et al.*, 1977). More complex tasks involving a sequential component show similar increases in those regions, but are also associated with elevated SMA activity bilaterally (Rao *et al.*, 1993; Roland *et al.*, 1977; Shibasaki *et al.*, 1993). In fact, simply rehearsing the sequence mentally without actually executing it is also associated with increased SMA activity, suggesting that the SMA is involved in the preparation of the sequential responses (Roland, Skinhøj, Larsen, and Lassen, 1980; Rao *et al.*, 1993; Roland, 1984a; 1984b). This is consistent with electrophysiological evidence in humans (Deecke, 1987; Lang, Obrig, Lindinger, Cheyne

and Deecke, 1990) and monkeys (Romo and Schultz, 1987; Tanji, Tanaguchi and Saga, 1980) which demonstrates that cells in the SMA fire well in advance of movement initiation. Roland (1984b) suggested that the SMA is responsible for "the formation of a motor subroutine specifying the sequence of isolated (ballistic) movements" (p. 209).

While it is clear that the SMA plays a role in the preparation of complex sequential motor responses, the exact nature of that role has been a source of debate recently (see Goldberg, 1985, and commentary). Various terms have been used to describe the SMA's role in the generation of motor responses, including "intention," (Deiber *et al.*, 1991; Fried *et al.*, 1991; Goldberg, 1985) "preparation," (Neafsey, 1985) and "initiation" (Freund, 1984; Rostomily, Mitchel, Berger, Ojemann, and Lettich, 1991).

Goldberg (1985) noted that the SMA is situated at the interface between limbic outflow and the motor system, and suggested that it may be involved in the translation of motive to intention and intention to action. Deiber *et al.* (1991) reported findings consistent with that idea. They measured activity in cortical areas while subjects performed various motor tasks involving moving a joystick in response to a tone. The tasks varied in terms of the required direction of movement, and the basis for movement selection. Internal cues, where subjects could prepare the response in advance of the trigger stimulus (the tone), and external cues, where the direction of movement was contingent on the nature of the tone, were used. While movement selection was invariably associated with elevated SMA activity, the largest increases were associated with a mode of movement selection based on internal cues. The internal cues involved either a fixed condition (the same direction each trial), a learned order, or a "random" order of response directions from trial to trial. The "random" order involved having the subject choose any direction on any given trial, with the stipulation that there should not be long sequences of trials with movements in the same direction. The greatest activation over the SMA was associated with the random condition, which was presumably the one in which the nature of the response was the most internally

guided. Deiber *et al.* (1991) speculated that the increased SMA activity that was observed in the trials where internal cues were used for movement selection reflects preparatory activity of the SMA during the waiting period between trials. This would be consistent with SMA activity during mental rehearsal of movements (Rao *et al.*, 1993; Roland *et al.*, 1980) and well (up to 2 seconds) before initiation of movements that are executed (Deecke, 1987; Romo and Schultz, 1987; Tanji, Tanaguchi and Saga, 1980). It is also consistent with the observation that stimulation of SMA cells, while it most often leads to execution of complex movements, occasionally leads only to an "urge" to perform a movement, or an anticipation that a movement is about to be evoked (Fried *et al.*, 1991).

The supplementary motor area also appears to play a central role in bimanual coordination (Goldberg, 1985; Tanji, 1994). The central location of the SMA, its strong reciprocal interhemispheric connections and the lack of similar interhemispheric connectivity in M1 (Rouiller *et al.*, 1994), and its relation to other components in the motor system, all suggest that it would be well situated to play an important role in the coordinated integration of the activity of the two limbs. The observation that the SMA appears to always be activated bilaterally (Roland, 1984a; 1984b) is consistent with a central role in the bilateral coordination of movement. The primary motor area is usually activated only (or predominantly) contralaterally in association with unilateral movements of the extremities (Ikeda *et al.*, 1995; Rao *et al.*, 1993; Shibasaki *et al.*, 1993) but bilaterally in association with movements of the bilaterally distributed speech apparatus (Petersen, Fox, Posner, Mintun and Raichle, 1988; Roland *et al.*, 1980; Roland, 1984a; Wohlert, 1993). Fried *et al.* (1991) reported that unilateral stimulation of the SMA was capable of eliciting unilateral and bilateral movements, particularly when the right SMA was stimulated. In interpreting this observation, they noted that one of the most persistent deficits associated with SMA damage in humans is a deficit in bimanual coordination (Freund, 1985; Laplane, Talairach, Meininger, Bancaud, and Orgogozo, 1977). Finally,

Tanji, Okano and Sato (1988) identified cells in the SMA that were active specifically in association with either unilateral left or right, or bilateral hand movements in monkeys. Cells in M1 responded in association with contralateral hand movements, regardless of whether they occurred in isolation or in the context of bimanual movements. Thus, the activity of the SMA cells appeared to be related to the *type* (unimanual or bimanual) of movement selected and performed, whereas the activity of M1 cells was related simply with activity of the contralateral muscles.

It is tempting to view bimanual coordination as a special case or subset of the SMA's more general role in the sequencing of complex movements. In a coordinated act involving the two limbs, the two limbs could be seen as separate muscle groups whose activity must be precisely sequenced (Peters, 1983). Lang *et al.* (1990) reported findings which suggest that the SMA is important in the temporal programming of bimanual movements. They measured cortical potentials while musicians performed two bimanual tapping tasks. One task simply involved tapping the two index fingers in synchrony. The other task involved a more complex rhythm where two taps from one hand were synchronized with three taps with the other hand. The latter task is extremely difficult for non-musicians to perform because it involves generating competing temporal patterns for the two limbs. Musicians, on the other hand, should be aware of the overall rhythmic pattern that the two sequences would generate when performed simultaneously, making performance of the task easier. Lang *et al.* (1990) found that, while the simple task was associated with activation of the two primary motor cortices, the complex task was associated with activation in both the primary and supplementary motor areas. Once again, activation over the SMA preceded movement onset by as much as four seconds. Lang *et al.* (1990) concluded that the SMA is responsible for the initiation of movements in which the individual components must fit into a precise timing plan. Their observations suggest that the SMA is important for bimanual coordination where the activity of the two limbs

must be precisely coordinated. As indicated by the lack of SMA activity in the simple task, the SMA was not necessary when those temporal demands were not present.

While one may view bimanual coordination as merely a subset of a general sequential or temporal programming role, the bilateral symmetry of our paired limbs does carry with it a unique set of demands. Brinkman (1981) noted an interesting deficit of bimanual coordination in monkeys in which the SMA had been lesioned unilaterally. The monkeys had been trained preoperatively to retrieve a morsel of food placed in a hole in a transparent plastic plate. Normal monkeys did this by pushing it through the hole with a finger of one hand and catching it on the other side with the other hand. Monkeys with lesions of the SMA would use both hands to push the food through the hole in a mirror-symmetric fashion, causing it to fall to the floor. Perhaps most interestingly, when the corpus callosum in these monkeys was subsequently sectioned, they were once again able to successfully perform the bimanual task (Brinkman, 1984). A similar deficit was noted by Chan and Ross (1988) in a Chinese man with unilateral right SMA damage. This individual demonstrated a tendency to produce mirror-symmetric movements when asked to either perform or mime various activities. For example, when pretending to steer a car, both hands would move in an arc either up or down toward each other. He would often, when eating, pick up a bowl or a pair of chopsticks with both hands simultaneously in a mirror-symmetric fashion, rather than holding the bowl in his left hand and the chopsticks in his right hand. This phenomenon is very reminiscent of the behaviour of Brinkman's (1981; 1984) monkeys.

Also reminiscent of Brinkman's data are the findings of Pascual-Leone, Cohen, Wassermann and Hallett (1994), who used transcranial magnetic stimulation to disrupt SMA and M1 activity while intact humans performed a bimanual tapping task. The participants played a bimanual sequence of three piano keys in either a mirror-symmetric or a parallel manner. Stimulation of M1 resulted in a delay of subsequent taps, but did not

otherwise interfere with bimanual coordination. Stimulation of SMA resulted in asynchronous key presses in both versions of the task, and was associated with sequencing errors, frequently mirror reversals, in the parallel version of the task.

Preilowski (1972) reported a study of the effect on bimanual coordination of sectioning the anterior corpus callosum. He used a bimanual crank-turning task, where the subjects were required to turn two cranks with their two hands in order to move a "pen" on an X-Y recorder. Each crank controlled movement of the pen in either the up-down or left-right dimension, much like the "Etch-a-Sketch" toy. The task involved manipulating the pen through a narrow track oriented to require asymmetric output from the two hands. Individuals whose anterior corpus callosum had been sectioned (for the control of severe epilepsy) were impaired on the task in terms of both speed and accuracy, when compared to normal and epileptic controls. In addition, when visual feedback was removed after considerable practice, the partial callosal patients could not maintain the asymmetric output, indicating that their movements were under closed-loop visual guidance. In contrast, the controls were able to maintain reasonably good performance if visual guidance was removed midway through a trial. Preilowski (1972) suggested that the posterior corpus callosum was important for the visual guidance of the bimanual movements in the partial callosal patients. In support of this notion, Preilowski (1975) noted that complete callosotomy virtually abolished any ability to perform the task. Thus, it appears that an intact anterior corpus callosum is necessary to allow for the kinesthetically based feed-forward mode of bimanual control necessary for the rapid and more accurate performance observed in normal subjects after practice (Preilowski, 1972; 1975). Goldberg (1985) described a very similar mode of control in his review of the SMA's functions. He referred to the SMA's mode of control as "projectional," relying on "model-based prediction." Each constituent movement in such a control system is essentially ballistic rather than being guided by sensory feedback. Goldberg (1985) noted that this is a feed-forward type of

control that uses kinesthetic information primarily, and contrasted it to a "lateral" system that is feedback-based, and more reliant on visual information.

The mode of control described by Goldberg (1985) in the context of the supplementary motor area has also been described by a number of authors who were studying speech-motor control (Abbs and Gracco, 1983; Gracco, 1988; Gracco and Abbs, 1983; Shaiman, 1989). Speech-motor control is clearly dependent on kinesthetic information, and is executed in a predictive or feed-forward manner. The execution of individual movements is essentially ballistic, but the overall process is sensitive to sensory, particularly kinesthetic, inputs (Abbs and Gracco, 1983). Compensatory responses based upon kinesthetic activity would thus be available to the system, but would only be called upon when "functionally necessary" (Shaiman, 1989).

It should not be surprising, given the similarity between the nature of speech-motor control and the type of control exerted by the supplementary motor area, that the SMA has also been implicated in speech-motor control. One of the earliest reports of the SMA's unique properties was Brickner's (1940) report that electrical stimulation of Area 6 (now considered to be part of the SMA) led to "perseveration of speech." The stimulation was applied while neurosurgical patients were reciting the alphabet. Upon stimulation, the patient would repeat the letter he was on over and over until the stimulation was ceased.

Electrophysiological and neuroradiological evidence also suggests that the SMA plays an important role in the complex timing inherent in speech-motor control. Electrical stimulation of the SMA in awake patients has been shown to cause speech arrests and hesitations of speech (Fried *et al.*, 1991; Brickner, 1940). A number of authors have also reported that stimulation of the SMA (usually the left side) leads to vocalizations (Fried *et al.*, 1991; Morris, Dinner, Luders, Wyllie and Kramer, 1988; Penfield and Welch, 1951). Supplementary motor area activation always occurs bilaterally during speech (Larsen,

Skinhøj and Lassen, 1978), and appears prior to initiation of the speech (Deecke, Kornhuber, Lang, Lang and Schreiber, 1985; Wohlert, 1993).

Damage to the supplementary motor area has been shown to disrupt speech in a variety of ways. There is typically a lack of or poverty of spontaneous speech, described as a partial mutism rather than an aphasia, since the structure of speech remains intact (Goldberg, 1985; Rostomily *et al.*, 1991). Attempts to initiate speech are laboured, and are associated with a struggle and with hesitations (Goldberg, 1985; Jonas, 1981; Rostomily *et al.*, 1991). Speech disruptions usually are associated with damage to the SMA in the dominant hemisphere, though if the damage is unilateral, they are generally transient, gradually improving from mutism, to nonspontaneity to hesitant speech (Rostomily *et al.*, 1991).

It is interesting to speculate about the relationship between bimanual coordination and speech-motor control. Control of the speech apparatus necessarily involves the precise integration of the commands reaching each half of the tongue, lips, jaw, and larynx. As discussed above, the SMA and M1 are activated bilaterally in association with both speech and nonspeech movements of the speech apparatus. The precise bilateral integration of the efferent signals from these cortical areas would be critical to the generation of fluent speech, and would presumably be best carried out by a single unilateral control center. As will be discussed below, Brain (1945), Peters (1976), Bishop (1990) and others have hypothesized that this demand is what led to the development of cerebral dominance for speech. Yet unilateral cortical control of speech must eventually be mapped onto a bilateral efferent system, and it is possible that the SMA plays an important role in this regard. Just as the SMA's role in the temporal programming in speech may be a subset of a general feed-forward mode of control, its role in the bilateral integration of the activity of the speech apparatus may be a subset of a general role in bilateral control. This would include the SMA's well-documented role in bimanual coordination.

Supplementary Motor Area and Stuttering

In light of the supplementary motor area's proposed functions, the notion of an anomaly of SMA functioning being associated with stuttering has considerable face validity. In particular, the SMA's role in the preparation and initiation of complex movement sequences, and its specific role in speech-motor control, is consistent with the most basic feature of stuttering - a difficulty with the initiation and motor timing of speech utterances. Many of the characteristics of SMA damage - laboured speech, hesitations and difficulty initiating speech - bear similarities to those involved in stuttering. While there is no evidence that people who stutter have gross brain damage in the SMA or any other area (Cox, 1982), the notion of a "fragility" in the supplementary motor area's functioning is consistent both with the basic features of stuttering and a small but growing body of empirical evidence (Caruso *et al.*, 1988; Pool *et al.*, 1987; Pool *et al.*, 1991; Webster, 1988).

As reviewed earlier, there is a growing consensus amongst those who consider stuttering to be an organic disorder that it involves a disorder of the necessarily precise sequencing and timing inherent in speech-motor control (Caruso *et al.*, 1988; Kent, 1984; MacKay and MacDonald, 1984; Van Riper, 1982; Webster, 1986a). Caruso *et al.* (1988) noted a selective deficit in the sequencing of the multiple movement components involved in fluent utterances. As has been discussed, temporal programming of complex sequential responses appears to be the one of the specific roles of the supplementary motor area. Caruso *et al.* (1988) noted this, and concluded that "... impairments in stutterer's abilities for timing/sequencing argue for consideration of the premotor cortical areas, particularly the SMA, as areas potentially involved in the disorder of stuttering" (p. 450).

Electrophysiological and neuroradiological evidence appears to support the hypothesis that stuttering is associated with aberrant SMA functioning. Pool *et al.* (1987) used brain electrical activity mapping to measure cortical activity of stutterers. They

reported abnormalities in the electrical activity over frontal midline regions, and suggested that they reflected "electrical dysfunction" in the supplementary motor area. That study, however, contained a number of weaknesses, including a lack of a control group of fluent speakers, no description of the conditions under which EEG measurements were taken, and a small number of subjects. In addition, the stutterers were grouped with individuals with spasmodic dysphonia, another speech-motor disorder which is similar but nevertheless distinct from stuttering. In spite of those weaknesses, the findings and conclusions of Pool *et al.* (1987) were provocative. More recently, Pool *et al.* (1991) used regional cerebral blood flow to study cortical activity in 20 stutterers as well as 78 age- and gender-matched control subjects. The measurements in this study were performed during rest, with no communication between the subjects and the experimenters. Nevertheless, a variety of anomalies were observed in the cortical activity of the stutterers compared to the control subjects. Pool *et al.* (1991) noted that blood flow was globally reduced in stutterers compared to the control subjects. They also noted flow asymmetries below the median (indicating left hemisphere hypoperfusion) in the anterior midline region (suggestive of SMA involvement) in nine out of 10 severe stutterers. Pool *et al.* (1991) concluded that their results were consistent with "classic anatomoclinical principles of speech motor control" which would suggest SMA involvement in stuttering.

Webster (1988) referred to the SMA as a possible locus of the speech-motor fragility in stuttering based upon the bimanual coordination deficits demonstrated by stutterers on the bimanual handwriting task (Greiner *et al.*, 1986; Webster, 1988). In those studies, stutterers demonstrated a stronger tendency to generate mirror reversals with their nondominant hand when rapidly tracing the letters with their two hands simultaneously, without visual feedback. As discussed above, one of the roles of the supplementary motor area appears to be bimanual coordination, and specifically the inhibition of mirror movements (Brinkman, 1981; 1984; Chan and Ross, 1988; Pascual-Leone *et al.*, 1994).

While the tendency of the stutterers to generate mirror-symmetric movements was extremely subtle by comparison with those described by Brinkman (1981; 1984) and Chan and Ross (1988), it nonetheless indicates a subtle deficit in bimanual coordination, specifically in the inhibition or non-mirror transformation of movements by the nondominant hand. One may also speculate that the observation of increased concurrent task interference in people who stutter when bimanual tasks were used (Webster, 1986b; 1988) and the lack of elevated interference when a hand and foot were used (Forster and Webster, 1991) point specifically to the mechanisms for bimanual motor coordination, rather than to generalized concurrent task interference.

Also interesting are the electromyographic data of Travis (1934), which were originally used to support the cerebral dominance hypothesis of stuttering. As will be discussed below, Travis (1934) measured the electromyographic activity of the speech musculature during stuttering, and noted asynchronies in the activation of the bilaterally paired muscles. This is consistent with the idea of a deficit in the bilateral coordination of the speech apparatus. As noted previously, the production of fluent speech presumably requires the precise temporal integration of the activity of the two sides of the speech apparatus. Bilateral coordination may be one aspect of the important role the SMA plays in the overall sequential programming of speech tasks.

Summary: The supplementary motor area appears to play a variety of roles in speech-motor control, including: 1) temporal programming of multiple movement components involved in complex sequential responses, including speech; 2) a preparatory role in the organization and initiation of complex movements possibly involving the translation of motive to intention, and intention to action; and 3) bimanual or bilateral coordination. The first of these roles, temporal programming of sequential responses, has been related to stuttering as the primary deficit underlying the disorder, and has been shown to be disrupted in the fluent speech and in manual performance. The second role,

organization and initiation of responses is clearly disrupted in stuttering, as manifested by hesitations and blockages in speech, and has also been shown to be impaired in the manual performance of people who stutter. Finally, the third function, bimanual coordination, has also been shown to be disrupted in people who stutter (Greiner *et al.*, 1986; Webster, 1986b; 1988).

The notion of a problem with supplementary motor area functioning appears to offer an explanation of the phenomenon of covert stuttering. Perkins (1984; Moore and Perkins, 1990) has argued that the defining characteristic of stuttering is a loss of control of the speech apparatus. He also stressed the existence and importance of covert stuttering to the disorder. As discussed earlier, covert stuttering involves the sensation of a loss of control, but one that is not manifested as actual disfluent speech (i.e. is not detectable to a listener), perhaps because the momentary loss of control is overcome, or because the individual circumvents or avoids the sound which is associated with it. The supplementary motor area is active well in advance of movement onset, and it may be active for movements that are planned but never executed. It is thus plausible that a problem with SMA could manifest itself in terms of a phenomenon such as covert stuttering, in addition to the more overt characteristics of the disorder.

Interhemispheric Relations in People Who Stutter

The general notion of "interhemispheric relations" is intended to encompass a broad range of issues surrounding the structural and functional relationships between the two cerebral hemispheres. The term is deliberately imprecise, in part because of the diversity of the theories that are typically included within the general rubric of interhemispheric relations, and in part because the precise nature of the anomaly in stuttering remains elusive in spite of the research that has been conducted.

One may partition the broad area of interhemispheric relations into three areas of interest: (1) structural theories of hemispheric specialization; (2) theories of hemispheric activation and hemispatial attention; and (3) theories of interhemispheric communication and interaction. The following sub-sections will provide explicit reviews of the first two areas. Because the issue of interhemispheric communication is difficult to disentangle from other issues surrounding the structure and function of the two cerebral hemispheres, a number of references to that topic will appear throughout the remainder of this section.

Structural Theories: The Cerebral Dominance Hypothesis

Interest in a possible association between interhemispheric relations and stuttering began early in this century with the cerebral dominance hypothesis (Orton, 1934; Travis, 1937). The cerebral dominance hypothesis was a structural theory of the first type mentioned above, and attributed stuttering (and other developmental disorders) to a diminished gradient of hemispheric specialization. Since modern research on interhemispheric relations in stuttering has its historical origins in the cerebral dominance hypothesis, it provides a useful starting point in the discussion of interhemispheric relations.

In 1912, Ballard demonstrated that left-handed school children had a four-fold higher incidence of stuttering than did right-handed school children. Quinan (1921) later reported that three times as many nonright-handed than right-handed children stuttered. The idea that abnormal patterns of handedness and, by implication, cerebral dominance are associated with stuttering provided the basis for the cerebral dominance theory of Orton (1937) and Travis (1934).

Orton (1937) considered poor lateralization for speech and language to be a causal factor in a number of developmental disorders, including dyslexia (which he called developmental alexia; see also Orton, 1925), developmental agraphia, developmental word deafness, developmental motor aphasia, and stuttering. Since the work of Orton (1937),

various authors have attempted to implicate atypical patterns of cerebral dominance with a variety of developmental disorders, including developmental language disorders (Springer and Eisenson, 1977) and autism (Colby and Parkinson, 1977) in addition to stuttering (Jones, 1966;1967) and dyslexia (Orton, 1937; Kershner and Morton, 1990). Yet, as Bishop (1990) suggests, "stuttering is perhaps the one (condition) where a theoretical explanation in terms of disordered laterality seems most plausible." Brain (1945), in a review of the available evidence on cerebral asymmetry, argued that it would be important to have unambiguous unilateral control of the finely graded and precisely timed activity of our bilaterally paired speech apparatus. This argument was more recently articulated by Peters (1976), and remains compelling today. It follows from that argument that a breakdown in the bilateral integration (i.e. resulting from competition between bilateral speech centres) of those bilaterally paired structures would lead to a breakdown in the flow of speech.

The cerebral dominance hypothesis of stuttering as initially stated by Travis (1934), is the notion that the speech apparatus of the person who stutters is constantly receiving competing signals arising from speech centers in the two cerebral hemispheres, rather than from one dominant hemisphere. In support of this idea, Travis (1934) reported that stutterers had abnormalities in the simultaneous activation of their bilateral speech muscles as evidenced by electromyographic measurements taken during their stuttered speech. This evidence, coupled with the apparently higher incidence of stuttering among nonright-handers, was believed to support the notion that the bilateral speech centers of the stutterer were sending conflicting signals to the speech apparatus, resulting in a dissociation of the activity of the homologous left and right-sided muscle groups.

Orton (1937) believed that stuttering and other developmental disorders were associated with delayed neurological development which led to poorly established cerebral dominance. In the case of dyslexia, he argued that the lack of cerebral dominance would

result in competing visual engrams in the bilaterally symmetrical cerebral hemispheres. The presence of these competing images would lead to confusion and a tendency to mirror-invert letters or words. Orton (1925) used the term "strephosymbolia," meaning "twisted symbols" to describe this phenomenon. There is an inherent similarity between Orton's "strephosymbolia" and the notion of bilateral speech centres leading to a speech-motor struggle or confusion in the case of stuttering. Orton (1937) actually sought a common underlying relationship between the various developmental disorders he described. He concluded that

...difficulty with the revival of sequences affecting recognition in the two sensory syndromes - reading and the understanding of speech - and affecting reproduction in the motor syndromes - motor speech delay, agraphia, stuttering and apraxia - is the only factor which we have been able to find common to the symptomatology of the group as a whole. (p.150)

Orton (1937) believed that the development of a dominant hemisphere could be facilitated by the training of one hand in motor skills, and that this would ultimately lead to an improvement in the disorder. In particular, he encouraged parents not to interfere with a child's "natural" handedness tendency, because he believed that doing so would discourage the establishment of a single dominant hemisphere. This belief was based on evidence at the time of an association between left-handedness and developmental disabilities. (Bryngelson, 1931; 1935; Dearborn, 1933; Quinan, 1921; Travis and Lindsley, 1933), and the fact that there appeared to be a positive correlation between forced handedness changes and an abnormally high incidence of developing those disorders (Ballard, 1912; Bryngelson, 1935; Bryngelson and Rutherford, 1937). Ballard (1912) in particular found that the rate of stuttering among children whose handedness had been forcibly switched from left to right was four times higher (4.3%) than among those whose handedness had not been interfered with (1.1%). Bryngelson (1940) reported that of 152 male stutterers,

34% were "ambidextrous" and 61% had been forcibly shifted from left- to right-handedness, whereas the same figures for fluent speakers were 4% and 5% respectively.

Two important assumptions of Orton's (1937) reasoning are that: 1) cerebral dominance develops over time; and 2) cultural or environmental influences on handedness can influence the development of cerebral dominance. More recent evidence supports neither of the above assumptions. First, more recent evidence seems to support the notion that hemispheric asymmetries are present from birth (Woods and Teuber, 1973; Dennis and Kohn, 1975; Dennis and Whitaker, 1976; Woods and Carey, 1979; Rankin, Aram and Horwitz, 1981; Vargha-Khadem, O'Gorman and Watters, 1985). Second, handedness and cerebral dominance are only loosely related; while most right-handers have left hemisphere dominance for speech and language, the majority of left-handers also have left-hemisphere dominance for speech and language, and a small proportion of right-handers have right-hemisphere speech and language dominance (Bryden, 1982).

Perhaps most importantly, a considerable body of research has since found no association between handedness and learning disabilities or stuttering (Bishop, 1984; Daniels, 1940; Felton, Wood, Brown, Campbell and Harter, 1987; Pennington, Smith, Kimberling, Green and Haith, 1987; Records, Heimbuch and Kidd, 1977; Rutter, Tizard and Whitmore, 1970; Van Dusen, 1939; Webster and Poulos, 1987; Wolf and Goodglass, 1986). Ojemann (1931) and later Burt (1937) noted that a shift of handedness was clearly not sufficient condition to cause a speech disturbance. Burt (1937) argued that a small association (such as reported by Ballard, 1912) between shifted handedness and stuttering could have been brought about by the stress associated with the harsh measures used to enforce the shift. In addition, the direction of causality in Brynogleson's (1940) more dramatic data is unclear. In light of the early reports of an association between handedness and stuttering (Ballard, 1912; Quinan, 1921), it is certainly probable that many of the

forced handedness shifts were imposed by parents, teachers or speech pathologists in response to the stuttering.

In response to a number of negative reports (Burt, 1937; Daniels, 1940; Ojemann, 1931; Williams, 1955; Van Dusen, 1939), interest in the cerebral dominance hypothesis fell off during the 1940s and 1950s. However, a report by Jones (1966) rekindled interest in the theory. Jones (1966) reported data on four neurosurgical patients who had a history of stuttering. Prior to the surgery, which was for cerebral pathologies ostensibly unrelated to the stuttering, Jones used the Intracarotid Sodium Amytal test (Wada and Rasmussen, 1960) to determine which hemisphere was dominant for speech. This technique involves injecting a barbiturate anesthetic into one of the carotid arteries of an awake patient. As the barbiturate reaches the hemisphere on the side of the injection, it causes a temporary cessation of conscious cognitive activity from that hemisphere. If the hemisphere first reached by the anesthetic is the dominant hemisphere for language, the patient will experience a transient aphasia (Wada and Rasmussen, 1960). In those rare cases in which speech is bilaterally distributed, anesthetizing either hemisphere will cause a transient aphasia.

Jones (1966) reported that in all four patients, the mechanisms for speech were distributed bilaterally, as evidenced by interrupted speech when either hemisphere was blocked by the barbiturate. Jones (1966) went on to report that after the surgery (which involved clipping an aneurysm in the region of the Circle of Willis in three cases and removing a frontal neoplasm in the fourth), all four patients spoke fluently. Finally, Jones stated that subsequent Sodium Amytal tests revealed that the patients all had unilateral control of speech postoperatively. Jones (1966; 1967) argued that his results support the cerebral dominance hypothesis.

There are a number of reasons that Jones' conclusions must be held with some caution. First, it is clearly possible to have bilateral speech representation without being a

person who stutters; Bryden (1982) concluded after an exhaustive review of the literature that approximately 20% of left-handed individuals have bilateral speech representation. It is certainly not the case that one in five left-handers stutters (Webster and Poulos, 1987; Records, *et al.*, 1977). On the other hand, the proportion of right-handers with bilateral speech representation is close to 0% (Bryden, 1982), and this is clearly much lower than the lifetime risk of stuttering, which is probably between 4% and 5% (Andrews *et al.*, 1983).

Two studies followed which attempted to generalize Jones' (1966; 1967) sodium amytal findings to stutterers for whom there was no known history of cerebral pathology (Andrews, Quinn and Sorby, 1972; Luessenhop, Boggs, Laborwit and Walle, 1973). All 6 neurologically healthy individuals in those studies had unilateral left-hemisphere dominance for speech. The one subject who had a history of cerebral pathology showed bilateral speech representation. Thus, as was pointed out by Andrews *et al.* (1983), of the 11 stutterers for whom sodium amytal test results have been reported, all 6 with no history of cerebral pathology had normal left-hemisphere dominance for speech, while the 5 with known cerebral pathology all appeared to have bilateral speech representation.

In summary, it is: 1) possible to have bilateral speech representation without being a person who stutters; and 2) the norm for people who stutter to have unilateral left hemisphere dominance for speech. Together, these facts contradict the conclusions of Jones (1966; 1967) and do not support the cerebral dominance hypothesis as originally stated by Orton (1937) and Travis (1934). Nevertheless, the findings reported by Jones (1966) were sufficiently provocative to stimulate a resurgence of interest in the cerebral dominance hypothesis as it relates to stuttering, and in neuropsychological research on stuttering in general. The following section will provide an overview of more recent approaches to hemispheric specialization and cerebral dominance, and this will be followed by a discussion of the neuropsychological investigations into stuttering that arose subsequent to the report of Jones (1966).

Behavioural Indices of Cerebral Laterality

Prior to the development of the intracarotid sodium amytal test by Wada and Rasmussen (1960), the only way to directly assess cerebral laterality was to observe the effects of unilateral brain damage (e.g. Luria, 1970). While the development of the sodium amytal test was clearly a major breakthrough, it was nonetheless an invasive technique that was in most cases reserved for surgical patients. In 1961, Doreen Kimura at the University of Western Ontario described a non-invasive, behavioural technique by which the laterality of individuals could be indirectly assessed. The technique, called dichotic listening, involved presenting different spoken digits to the two ears (through earphones) simultaneously and having the subject report what was heard from each ear. Kimura (1961) demonstrated that digits arriving to the right ear are more accurately reported than those arriving at the left ear. She suggested that this finding reflects the dominance of the left hemisphere for language, and the fact that each hemisphere has stronger connections to the contralateral ear. While each hemisphere receives projections from both ears, Kimura (1961) argued that the stronger contralateral pathways would suppress input arriving from the ipsilateral pathways. Kimura (1963) later demonstrated the same effect in children as young as four years old, a finding which suggested that cerebral dominance for language was present already by four years of age. In addition, she later (Kimura, 1964) reported a left-ear advantage for the perception of melodies by adults, suggesting that this function is lateralized to the right hemisphere.

An analogous technique involving the visual modality was used to assess cerebral dominance by Bryden and Rainey (1963) and Goodglass and Barton (1963). This task involved flashing a stimulus tachistoscopically to one side of a fixation point upon which the subject was focusing. The stimulus is flashed for a brief period of time chosen specifically to be less than the time required to initiate an eye movement. The paradigm was originally used as a means to test hypotheses regarding learned directional reading

habits and post-exposural scanning mechanisms (Heron, 1957; and Forgays, 1952). Heron (1957) observed a right visual field advantage for the recognition of letter strings presented unilaterally, but a left visual field advantage when the strings extended across the fixation point. Heron (1957) argued that his results reflected a left → right attentional scan of the post-exposural trace. Specifically, he suggested that a left → right scan would favour right-lateralized stimuli over left-lateralized stimuli because the stimulus trace in the left visual field would decay while attention was shifted to the far left (i.e. the beginning) of the stimulus array. When the stimuli extended across the fixation point, the letters at the left of the array were attended to before those to the right, leading to a left visual field advantage.

In spite of Heron's (1957) interpretation of his results, the tachistoscopic presentation of lateralized stimuli was well-suited to provide a visual analogue to the dichotic listening test, and was appropriated by neuropsychologists as a means to test hypotheses relating to hemispheric specialization (Bryden and Rainey, 1963; Goodglass and Barton, 1963). The human visual system is organized such that information in each visual half-field is initially transmitted exclusively to the contralateral hemisphere. Thus, unilateral presentation of a stimulus presented for a sufficiently brief time ensures that it will be presented initially to the contralateral hemisphere. It is now well-established that when verbal stimuli are used, they are more accurately identified when presented to the right visual half-field (RVF) than when presented to the left visual half-field (LVF) (see Bradshaw and Nettleton, 1981; Bryden, 1982; Beaumont, 1982). The convention²! neuropsychological explanation for this finding is that information presented to the left hemisphere has more direct access to the left-hemisphere-based language mechanisms. Bryden and Mondor (1991) have pointed out that Bryden's early work (e.g. Bryden and Rainey, 1963) using the divided visual field paradigm employed single-letter displays, specifically out of concern about scanning artifacts.

The promise that both these techniques offered led to an explosion in research on cerebral laterality during the 1960's and 1970's. In addition to dichotic listening and divided visual field studies, other behavioural paradigms were developed which allowed inferences about cerebral asymmetry to be drawn. These have included methodologies such as dichoptic (Bradshaw, Burden and Nettleton, 1986) and cognitive-motor concurrent task paradigms (Kinsbourne and Cook, 1971). This period also saw the development of a number of radiological (e.g. regional cerebral blood flow - rCBF, and positron emission topography - PET) and electrophysiological (evoked potential, hemispheric alpha asymmetry, brain electrical activity mapping) techniques that could be used to assess (among other things) patterns of functional cerebral lateralization even more directly than the behavioural methods. A great deal of effort has been devoted to the study of the differences in the processing capabilities of the left and right hemispheres. The left hemisphere is superior at processing the linguistic aspects of speech and language, as well as verbal memory (Lezak, 1983), and various aspects of motor control (Goodale, 1988; Kimura, 1977). The functions that appear to be processed more efficiently by the right hemisphere include visuo-spatial processing (Sperry, 1974), spatial memory (Lezak, 1983) emotional expression (Ahern and Schwartz, 1985; Davidson and Fox, 1982; Etcoff, 1986; Fox and Davidson, 1988; Gianotti, 1972), perception of faces (Heilman, Scholes and Watson, 1975), and both production and perception of prosody in speech (Ross, 1981).

A number of attempts have been made to find broader dimensions upon which the various "lateralized" brain functions lie, so that the apparent differences between the hemispheres might be reduced to a simple dichotomy of roles rather than lists of lateralized functions. Bradshaw and Nettleton (1981) have reviewed this literature thoroughly, and have attempted to summarize the findings. They concluded that the left hemisphere is superior at processing information in a sequential, analytic, time-dependent fashion. Since language is a sequential, time-dependent information class, it is processed more efficiently

by the left hemisphere. The right hemisphere, on the other hand, was seen as a holistic processor, better equipped to process time-independent information such as is the case in the spatial mapping of extrapersonal space and the perception of emotional affect.

Bradshaw and Nettleton (1981) concluded that "there is a continuum of function between the hemispheres, rather than a rigid dichotomy, the differences being quantitative rather than qualitative, of degree rather than of kind" (p. 51).

Nevertheless, Zaidel (1983) pointed out that, while a continuum rather than a dichotomy, the account of hemispheric specialization proposed by Bradshaw and Nettleton (1981) is essentially a list of adjectives rather than a theory. Zaidel (1983) has stressed the poor state of the literature on laterality, arguing that there is a lack of consensus or convergence in theoretical viewpoints, a general lack of rigour in experimental design and formulation of assumptions, and a tendency to reduce theories to dichotomous adjectives. Zaidel (1983) described the growth of the literature on hemispheric asymmetry as being more lateral than vertical; wide and shallow rather than focused and cumulative. Efron (1990) has also harshly criticized the state of the literature, arguing that standard notions of hemispheric specialization are based on "profound epistemological and conceptual confusions that are proving fatal to the field of laterality research" (p. 102). Both Zaidel (1983) and Efron (1990) expressed concern about a tendency in the literature to assume that differences between the hemispheres reflect exclusive hemispheric specialization rather than subtle differences in processing styles.

A basic assumption of the structural models of Kimura (1961) and Bryden (1965) was that perceptual asymmetries are brought about as a result of the ease of access with which information in the early stages of processing could reach the specialized mechanisms in the dominant hemisphere. An important aspect of this still unproved assumption is that the information is always, ultimately, processed in the "dominant" hemisphere.

Moscovitch (1986) stressed the importance of distinguishing between interhemispheric transfer, and relative efficiency (or "direct access"; Zaidel, 1983) models of perceptual asymmetry. According to the interhemispheric transfer model, verbal material presented through the right ear or the right visual half-field has more direct access to the left-hemisphere's language mechanisms than does information presented to the left ear or visual half-field. Information presented initially to the nondominant hemisphere must thus cross the corpus callosum, and this is believed to be associated with both a time delay (manifested in slower response times) and a potential for loss of information (manifested in decreased accuracy).

Moscovitch (1986) pointed out that another plausible possibility (his "relative efficiency" model) is that the information is in fact processed, albeit less efficiently, in the hemisphere to which it was initially presented. Schweinberger, Sommer and Stiller (1993) reported electrophysiological evidence that both faces and words that were tachistoscopically presented to one or the other hemisphere, were initially processed in the hemisphere to which they were projected.

A number of authors have noted that factors other than the structural hemispheric processing capabilities may influence performance on divided visual field and dichotic listening tasks (Bryden, 1978; Bryden and Mondor, 1991; Cohen, 1982; Kinsbourne, 1970). In particular, a substantial body of evidence has accrued recently suggesting that attentional resources may be preferentially or disproportionately allocated to the right visual field and ear (Lambert, Beard and Thompson, 1988; Leiderman, 1986; Levinthal, 1988; Mondor and Bryden, 1991; 1992a; 1992b; 1992c). This possibility would clearly have a profound influence upon how one would interpret divided visual field and dichotic listening findings. The role of attention in the expression of cerebral laterality will be discussed in more detail in a following section.

Summary: It is clear today that the simple structural explanations that were initially advanced to account for perceptual asymmetries in dichotic listening and visual half-field studies were almost certainly oversimplifications. It may be partly a consequence of the fact that these explanations were widely accepted that theories of hemispheric specialization have also tended to be oversimplified, relying on simple dichotomies or linear dimensions to describe the differences between our cerebral hemispheres. In addition to this concern, one issue of considerable relevance to the present research is the distinction between structural models and dynamic, or attentional, models of perceptual asymmetry.

Hemispheric Specialization in Stuttering

The behavioural paradigms for assessing cerebral laterality that were gaining acceptance around the time of Jones' (1966; 1967) report of apparent bilateral speech centres in stutterers offered the opportunity to assess the cerebral dominance hypothesis of Travis (1934) and Orton (1937). Perhaps not surprisingly, a substantial body of research appeared that employed those methods, as well as (more recently) electrophysiological and neuroradiological techniques, to investigate cerebral dominance in stuttering (as well as in dyslexia and other developmental disorders).

The first study to use any of these techniques to investigate cerebral lateralization in stutterers appeared to support the cerebral dominance hypothesis and the conclusions of Jones (1966; 1967). Curry and Gregory (1969) used the dichotic listening test and found no ear advantage in a group of stutterers, in contrast to the expected right ear advantage in fluent controls. However, two subsequent studies, both of which used substantially larger sample sizes, failed to replicate that finding (Quinn, 1972; Slorach and Noehr, 1973). Both studies found statistically significant right ear advantages in both stutterers and fluent controls. Quinn (1972) did note, however, that there was a higher incidence of reversed asymmetry on the task in the stutterers. Slorach and Noehr (1973) reported only averaged data. It is interesting to note that at the time of Quinn's (1972) report, all five studies that

had used dichotic listening paradigms in stutterers (three of which were reported as abstracts of conference presentations) had found a higher proportion of left-ear advantages on the task (see Quinn, 1972).

Subsequent studies using dichotic listening to assess cerebral asymmetry in people who stutter yielded a variety of positive results, including no ear preference (Blood, 1985), a smaller right ear preference (Blood and Blood, 1989; Davenport, 1979), and a higher frequency of left ear preference (Blood, 1985; Rosenfield and Goodglass, 1980; Sommers, Brady and Moore, 1975). Davenport (1979) reported a negative correlation between stuttering severity and the strength of the right ear preference. Blood and Blood (1986), however, found that there was no relationship between fluency and ear preference. Adding to the above confusion is the fact that at least two studies have reported no differences whatsoever between people who stutter and fluent controls on dichotic listening tasks (Pinsky and McAdam, 1980; Sussman and MacNeilage, 1975).

The most remarkable aspect of the literature on dichotic listening performance in people who stutter is the variability both within and between studies. Taken as a whole, these studies do seem to suggest that people who stutter differ from fluent speakers in terms of the consistency with which they will demonstrate a right ear preference on the task. This conclusion is reinforced if one distinguishes between studies that have used meaningful rather than non-meaningful target stimuli. It was pointed out by Moore (1984) that investigations using meaningful linguistic stimuli in dichotic listening have all shown evidence of greater right hemisphere involvement in the processing of the stimuli. Those dichotic listening studies that have reported no differences between the groups (Pinsky and McAdam, 1980; Sussman and MacNeilage, 1975; Slorach and Noehr, 1973) used non-meaningful stimuli such as consonant-vowel syllables. This notion is reinforced by the fact that two tachistoscopic visual half-field studies (Hand and Haynes, 1983; Moore, 1976) and one study involving measurement of evoked potentials (Ponsford, Brown, Marsh and

Travis, 1975), all using meaningful stimuli, reported evidence of anomalous hemispheric asymmetries in people who stutter.

Moore and his colleagues (Moore, 1984; 1986; Moore, Craven and Faber, 1982; Moore and Haynes, 1980) have suggested that people who stutter, while having their speech mechanisms lateralized to the left hemisphere, inappropriately engage their right hemisphere during speech and language processing. They argue that stuttering is brought about by attempting to process speech using the right-hemisphere's non-segmental information processing strategies. The term "segmental" refers to the supposed ability of the left hemisphere to parse information sequentially (Bradshaw and Nettleton, 1981), as appears to be the necessity in speech and language.

Supporting Moore's argument are a number of studies demonstrating greater right hemisphere activation in people who stutter during various linguistic processing tasks (Moore, 1986; Moore, Craven and Faber, 1982; Moore and Haynes, 1980). Further support for Moore's (1984) argument comes from the findings of Wood, Stump, McKeehan, Sheldon and Proctor (1980) who measured regional cerebral blood flow (rCBF) in two stutterers during attempted reading both on and off the medication haloperidol. They found greater blood flow over Broca's area in the right than the left hemisphere while the subjects were off the medication and stuttering. When on the medication, the subjects were reported to be essentially fluent, and demonstrated greater blood flow over the left hemisphere Broca's area.

The tasks in each of the studies reported by Moore (Moore, 1986; Moore, Craven and Faber, 1982; Moore and Haynes, 1980) involved listening to linguistic stimuli, rather than expressive behaviour. In order to explain the fact that people who stutter do not have a generalized linguistic processing deficit, Moore and Haynes (1980) suggest that "perception and comprehension of speech/language may be relatively unaffected with nonsegmental, right hemispheric processing" (p. 243). This explanation would appear to

contradict the very theory of hemispheric specialization (e.g. Bradshaw and Nettleton, 1981) upon which the model is based. Language comprehension relies heavily on the sequential, time-dependent segmentation of the incoming sound stream. In fact, inasmuch as the rate at which incoming linguistic material is not under the control of the listener, one could argue that the ability to fluently process information during language comprehension should be, if anything, even more heavily dependent on the left hemisphere's segmental information-processing properties than speech output, the rate of which is under the control of the speaker.

Despite the fact that Moore's (1984; 1986; Moore *et al.*, 1982; Moore and Haynes, 1980) model cannot account for the uniquely expressive nature of the disorder, his contribution is indisputably significant. His was the first theory to attempt to explain the apparent aberrant interhemispheric relations in people who stutter without relying on the essential elements of the cerebral dominance hypothesis. It is fundamental to Moore's "segmentation dysfunction hypothesis" that the left hemisphere of the person who stutters is specialized for the processing of sequential or segmental information just as is the case in fluent speakers. What leads to stuttering, according to the segmental dysfunction hypothesis, is the inappropriate attempt to use the holistic or non-segmental right hemisphere strategy to process speech and language.

Nevertheless, notwithstanding Moore's findings, and irrespective of the type of stimuli used, it is impossible to dispute the observation that people who stutter are capable of demonstrating the same right ear advantage on dichotic listening measures (Pinsky and McAdam, 1980; Slorach and Noehr, 1973; Sussman and MacNeilage, 1975; Blood, 1985) and electrophysiological measures (Pinsky and McAdam, 1980) as fluent speakers. In addition, the mere fact that any studies demonstrating no difference between stutterers and fluent controls have been published raises the concern that publication bias (the

tendency of scientific journals to publish only positive results) may have influenced the balance of the available evidence in this regard (Bishop, 1990).

As discussed earlier, recent studies have found no evidence of an association between handedness and stuttering (Records *et al.*; Webster and Poulos, 1987). In addition, direct assessment of cerebral dominance by the sodium amytal test in neurologically healthy people who stutter has consistently demonstrated normal left hemisphere lateralization for speech and language (Andrews *et al.*, 1972; Luessenhop *et al.*, 1973). This alone would seem to rule out a cerebral dominance explanation of the experimental findings reviewed above. On the other hand, the more general notion of aberrant interhemispheric relations in people who stutter (Travis, 1978) seems to be supported by the available literature. The apparent contradiction between normal right-handedness and reversed perceptual or electrophysiological asymmetries may be reconcilable by distinguishing between hemispheric specialization and hemispheric activation (Moore, 1984; Forster and Webster, 1991).

Clearly the most notable feature of the literature on cerebral dominance in stuttering is the variability in the patterns of cerebral dominance evidenced by people who stutter (Blood, 1985; Blood and Blood, 1986; Davenport, 1979; Quinn, 1972; Sommers *et al.*, 1975). Blood (1985), in perhaps the most ambitious and well-controlled study of perceptual asymmetry in stutterers, was unable to come to any firm decision regarding the validity of the cerebral dominance hypothesis. In fact, he made it very clear that the most salient and impressive finding in his dichotic listening study was the heterogeneity of the stuttering population, which included individuals with right, left, and no ear advantage. Blood (1985) attributed this finding to the probable presence of sub-groups among the population of people who stutter. An alternative explanation of the heterogeneity in the performance of people who stutter on these measures is that it reflects the influence of dynamic situational and task-related factors on the expression of perceptual asymmetries.

As will be reviewed here, there is a substantial body of evidence (to be described below) which suggests that attentional factors may play an important role in the expression of lateral asymmetries on dichotic listening and tachistoscopic visual half-field tasks. People who stutter may have an attentional system that makes them more susceptible to such influences, or that alters the manifestation of those influences.

Summary: People who stutter are either no more likely (Webster and Poulos, 1987) or only marginally more likely (Records *et al.*, 1977) to be left-handed than fluent speakers. Intracarotid sodium amytal tests on individuals with no history of neurological illness or injury have consistently found normal left hemisphere lateralization in people who stutter. Taken together, these findings indicate that people who stutter have normal left-hemisphere structural dominance for speech, language and praxic motor functions. Nevertheless, people who stutter do appear to be more likely to demonstrate either no asymmetry or reversed asymmetry on tachistoscopic visual half-field and dichotic listening tasks, particularly when meaningful linguistic materials are employed as the stimuli. In addition, electrophysiological and neuroradiological studies have demonstrated evidence of elevated right hemispheric activation in people who stutter during speech and language tasks. These findings may be reconciled with a normal pattern of cerebral dominance by postulating an anomalous pattern of hemispatial/hemispheric attention in people who stutter. This idea will be discussed further in the next section.

Hemispheric Activation and Attentional Asymmetries

Cohen (1982) has contrasted two different classes of models of perceptual asymmetry. The traditional structural explanations (Kimura, 1961) and modern variations of them (Sidtis, 1982; Zatorre, 1989) belong to one broad class of models. Various manifestations of attentional models comprise the other broad class. Cohen (1982) argued that structural models are simply unable to account for the variability from trial to trial in performance on tasks which purportedly assess structural asymmetries. He suggested that

only by marrying structural and attentional models can one provide a complete account of performance on such tasks. While attentional models vary considerably in terms of their specifics, they share in common the notion that the distribution of attention in perceptual space will have a profound influence on perceptual asymmetries observed experimentally. Because of the difficulty in specifying the internal and external factors that will influence the distribution of attention in space from moment to moment, a crucial weakness of attentional models is a loss of predictive power. This may be one reason why the influence of attentional factors in perceptual asymmetries has been largely relegated to the error term in most past research.

Attention may be broadly defined as a hypothetical construct for the mechanism by which the brain modulates the way information is processed (Bowers and Heilman, 1980). In general terms, the attentional models of perceptual asymmetry state that any bias of attention toward one side of space would be expected to have a profound influence on both the size and the direction of an experimentally observed asymmetry. Attentional models may be further categorized into two broad classes: (1) those which propose that cognitive factors may influence the orientation of attention in a material- or context-specific way (Kinsbourne, 1970; Hellige, 1978), and (2) those which propose that there is a consistent attentional bias to one side of space (the right side in right-handers). These two notions, which are not mutually exclusive, will be briefly reviewed below.

Context-Specific Attentional Biases

One of the earliest and most prominent attempts to explain lateral asymmetries in terms of attentional processes was proposed by Kinsbourne (1970). Kinsbourne (1970) described lateral asymmetries of attention as the manifestation of attentional biases to one side of space brought about by activation of the contralateral cerebral hemisphere. He proposed that cognitive activity which led to preponderant activation of one hemisphere (e.g. overt or covert verbal activity) would lead to a bias of attention toward the

contralateral hemispace. Kinsbourne (1970) argued that the human brain retains a general tendency, present in subhuman species, for orientation to one side of space to be associated with preparatory activation in the contralateral cerebral hemisphere. Verbal activity, he argued, would lead to an expectancy for verbal input - a "verbal set." While involving a "symbolic function unrelated to spatial location" (p. 196) this would nevertheless lead to orientation to the contralateral hemispace, essentially as an artifact of the brain's phylogenetic development. In support of this idea, he had subjects perform a verbal task concurrently with a supposedly neutral visual task involving detection of gaps in squares which were presented tachistoscopically. He reported that concurrent verbal activity led to an asymmetry favouring the detection of gaps in the right visual field. The implication of this finding is that lateral asymmetries observed with divided visual field and dichotic listening paradigms may be influenced by expectancy independently of structural lateralization.

Attempts to replicate Kinsbourne's (1970) results have failed, both when the methodology has been copied faithfully (Boles, 1979) and under modified conditions (Boles, 1979; Gardner and Branski, 1976). However, subsequent research has lent some support to the general notion that lateral asymmetries can be modified by manipulating task expectancy (Hellige, 1978; Kershner, Thomae and Callaway, 1977; Klein, Moscovitch and Vigna, 1976). In fact, Spellacy and Blumstein (1970) had already shown that the context in which auditory stimuli were placed could affect the dichotic listening asymmetry. When consonant-vowel-consonant syllables were placed in the context of melodies and sound effects the typical right ear advantage on a dichotic listening task was reversed to a left ear advantage (Spellacy and Blumstein, 1970).

One direct way of testing Kinsbourne's hypothesis that dichotic listening and divided visual field asymmetries result from a material-specific hemispheric priming is to manipulate the priming bias by manipulating the expectancy for the primary task. A series

of trials involving two different tasks (i.e. one "left-hemisphere task" and one "right hemisphere task") may be presented with trials of the two tasks intermixed, either randomly (Berlucchi, Brizzolara, Marzi, Rizzolatti and Umiltà, 1974; Dee and Hannay, 1974; Hellige, 1978; Kallman, 1978) or in blocks (Klein, Moscovitch and Vigna, 1976). Berlucchi *et al.* (1974) obtained the expected RVF advantage for discrimination of letter pairs and left visual half-field advantage for discrimination of unknown faces using randomly intermixed presentation of the stimulus types. Kallman (1978) obtained similar results with a dichotic listening task involving pairs of speech sounds and musical sounds. These results indicate that attentional factors related to hemispheric priming cannot fully explain asymmetries on the dichotic listening or divided visual field tasks used (see also Beaumont and Colley, 1980; Goodglass and Calderon, 1977). On the other hand, none of these studies involved a comparison of performance with mixed versus "pure" presentation of the different stimulus types. Thus, while these results cannot be explained by Kinsbourne's (1970) model, they lack the important control condition necessary to identify the relative contribution of attentional and structural factors. Hellige (1978) reported a series of studies that included such a comparison. He obtained a LVF (right hemisphere) advantage for recognition of polygon forms when they were presented in a pure list, but a RVF (left hemisphere) advantage on the same task when the trials were presented randomly intermixed with word recognition trials. Hellige (1978) also reported that holding two nouns in memory while performing the polygon recognition task also led to a RVF (left hemisphere) advantage on the task, and that the combined effect of the two manipulations was not additive. Thus, while "the presence of an attentional bias prior to the presentation of a stimulus pair is not a necessary condition" for demonstrating laterality patterns (Kallman, 1978, p. 227), it appears that attentional factors are able to influence the magnitude and direction of those patterns.

Neither structural models nor attentional priming models appear to provide a complete explanation of divided visual field or dichotic listening asymmetries on their own. Most authors concede that a variety of factors interact to give rise to observed laterality patterns, and that these might include pre- and post-exposural attentional factors and strategic coding mechanisms, as well as structural asymmetries (e.g. Beaumont and Colley, 1980; Bryden, 1978; Cohen, 1982; Hellige, 1978; Kershner *et al.*, 1977; Klein *et al.*, 1976; Spellacy and Blumstein, 1970). Hellige (1978) has proposed a modification of Kinsbourne's (1970) hemispheric priming model that, to some extent, bridges the gap between attentional and structural models. He argued that priming exerts its effect by way of selective hemispheric activation, rather than selective perceptual orientation, and that the hemisphere that becomes more activated depends upon the strategy being emphasized in processing the stimuli. The evidence reviewed above does indicate that laterality patterns can be modified by modifying cognitive set; they do not, however, uphold the crucial prediction of the selective perceptual orientation model (Kinsbourne, 1970) that by mixing verbal and nonverbal stimuli both asymmetries will be diminished.

Perhaps the best conclusion that one can draw regarding hemispheric priming is that, under appropriate circumstances, laterality patterns evident in dichotic listening and divided visual field tasks can be manipulated by manipulating cognitive set. While the mechanisms underlying this relationship do not appear to relate to selective perceptual orientation in space (Hellige, 1978; Hellige and Cox, 1976), they may be viewed as "attentional" inasmuch as they involve a manipulation in the manner by which the individual perceives and processes information.

Inherent Rightward Attentional Bias.

The idea that attentional factors might influence lateral asymmetries of visual perception was originally proposed by Heron (1957). Heron (1957) was primarily concerned with the relation between post-exposural attentional processes and English

reading habits, and interpreted his findings in the context of Hebb's (1949) cell-assembly theory. However, Heron (1957) drew a careful distinction between "set," which refers here to a pre-exposural tendency to attend to one visual half-field, and post-exposural scanning processes. In fact, Heron (1957) manipulated set in one of his experiments by informing subjects of the location of the impending stimulus presentation. He reported that under this "informed" condition, performance improved in the left, but not the right visual field, and concluded that "one factor involved in the more efficient recognition of letters in the right visual field is a tendency for the uninstructed [subject] to attend to the right" (p.46).

As Heron (1957) acknowledged, there is no reason to assume that pre- and post-exposural attentional factors are mutually exclusive. Nevertheless, his suggestion that the uninstructed subject has an inherent pre-exposural rightward attentional bias has been largely ignored in lieu of his post-exposural scanning theory which was the primary focus of his paper. The more recent notion of an inherent rightward attentional bias in right-handed individuals (e.g. Bryden *et al.*, 1983; Bryden and Mondor, 1991c) is generally attributed to contemporary theorists (Bryden, 1978; Cohen, 1982).

Dichotic listening ear advantages can be modified by modifying the degree to which subjects are free to deploy attention as they choose (Bryden, 1978; Bryden *et al.*, 1983; Hiscock and Kinsbourne, 1977; 1980; Hiscock and Stewart, 1984). The directed attention variation of the dichotic listening task was developed as a means of controlling the deployment of attention. Subjects are asked to report exclusively the stimuli they hear from one ear, with the designated ear varied either from trial to trial or block to block. Bryden *et al.* (1983) demonstrated that performance was more accurate for both ears under directed attention than free-recall, and that the right ear advantage was more robust under directed attention. This could be interpreted as evidence that attentional factors only add uncontrolled variance to ear asymmetries, and that the direction of the asymmetry does

reflect underlying structural factors. However, Bryden *et al.* (1983) were particularly impressed with the finding that 21% of the responses given under the directed attention condition were from the supposedly unattended ear. In addition, more responses were reported from the "unattended" right ear than the "unattended" left ear, suggesting that the verbal instructions were inadequate as a means of controlling the allocation of attention. In fact Bryden (1978) had already discussed that very possibility:

Even instructions to attend to or monitor one ear or visual field to the exclusion of the other may not be entirely adequate as a control over attentional biases, for it may well be easier to attend to one ear than to the other. (p. 124)

The visual analogue to the directed attention dichotic listening task is more difficult to implement because, once told where a stimulus will appear, there is little to prevent an individual from simply looking in that direction. However, it is now well established that visual attention can be manipulated in space independently of eye movements (Eriksen and St. James, 1986; Gawryszewski, Riggio, Rizzolatti and Umiltà, 1987; Posner, Snyder and Davidson, 1980; Remington and Pierce, 1984). Thus, a visual analogue to the directed attention dichotic listening paradigm is possible, given that fixation is controlled adequately (i.e. eye movements are minimized). Hardyck, Chiarello, Dronkers, and Simpson (1985) used a variety of methods to direct attention to the appropriate visual field in a divided visual field lexical decision task. In addition to instructional manipulation of attention, they tried blocking trials to each visual field and using pre-trial cues to indicate the appropriate visual field. Eye movements were monitored, and stimulus presentation was made only after steady fixation for a duration of 83 ms within .250° of fixation. The authors reported that a RVF advantage was consistently obtained on the task, regardless of the method used to direct attention. These findings were interpreted as evidence that the observed divided visual field asymmetries were related to processing differences between the cerebral

hemispheres, rather than to the allocation of attention in space. However, it is possible that their attempts to manipulate attention were insufficient to overcome the attentional bias.

While the authors reported that their manipulations did lead to improved performance in the attended visual field, it remains possible that the asymmetries that persisted might reflect the relative ease with which attention was focused in the two fields. In short, the authors' conclusions rest on the untestable assumption that their attentional manipulations were completely effective.

Other evidence, both direct and indirect, suggests that the allocation of attention in space does contribute to the RVF advantage for linguistic tasks. For example, bilateral presentation of visual stimuli in divided visual field tasks has been shown to lead to substantially larger RVF advantages than the more common unilateral presentation (Boles, 1987; 1990; Hines, Sutker, Satz, and Altman 1976; Leiderman, 1986; Levinthal, 1988; McKeever and Huling, 1971; Seitz and McKeever, 1984). Leiderman (1986) demonstrated that this effect is not simply an artifact of stimulus load, and provided evidence suggesting that it is related to the distribution of attention between the visual fields. She also reported that the enhancement of the RVF superiority was limited to free report conditions. Under partial report conditions, where a pre-trial cue was used to indicate the location of the relevant stimuli, the enhancement was absent. Stated differently, when subjects were free to deploy their attention as they chose, the RVF advantage was larger than when they were constrained by the partial report conditions. Leiderman (1986) argued that her results reflect a strong tendency for individuals to bias attention toward the right visual field during bilateral trials. Levinthal (1988), who found a RVF advantage for word recognition under bilateral but not unilateral presentation, also argued that attentional bias interacts with structural factors to produce visual field asymmetries.

Perhaps the most compelling evidence that perceptual asymmetries are at least in part related to a predisposition to attend to the right visual field and ear has come from a series of studies recently reported by Mondor and Bryden (1991; 1992a; 1992b; 1992c; Mondor, 1993). These studies have all been based upon the principle that in order to demonstrate a role for attention in ear and divided visual field asymmetries one must compare performance under standard conditions with performance under conditions that equate the distribution of attention between the ears or visual half-fields. This is essentially the same basic approach that had been used earlier, both recently (e.g. Hardyck *et al.*, 1985; Liederman, 1986; Bryden *et al.*, 1983) and originally by Heron (1957). However, a critical difference between the recent work of Mondor and Bryden (1991; 1992a; 1992b; 1992c) and the earlier work lies in the means by which attention was manipulated. In all of the previous work, attention was directed to the appropriate location by means that relied on the subjects' active allocation of attention in compliance with either verbal instructions or a cue that acted as a form of instruction. Jonides and Yantis (1988) contrasted this type of cue with those that are able to capture attention automatically. Bryden and Mondor (1991) argued that attentional cues may be classified as either "push cues" or "pull cues." Push cues are those which direct attention to another location in space. Pull cues are those that capture attention, by attracting it to an area of space. Jonides and Yantis (1988) demonstrated that an abrupt visual onset acts as a very effective means of capturing attention. Once attention has been drawn to that area of space, other stimuli in that area may also be processed.

Taking advantage of the unique ability of abrupt visual onset to capture attention, Mondor and Bryden (1992a) used a "pull cue" to attract attention to the location of the forthcoming stimulus. They used both letter identification and lexical decision tasks. In each case, a dot appeared above the location of the target stimulus either just before the presentation of the target stimulus or simultaneously with it. The time delay between the

precue and the target stimulus (the "stimulus onset asynchrony" or SOA) ranged from 0 ms (simultaneous presentation) to 167 ms. The rationale of Mondor and Bryden (1992a) was that the abrupt onset of the dot precue would capture visual attention to the appropriate visual field. If the pre-exposural distribution of attention to the right and left visual fields contributes to the RVF superiority for linguistic tasks, then the asymmetry should be attenuated or abolished by the precueing technique. This is what was observed. At short SOAs, a RVF superiority was observed. At longer SOAs, (i.e. when more time was available to orient attention) the asymmetry was diminished, and in some cases there was no asymmetry whatsoever. As will be discussed later in the Introduction, a similar paradigm was used in the present research, in an effort to identify the contribution of pre-exposural attentional factors to the anomaly of interhemispheric relations in people who stutter.

Mondor and Bryden (1991; 1992b; 1992c) have observed an analogous effect of a tone precue on dichotic listening asymmetries. In this paradigm, the subject is instructed to report only the stimuli heard in the ear in which the tone precue was presented. Precues with a sufficiently large SOA substantially attenuated the right ear advantage for the identification of consonant-vowel syllables (Mondor and Bryden, 1991; 1992b) and words (1992c). The authors demonstrated clearly that the precue works by orienting attention to the location in which it is presented, in both the visual (Mondor and Bryden, 1992a) and auditory modalities (Mondor and Bryden, 1992b). The alternative possibility that the cue simply alerts the subject to the forthcoming trial is unlikely for at least two different reasons. First, the reduction in the ear asymmetry was shown by Mondor and Bryden (1991) to be related to a selective improvement in left ear performance. Second, the use of invalid trials (trials where the tone was presented to the visual field opposite to that in which the target stimulus appeared) clearly illustrated that attention was being drawn to the channel in which the precue was presented (Mondor and Bryden, 1992a; 1992b).

Mondor and Bryden (1992c) have also investigated the role of attentional factors in performance of a dichotic listening task believed to represent right hemisphere processing mechanisms. They were specifically interested in whether the attentional bias toward the right visual field is material specific or whether it reflects a consistent tendency to bias attention to the right. This is a critical issue if one wishes to relate the attentional bias to attentional mechanisms, rather than to the more general notion of hemispheric activation. Mondor and Bryden (1992c) performed two experiments with right-handed individuals in which stimuli were varied on two dimensions: linguistic content (i.e. the word) and emotional content (i.e. the emotional tone with which the word was spoken). In both experiments, two blocks of trials were performed. On one block of trials, subjects were required to identify the word. In another block they were required to identify the emotional tone with which the word was spoken. In Experiment 1, the authors reported a right ear advantage for identification of words that was diminished substantially at longer SOAs. However, they observed a small, but statistically significant left ear advantage for emotion identification. If the attentional bias toward right hemisphere was constant rather than material-specific, one would expect the left ear advantage to increase, as the allocation of attention was increased by the stimulus precue. This was not observed. However, accuracy of emotional identification was very high, prompting the experimenters to replicate the experiment with greater task difficulty. When this was done, (Experiment 2) the left ear advantage for emotion identification was reversed to a right ear advantage, which was itself abolished under longer SOAs.

Mondor and Bryden (1992c) argued that this finding represents "the first evidence that the asymmetric distribution of auditory attention may be completely responsible for the very direction of a perceptual advantage" (p. 399). In addition, while the left ear advantage under short SOAs in Experiment 2 was not in the direction the authors predicted, the combined results of Experiment 1 and Experiment 2 clearly indicate the presence of a

consistent attentional bias toward the right side of space. The authors suggested that the influence of attentional factors may be limited to situations with sufficient task difficulty. According to this view, the increase in the difficulty of the task led to the predomination of the attentional asymmetry and consequently a right ear advantage in Experiment 2. This interpretation is certainly consistent with the fact that left ear (and visual field) advantages are less frequently reported and generally smaller than right ear (and visual field) advantages (Kimura, 1973) when they are reported. Even if there is a structural asymmetry favouring the right hemisphere on a given task, the demonstration of a left ear (or visual field) asymmetry on that task will be predicated on overcoming an inherent tendency for subjects to bias their attention to the right side of space.

While negative reports do exist (e.g. Hardyck *et al.*, 1985), it appears that an attentional bias favouring right hemisphere is capable of influencing perceptual asymmetries. In fact, as suggested by the work of Mondor and Bryden (1992c), this influence may work to either exacerbate, attenuate or even change the direction of perceptual asymmetries. This position has particular relevance to theories (such as the cerebral dominance hypothesis) which relate developmental disorders to aberrant interhemispheric relations. These views typically depend upon traditional notions of cerebral laterality which clearly need to be reassessed in light of the evidence pointing to the role of attentional factors in laterality (Bryden and Mondor, 1991).

Summary: No purely structural model is able to account for the considerable variability in performance from trial to trial that is evident on dichotic listening and divided visual field tasks. Evidence has been reviewed suggesting that both material-specific factors and an inherent attentional bias may contribute to perceptual asymmetries.

Attentional Processes in People Who Stutter

The suggestion that attentional factors may be involved in stuttering is not a new one. Emotional arousal, particularly when it involves negative emotions such as anxiety

and fear, has a well-documented deleterious effect on the frequency and severity of stuttering (Bloodstein, 1981; Trotter and Bergmann, 1957; Van Riper, 1937; 1982). Negative affect may, as a cognitive set, be viewed as an attentional state. Furthermore, people who stutter often report that their speech fluency deteriorates when their attention is drawn to it, and improves when they "forget that they are stutterers" (Bloodstein, 1981: p. 246). This has been called "distraction," and refers to the idea that the person has been distracted from the "stimuli" that evoke stuttering (Bloodstein, 1981).

The distraction phenomenon may suggest a susceptibility of the person who stutters to interference from ongoing cognitive activity (e.g. "ineffective self-talk": Webster and Poulos, 1989). In support of that possibility, Webster, (1987; 1990a) has demonstrated that people who stutter have difficulty with a task that involves performing two cognitive operations simultaneously. The task involved transcribing to paper sequences of letters presented auditorially at a rapid rate. Because of the rate at which the letters were presented, this task required the concurrent attention to the letters being presented and to the task of writing them down. Stutterers transcribed fewer sequences correctly than fluent speakers, and this effect was more pronounced with longer sequences of letters (i.e. when the attentional demands of the task were increased). This is at the least suggestive of some kind of general deficit in attention in people who stutter.

As discussed above, attentional factors exert a potentially overwhelming influence on the expression of cerebral asymmetries. As such, an anomaly in those attentional mechanisms could offer an excellent explanation of the conflicting literature surrounding the cerebral dominance hypothesis. This could involve specifically those mechanisms that are involved in attentional asymmetries, or it could involve more general attentional mechanisms, with the effect on attentional asymmetries being secondary. As discussed above, the most direct means of testing the cerebral dominance hypothesis, intracarotid sodium amytal testing (Andrews, Quinn and Sorby, 1972; Luessenhop *et al.*, 1973) and

assessment of manual asymmetries (Records *et al.*, 1977; Webster and Poulos, 1987), appear to indicate that people who stutter are no more likely than fluent speakers to have abnormal cerebral lateralization. Yet the results of the majority of the well-controlled dichotic listening and divided visual field studies with people who stutter do indicate that people who stutter are much more likely to exhibit anomalous perceptual asymmetries than fluent speakers (Blood, 1985; Blood and Blood, 1986; Blood and Blood, 1989; Blood *et al.* 1986; Davenport, 1979; Rosenfield and Goodglass, 1980; Quinn, 1972; Sommers, Brady and Moore, 1975). Furthermore, there is evidence that people who stutter engage their right hemisphere more than fluent speakers when engaged in speech or language-related tasks (Moore, 1980; 1984; Moore *et al.*, 1982; Moore and Haynes, 1980; Ponsford *et al.*, 1975; Wood *et al.*, 1980). Given that an explanation based upon structural lateralization of speech can be ruled out, the most obvious alternative is the influence of attentional factors.

One possibility is that people who stutter have a less stable pattern of hemispheric activation than fluent speakers. This idea is particularly credible in light of the frequently reported observation that, regardless of overall group differences, a higher proportion of stutterers will exhibit either no asymmetry or a reversed asymmetry on dichotic listening and divided visual field tasks (Blood, 1985; Hand and Haynes, 1983; Moore, 1976; Quinn, 1972; Sommers *et al.*, 1975). Also consistent with this view are the results of the only study of perceptual asymmetry in stutterers in which there was an attempt to control for attentional factors. Blood *et al.* (1986) had stutterers and nonstutterers perform a dichotic listening task under directed attention and free recall conditions. Directed attention trials to the two ears were blocked. The authors reported a right ear advantage in both stutterers and nonstutterers in the directed attention condition. The fluent speakers also demonstrated a right ear advantage on the task in the free-recall condition. The stutterers, however, demonstrated no asymmetry under free-recall.

One interpretation of the observations of Blood *et al.* (1986) is that they reflect an attentional bias favouring the left ear in the people who stutter. According to this interpretation, a left ear bias acted to negate the right ear structural bias under free recall, but the influence of the bias was eliminated when the distribution of attention was controlled. While that conclusion would be premature based upon the results of a single study, it would certainly go some way toward explaining the variability in dichotic listening results across the literature.

Another possibility is that people who stutter have a more labile pattern of hemispheric activation. Similar atypical hemispatial attentional mechanisms have been associated with dyslexia (Boliek, Obrzut and Shaw, 1988; Dickstein and Tallal, 1987; Kershner and Morton, 1990; Kershner, 1995; Kershner, Henninger and Cooke, 1984; Obrzut, Conrad, Bryden and Boliek, 1988; 1981; Shucard, Cummins and McGee, 1984). Using a directed attention dichotic listening paradigm, Kershner and Morton (1990; Kershner, 1995) have demonstrated that reading disabled children have a tendency toward overactivation of either the left or right hemisphere, while possessing normal left hemispheric structural lateralization. Kershner and Morton (1990) suggested that "an instability in left-hemisphere language processing in learning disabled children may make them extra-vulnerable to bi-directional attentional demands which, in turn, can promote excessive activation in either the left or right hemisphere" (p. 183). The task involved directing attention to one ear for a block of trials and then switching to the other ear. In both studies (Kershner and Morton, 1990; Kershner 1995), learning disabled children had more difficulty switching attention between channels than the controls. Kershner and Morton (1990) hypothesized that the act of focusing attention on one ear may have led to excessive activation of the contralateral hemisphere in the reading disabled children. In other words, the reading disabled children had a tendency for the laterality of their attentional bias to switch, depending on the directed attention condition. A similar

mechanism might have been involved in the performance of the stutterers in the study by Blood *et al.* (1986), since they also blocked trials to each ear. However, since they did not report performance as a function of the ear previously monitored, it is impossible to assess this possibility.

Webster (1990b) has reported evidence from manual performance that is also consistent with the hypothesis that people who stutter have a more labile or weaker attentional bias. He had stutterers and nonstutterers perform a 2:1 rhythmic finger-tapping task, which involved tapping twice with one hand for each tap of the other hand under nonspeeded and unpaced conditions. Peters (1987) had earlier found that right-handers had a tendency to tap more regularly and more quickly with their right hand "leading" (tapping twice for each tap of the left hand) as compared to when their left hand was leading. Since the motoric demands of the task were relatively simple, Peters (1987) proposed that the asymmetry he observed in right-handers reflected a preference for attending to the right hand. Peters (1987) referred to Annett's Right-Shift genetic model of handedness. According to that model, left-handers lack a right shift gene that gives rise to a general bias favouring the right side of the body in right-handers. Consistent with this idea, Peters (1988) argued that, when differential experience is accounted for, performance asymmetries favouring the right hand and foot are best viewed as reflective of an attentional bias favouring the right side of the body. Webster (1990b) replicated the findings of Peters (1987) in fluent right-handers, but found that stutterers as a group had no asymmetry on the task. Webster argued that their lack of an asymmetry on the task could reflect a more labile or less consistent directional bias in people who stutter than in fluent speakers.

A related idea was discussed by Forster and Webster (1991), who suggested that people who stutter lack the inherent attentional bias found in typical right-handed nonstutterers. They had stutterers and nonstutterers perform a sequential finger tapping task concurrently with a stimulus-contingent foot-responding task. The foot-responding

task involved attending to a tone, and tapping a foot pedal in response to the tone's onset. They found that, in nonstutterers, responding with the left foot interfered with finger tapping more than responding with the right foot. In stutterers no reliable asymmetry was observed. Since the foot-responding task was largely an attentional task, the results were interpreted as consistent with the general idea that stutterers have a more labile system of hemispheric activation or attention. As discussed by Webster (1990b; Forster and Webster, 1991), people who stutter may be similar in this regard to left-handers who, according to Annett's Right-Shift model, lack the gene which gives rise to the directional asymmetry found in right-handers.

The notion that stuttering is associated with an aberrant pattern of hemispatial attention or hemispheric activation raises a number of questions. In particular the distinction between inherent attentional biases and material-specific attentional asymmetries may be especially important. Do people who stutter lack the inherent bias toward the right side of space which right-handers appear to possess? Or do they have a tendency for one or the other hemisphere to become overactive under certain internal (cognitive set, emotional arousal) or external conditions? Certainly, either possibility would go some way toward explaining the unusual laterality patterns demonstrated by people who stutter on dichotic listening and divided visual field studies. A lack (or weakening) of an inherent right-sided bias would lead to an overall inclination toward weaker asymmetries or reversed asymmetries on laterality tasks, depending on the role of other dynamic factors.

It is also possible that cognitive or material-specific attentional factors underlie the anomaly of interhemispheric relations in people who stutter. If an attentional (or activational) bias typically results from the engagement of lateralized cortical mechanisms, then an anomaly in the higher-level processes involved in the organization and integration of appropriate information-processing systems for a given task might lead to atypical laterality patterns. In support of this idea is electrophysiological evidence for overactivation

of the right hemisphere during speech and language tasks (Moore, 1980; 1984; Moore *et al.*, 1982; Moore and Haynes, 1980; Ponsford *et al.*, 1975; Wood *et al.*, 1980). Stuss and Benson (1986) have suggested that the recruitment and integration of different cortical information-processing modules is one important function of the prefrontal cortex.

It is also plausible that the corpus callosum could underlie an anomaly in overall patterns of hemispheric activation. It is unlikely that an attentional bias developed phylogenetically as a result of evolutionary pressure to attend more or less to one side of space. Rather, it is more likely that it represents an artifact of the asymmetric distribution of attentional mechanisms and/or interhemispheric communication in the brain. In this sense, it is unlikely that a lack of an attentional asymmetry itself would be problematic. However, an anomalous attentional asymmetry might be reflective of aberrant interhemispheric relations which, themselves, could potentially contribute to stuttering. This is certainly consistent with evidence that deficits in vigilance and sustained attention are a lasting consequence of corpus callosotomy (Dimond, 1976; 1979). In fact, Levy (1985) has argued that diminished attentional capacity and attentional regulation may be the deficits with the most serious consequence for overall psychological functioning in callosotomized patients. Levy (1985) stated further that the corpus callosum is "of central importance in attentional and regulatory functions involved in sensory processing, motoric action, emotion and cognition" (p. 13).

There is no reason to suggest that an anomaly of hemispatial attention or hemispheric activation should necessarily be considered pathological or maladaptive in any sense. In fact, it may be a manifestation of normal variation in the attentional or activational mechanisms of the human brain. A less asymmetrical pattern of cerebral organization has been associated with left-handedness (Bryden, 1982). Left-handers have, on average, a larger corpus callosum than right handers, and this has been related to the notion that they have a more bihemispheric and less asymmetrical representation of cognitive functions than

right-handers (Witelson, 1985). As discussed earlier, Peters (1987; 1988) has suggested that an attentional bias to the right side of space underlies handedness (and footedness) and that, in accordance with Annett's model of handedness, left-handers lack that attentional bias.

While it is possible that a similar mechanism may be involved in people who stutter, it is also possible that there is an entirely different basis to the atypical patterns of attention that are evident in this group. Humphries, Kaufmann and Galaburda, (1991) have speculated, on the basis of post-mortem examination of cortical cytoarchitecture of severe dyslexics (Galaburda and Kemper 1979; Galaburda, Sherman, Rosen, Aboitiz and Geschwind, 1985; Humphries *et al.*, 1990), that the apparent attentional anomalies in this group may be related to a mild pre-natal pathology in the inferior frontal lobe. It would certainly be worthwhile to explore the possibility that a similar basis underlies the attentional anomaly in people who stutter.

It is the proposal here (see also Forster and Webster, 1991) that the anomaly of interhemispheric relations in people who stutter is causally related to the phenomenon of stuttering, but only when present in combination with a speech-motor fragility. The proposed relationship between the fragility of sequential motor control and the anomaly of interhemispheric relations will be discussed in following section.

Summary: The distinction between a lack of an inherent attentional bias and a less stable pattern of hemispheric activation is just one of many complex issues surrounding the proposal that stuttering is associated with an atypical pattern of hemispheric activation. Perhaps most importantly, the issue of what internal and external factors influence hemispheric activation needs to be addressed. Of particular interest are the issues of what brain systems are responsible for modulating patterns of hemispheric activation and attention, and how such mechanisms might be altered in people who stutter. The answers to these questions are predicated on a clear understanding of the precise nature of the

anomalies of hemispheric attentional mechanisms. Unfortunately, our understanding of hemispheric attentional processes in normal individuals is unclear at best, and the situation is even more vague as it relates to stuttering.

Nevertheless, a strong argument can be made on both empirical and theoretical grounds that attentional factors do contribute to the expression of lateral asymmetries. Since speech and handedness are lateralized normally in people who stutter, perhaps the best interpretation of the atypical perceptual and motor asymmetries observed in this population is that they reflect the influence of attentional factors. This interpretation is supported by a small body of direct empirical evidence of irregular or anomalous attentional processes in people who stutter (Blood *et al.*, 1986; Forster and Webster, 1991; Webster, 1987; 1990a; 1990b). However, until the proposition receives more direct experimental support, the nature of the anomaly of interhemispheric relations in people who stutter will remain unclear. One purpose of the present research was to test the hypothesis that attentional factors underlie the anomaly of interhemispheric relations evident in people who stutter. To this purpose, a divided visual field task was administered to stutterers and nonstutterers, and an attentional precue was employed in a manner similar to that which was used by Mondor and Bryden. In addition, both linguistic and visuo-spatial tachistoscopic target stimuli were used, in an effort to explore the context-specificity of the anomaly of interhemispheric relations in people who stutter. These tasks will be described in greater detail later in the Introduction.

III. The Dual Factor Model of Stuttering in Persistent and Transient Childhood Stuttering

Overview of the Dual Factor Model

There are at least two different conditions, each of which has traditionally been considered in isolation of the other, that appear to be associated with stuttering in adults.

The dual factor model essentially constitutes an effort to marry our understanding of those two conditions in order to generate a broader account of stuttering in terms of neuropsychological, as well as situational and psychosocial considerations. As described by Forster and Webster (1991), the dual factor model contains three major elements. The previous sections of this thesis have attempted to address those points: (1) the brain mechanisms that underlie speech and language are lateralized in people who stutter as they are in the general population (i.e. the cerebral dominance hypothesis is not supported by the available evidence); (2) people who stutter appear to demonstrate an anomalous pattern of interhemispheric relations, possibly related to lateralized attentional mechanisms; and (3) people who stutter have a fragility in the neuromotor mechanisms underlying the organization and initiation of complex sequential responses. Neither of the two components of the dual factor model is believed to represent a condition that would be sufficient alone to produce the extreme manifestations of stuttering that may present in severe adult stutterers. This accounts, in part, for the conspicuous use of the terms "anomaly" and "fragility," to the exclusion of terms such as "deficit" or "disorder," when describing the two characteristics.

An important feature of the model is that the attentional and speech-motor components are seen as having a reciprocally interactive relationship with one another. One of the most notable characteristics of stuttering is the variability in both the frequency and severity of its most basic symptoms. Fluency may vary widely from moment to moment and from situation to situation. Anxiety and negative emotional arousal have a well-known and well-documented affect on the fluency of the person who stutters (Bloodstein, 1981; Trotter and Bergmann, 1957; Van Riper, 1937; 1982). Caruso, Chodo-Zajko, Bidinger and Sommers (1994) measured the effect on articulatory coordination of cognitive stress, and reported that stutterers' speech was more adversely affected by the stress than was the speech of nonstutterers. Other internal phenomena such as distraction (Bloodstein, 1981)

and ineffective self-talk (Webster and Poulos, 1989) also appear to have a significant influence on fluency. An advantage of the model outlined above is that it addresses the variability in the expression of stuttering in terms of a dynamic relationship between attentional and speech-motor mechanisms.

On the one hand, an anomaly of interhemispheric relations could be capable of affecting fluency indirectly in a number of ways, depending on the precise nature of the anomaly. For example, a hemispheric attentional anomaly of the kind described by Kershner and Morton (1990; Kershner, 1995) in dyslexics, could lead to overactivation of the right hemisphere during situations where left hemisphere activation is required. Evidence of right hemisphere overactivation during a variety of language tasks in people who stutter was reviewed earlier. Inappropriate or excessive right hemispheric activation could influence the speech-motor mechanisms in the left hemisphere and supplementary motor area in at least two different ways. First, processing resources ("attention") would be drawn away from speech-motor mechanisms, increasing their susceptibility to neural interference. Second, overactivation in the right hemisphere could directly interfere with speech-motor mechanisms, through both intrahemispheric and interhemispheric spread of activation into the areas that mediate speech-motor control. Either of these mechanisms could operate independently, or both might be involved.

In either case, one may postulate a mechanism whereby a hemispheric attentional anomaly that led to right hemispheric overactivation could exacerbate a speech-motor fragility to the point where it resulted in an actual breakdown in fluency, or perhaps the sensation of an impending breakdown in fluency (i.e. covert stuttering). A more generalized attentional lability or a problem with the organizing mechanisms of the frontal lobe could affect speech-motor control by similar or analogous mechanisms. By leading to the activation of inappropriate information-processing systems, or overactivation of complementary mechanisms, resources could be diverted from speech-motor mechanisms

or interfering activation could be generated. Either of these mechanisms could also presumably lead to a breakdown in the fragile mechanisms underlying sequential motor control in people who stutter.

On the other hand, a speech-motor breakdown may be able to, indirectly, influence attentional mechanisms and overall patterns of hemispheric activation. As discussed by Van Riper (1982) and Bloodstein (1981), the act of stuttering may be associated with fear on the part of the individual who stutters. Similarly, sounds, words or situations may also become associated with fear and anxiety. It is clear that negative affect of this kind is able to influence patterns of hemispheric activation (Ahern and Schwartz, 1985; Davidson and Fox, 1982; Etcoff, 1986; Fox and Davidson, 1988; Gianotti, 1972). Thus, a disfluency or the sensation of an impending disfluency could potentially trigger the right hemisphere activation that would ultimately worsen the disfluency. According to this idea, the speech-motor and attentional components of the model stuttering exist in a positive feedback relationship with one another. Webster (1990) has described this situation as a "neural amplification system." This model provides a valuable heuristic device through which one can account for a severe and potentially crippling speech disorder in terms of a relatively subtle speech-motor anomaly and a coexistent anomaly of interhemispheric relations. Blockages and the struggle described by Wingate (1964b) may be seen as a manifestation of that neural amplification system.

Separability of the Factors

Traditionally, organic explanations of stuttering have referred to a single underlying cause for the disorder: aberrant interhemispheric relations and an impairment of motor sequencing are two such examples that have been discussed here. The dual factor model invokes *two* underlying causes that interact with one another to give rise to the manifestation of stuttering. It is possible, however, that the two factors described in the

model reflect different manifestations of a common underlying structural or functional anomaly.

In fact, there are a number of parallels between the two components of the dual factor model that suggest the possibility of a common basis or link between the two components. For example, some authors (e.g. Verfaellie *et al.* 1988) consider "intention" to be one sub-component of a larger construct for attention. Intention was defined by Verfaellie *et al.* (1988) as the mechanism by which one selectively prepares for action - a "readiness to respond." The term "intention" has also been used to describe the function of the SMA (Deiber *et al.*, 1991; Fried *et al.*, 1991; Goldberg, 1985), and it has been suggested here that stuttering may involve a difficulty translating intention to action. The relationship between the concept of intention as a component of attention and the early onset readiness potential associated with the SMA (Deecke, 1987) also provides an interesting source of speculation.

The commonality goes beyond the use of similar terminology, however. Fried *et al.* (1991) noted that unilateral stimulation of the right SMA influenced limb movements bilaterally, while stimulation of the left SMA elicited responses almost exclusively from the right limb. The authors proposed that their results might reflect right hemisphere dominance for bilateral motor intention. They noted the similarity between their observations regarding bimanual coordination and the more bilateral representation of attentional space in the right hemisphere (Heilman and Van den Abell, 1979; 1980; Mesulam, 1981; Verfaellie *et al.*, 1988). The fact that the right hemisphere has a more bilateral representation of attentional space than the left hemisphere has been proposed as a possible neuropsychological basis for attentional asymmetries. It is thus interesting that Fried *et al.*, (1991) speculated that "the right supplementary motor cortex may be part of the right cerebral system dominant for attentional and intentional mechanisms directed at the body and the external space in which motor activity takes place."

The corpus callosum provides a second, and somewhat less esoteric, commonality between the two components of the dual factor model. It is possible that an anomaly in the corpus callosum could manifest itself in two separate functional neuropsychological systems. According to this idea, the two components of the dual factor model represent distinct functional systems, but arise out of a common underlying structural anomaly. This possibility is particularly appealing in light of the well-documented roles of the corpus callosum in both the functioning of the SMA (Fried *et al.*, 1991; Preilowski, 1972; 1975) and interhemispheric relations (Dimond, 1976; 1979; Levy, 1985; Moscovitch, 1986).

While it was not a primary purpose of the research, the design employed here does offer an opportunity to address the issue of the separability of the two factors in the dual factor model. This is because, as will be described below, it was expected that transient childhood stutters would demonstrate only one of the two factors described in the model. This would, of course, not be possible if the two conditions were merely different manifestations of a single underlying etiology. This idea will be discussed further in the Discussion.

Developmental Implications of the Dual Factor Model

Overview

One of the primary purposes of the present research was to investigate the relationship between transient childhood stuttering and stuttering that persists into adulthood. As discussed earlier, a substantial proportion of children who stutter will eventually stop stuttering before reaching adulthood. The exact rate of recovery is unclear from the literature, but may be as high as 80% (Andrews and Harris, 1964; Andrews *et al.*, 1983). One principle that has emerged from the literature is that the likelihood of spontaneously recovering from stuttering decreases with age, and becomes very small after puberty or early adolescence (Andrews *et al.*, 1983). This fact suggests that recovery from

stuttering may be linked to one or more of the many important developmental changes that occur in childhood and early adolescence. However, very few attempts have been made to investigate neurodevelopmental influences on stuttering (Greiner and Fitzgerald, 1992; Sommers *et al.*, 1975).

The neuropsychological model of stuttering described above was developed on the basis of research with adults, and was intended to refer only to persistent stuttering. However, it would be of both theoretical and clinical interest to attempt to extend the model to childhood stuttering, and to the relationship between transient childhood stuttering and stuttering that persists into adulthood. It would be difficult to address that issue by studying children who stutter directly because there is currently no reliable means of identifying those children whose stuttering will persist into adulthood.

The present research was intended to be a step in that direction, by allowing for a comparison of adults who stutter with adults who stuttered as children but who no longer do. The research used this approach to test the hypothesis that an anomalous pattern of hemispheric activation is a predisposing factor to stuttering in children. Evidence will be reviewed below suggesting that the speech-motor system may be "fragile" in all young children, in that the neural systems underlying it (particularly frontal lobe motor systems and the corpus callosum) do not reach maturity until late childhood or early adolescence. Thus, one of the two factors which, according to the model, are necessary for the expression of stuttering, may be met in all children as a part of the normal developmental course. The other factor, an anomaly of interhemispheric relations, would thus predispose the child to stuttering during childhood. According to this idea, these children would stutter temporarily but would grow out of it as the relevant speech-motor control mechanisms reached maturity.

A smaller population of children may possess a speech-motor system that will never operate as efficiently as it does in normal adults; this is the group of individuals in whom

the stuttering will persist into adulthood. This could presumably relate to a developmental anomaly: they may simply fail to achieve the normative developmental endpoint with respect to the speech-motor control mechanisms that are involved in stuttering. Another possibility is that the inability of these individuals to achieve that normative endpoint may reflect a mild form of focal structural damage to the neural mechanisms that underlie the speech-motor processes involved. A third possibility is that these individuals simply fall at one extreme of the continuum of normal variability with respect to those speech-motor control mechanisms.

Speech-Motor Control

The proposition that the expression of stuttering in some children is linked to the development of speech-motor control mechanisms has face validity for a number of reasons. Perhaps most importantly, it is clear that many aspects of speech and nonspeech motor control continue to develop throughout childhood (Connolly and Stratton, 1968; Denkla, 1973; 1974; Fagard and Pezé, 1992; Kolb and Fantie, 1989; Peters, 1983; Qvarnström, Jaroma and Laine, 1994; Robbins and Klee, 1987; Sharkey and Folkins, 1985; Watkin and Fromm, 1984). Moreover, both the corpus callosum and the frontal lobes continue to develop throughout childhood both structurally (Goldman-Rakic, Isseroff, Shwartz and Bugbee, 1983; Witelson and Kigar, 1988; Yakolev and Lecours, 1967) and functionally (Jeeves, Silver and Milne, 1988; Kolb and Fantie, 1989; Lazarus and Todor, 1987; O'Leary, 1980; Passler, Isaac and Hynd, 1985; Salami, 1978; Shute and Huertas, 1990). Not only are these structures clearly important to the development of motor control, but they are critical for the aspects of speech-motor control implicated in stuttering in this paper. The supplementary motor area is a frontal lobe structure that involves extensive interhemispheric connections via the corpus callosum. The corpus callosum itself may be important for the precise integration and sequencing of the bilaterally

paired speech apparatus. The presence and timing of transient childhood stuttering suggests that it could be related to the development of these neural mechanisms.

The following discussion will focus on the development of speech-motor control mechanisms, and will rely on the assumption that the developmental trends evident in a number of nonspeech motor processes are relevant to the speech-motor control mechanisms involved in stuttering.

The proposal here is that the developmentally immature speech-motor control mechanisms of young children might be relatively "fragile" compared to those of adults. One possibility is that the cortical networks that mediate speech-motor control may be poorly differentiated in children as compared to adults (Peters, 1983), resulting in a susceptibility to interference or overflow from activity in adjacent cortical areas. One way to assess this idea is the measurement of motor overflow or associated movements. Associated movements are "movements accompanying a motor or intended motor function but not necessary for its performance" (Connolly and Stratton, 1968, p. 49). Associated movements often occur in a muscle group that is contralateral and homologous to the one involved in the intended movement, but this is not necessarily the case. Most authors believe that associated movements reflect the overflow of activation from the cortical region representing the intended movement to adjacent or homologous regions (Cohen, Taft, Mahadevia and Birch, 1967; Fog and Fog, 1963; Kinsbourne and Hicks, 1978; Lazarus and Todor, 1987; 1991; Wolff, Gunnoe and Cohen, 1983). This is generally consistent with the observation that associated movements occur most frequently and to the greatest degree in muscles which are mediated by regions either physically adjacent to those that mediate the intended movement (i.e. the adjacent finger) or contralateral and homologous to them (Connolly and Stratton, 1968; Fog and Fog, 1963; Kinsbourne and Hicks, 1978). The degree to which associated movements can be inhibited may thus represent the degree of differentiation of the relevant cortical areas. The frequency of associated movements

decreases with age, and stabilizes in late childhood (Cohen *et al.*, 1967; Connolly and Stratton, 1968; Lazarus and Todor, 1987; 1991). In fact, Wolff *et al.* (1983) suggested that associated movements provide a better indicator of a child's "neurodevelopmental state" than chronological age.

The improvement throughout childhood in the ability to suppress associated movements parallels the development of the corpus callosum, in terms of overall size (Witelson and Kigar, 1988) myelination (Yakolev and Lecours, 1967), and speed of conduction (Salami, 1978). The development of the corpus callosum may be just as important for the suppression of associated movements ipsilaterally as contralaterally. Dennis (1976) has argued that the corpus callosum may be essential for the differentiation of the cerebral cortex within the hemispheres. She reported that two individuals with congenital callosal agenesis exhibited greatly elevated levels of ipsilateral associated movements (i.e. movements of adjacent fingers) compared to hydrocephalic and normal controls. Dennis (1976) argued that an inhibitory action of the corpus callosum during development may be essential for progressive sensory and motor differentiation within each hemisphere. Young children have an immature corpus callosum, and, perhaps more importantly, may not have had time for the progressive differentiation within the cerebral hemispheres to take place.

Studies of bimanual coordination in children have shown developmental changes that parallel the trend evident with associated movements. Fagard and coworkers (Fagard, 1987; Fagard and Pezé, 1992; Fagard, Morioka and Wolff, 1985) have used the bimanual crank-turning task developed by Preilowski (1972; 1975) to assess bimanual coordination in children (see also Jeeves *et al.*, 1988). These authors have consistently reported that the ability to manipulate the two limbs independently continues to improve until around 10 years of age. Since the age differences interact with a variety of factors (e.g. visual feedback, ratio of required output from the two limbs) it is clear that the trend does not

simply reflect a trivial age-related improvement. In fact, Jeeves *et al.* (1988) reported that the pattern of performance with and without visual feedback in 6-year old children was more similar to that of surgical and congenital acallosals than 10-year old children or adults.

The bimanual coordination performance of young fluent children is in a number of respects reminiscent of the bimanual coordination deficits evident in adults who stutter. As discussed earlier, Greiner *et al.*, (1986) and Webster (1988) reported an elevated tendency for adult stutterers to make unintentional mirror-reversals of letters with their nondominant hand during bimanual handwriting performance. Greiner and Fitzgerald (1992) had child stutterers and fluent controls perform a number of variations of the same bimanual handwriting task. Three different age groups of children performed the task: 6 to 8, 9 to 11 and 12 to 15. In the fluent controls, mirror-reversals were common in the youngest age group, but were diminished by over 90% in the 9 to 11 year old group, and were almost totally absent among the oldest group. These data are significant on their own, in that they concur with the developmental trends observed for associated movements and bimanual coordination (Cohen *et al.*, 1967; Connolly and Stratton, 1968; Fagard, 1987; Fagard and Pezé, 1992; Fagard, Morioka and Wolff, 1985; Jeeves, Silver and Milne, 1988; Lazarus and Todor, 1987; 1991; and with the known time-course of callosal development (O'Leary, 1980; Salami, 1978; Witelson and Kigar, 1988; Yakolev and Lecours, 1967).

Collapsed across all three age groups, the stutterers produced over three times as many mirror-reversals as the nonstutterers. Interestingly, the youngest group of stutterers did not produce significantly more reversals than the controls of the same age (though there was a trend in that direction). However, the dramatic improvement noted in the older age groups of fluent children was not present in the performance of the stutterers. They showed essentially no improvement from the youngest to the oldest age group. These findings are particularly interesting because they indicate that, while performance on the bimanual handwriting task is able to clearly differentiate the two groups at older ages, it

does not differentiate young children who stutter from their fluent peers. This observation would appear to support the idea that a developmentally immature speech-motor system may be sufficient to trigger stuttering in some children. The statistically nonsignificant trend towards more mirror reversals in the youngest group of stutterers may reflect the contribution of the minority whose stuttering is more severe and destined to persist into adulthood.

Greiner and Fitzgerald (1992) concluded that interhemispheric communication remains immature through at least 15 years of age in people who stutter. However, the inability to differentiate between transient and persistent stutterers in the younger age groups in their sample creates a problem for interpretation of their data. The three age groups are almost certainly composed of differing proportions of transient and persistent stutterers. Interpretation of the developmental trend (or lack of one) across age groups of stutterers is therefore problematic. It is possible, for example, that the lack of a developmental trend in the stutterers may reflect a growing proportion of persistent stutterers in the older age groups, which could effectively cancel out any age-related improvements that might exist in transient childhood stutterers. The data do indicate, however, that as stuttering persists into later childhood and adolescence, bimanual coordination deficits become more predominant.

Three major inferences can be drawn from the data of Greiner and Fitzgerald (1992): (1) the developmental trend for performance of the task in fluent controls closely follows the established time-course for bimanual coordination and callosal development; (2) the mechanisms underlying bimanual coordination are immature in young children who stutter, but apparently no more so than in fluent children of the same age; and (3) compared with their fluent peers, adolescents and young adults who stutter do appear to be delayed or blocked developmentally with respect to those brain mechanisms.

Thus, children who stutter appear to be similar to their fluent peers, though "impaired" relative to fluent adults on the bimanual handwriting task. It is perhaps significant that similar findings have been obtained from studies directly comparing the speech-motor coordination of young children who stutter with their fluent peers. Conture, Colton and Gleason (1988), and Caruso, Conture and Colton (1988) have examined various aspects of the temporal coordination of fluent speech utterances of young children (the mean ages in the two studies were approximately four and five years of age, respectively). They identified no differences between the two groups on the measures they employed. This is particularly noteworthy in light of the fact that, in a separate study (discussed earlier), the first author of the second study mentioned above (Caruso *et al.*, 1988) did observe differences in the stability of the temporal coordination of fluent speech in *adult* stutterers and nonstutterers. Similarly, Howell, Sackin and Rustin (1995) recently reported speech-motor coordination differences between children who stutter and their fluent peers in an older age range (7 - 12 yrs.). Taken together, these studies suggest what Greiner and Fitzgerald (1992) observed in bimanual coordination: The speech-motor control mechanisms of young children who stutter are indistinguishable from those of their fluent peers, but differences between the groups do emerge as the two populations age, presumably reflecting a greater proportion of persistent stutterers among the heterogeneous samples of "stutterers" with increasing age.

In light of these observations, it is perhaps significant that many "normal" preschoolers experience a brief period of "developmental disfluency" (Andrews *et al.*, 1983; Bloodstein, 1981; Johnson *et al.*, 1959; Yairi, 1982), and that most "stutterers" of this age will eventually recover (Andrews and Harris, 1964). Whether developmental disfluency is qualitatively different from early stuttering is unclear and a matter of some controversy (Andrews *et al.*, 1983). However, the basic characteristics of early stuttering

and developmental disfluency appear to be similar (Bloodstein, 1970; Westby, 1979; Yairi and Clifton, 1972), suggesting a continuity between them.

Qualitatively, both early stuttering and developmental disfluency are different in a number of respects from the disfluency which is associated with the speech of older children and adults who stutter. First, prolongations and blockages of sounds are not typically present in either early stuttering or early normal disfluency (Johnson *et al.*, 1959; Bloodstein, 1960a; 1960b). The disfluencies are limited largely to repetitions of syllables and whole-words in both groups. Second, the struggle and associated accessory activities that are so characteristic of adult stuttering are not present in early forms of disfluency. Many "spontaneous recoveries" occur at this age when the disfluency is considered to be stuttering (Andrews and Harris, 1964; Bloodstein, 1981). The longitudinal picture data of Andrews and Harris (1964) reveal a group whose period of stuttering was so early (age 2-5) and so brief (from 3 months to 1 year) that the authors referred to them as "developmental stutterers."

It is appealing to speculate that both early stuttering and early normal disfluency may reflect an underlying state of immaturity of the speech motor mechanisms. This idea is certainly consistent with the data of Greiner and Fitzgerald (1992). It is also consistent with the observation that both the prevalence of stuttering (Andrews and Harris, 1964) and the severity of normal disfluency (Yairi, 1982) diminish as those mechanisms progress toward maturity.

Interhemispheric Relations and Attentional Mechanisms

Evidence relating to the role of development in the anomalous interhemispheric relations of people who stutter is less clear and more difficult to assess than is the case with speech-motor control. One study has attempted to examine cerebral dominance in children and adults who stutter. Sommers *et al.* (1975) assessed dichotic listening ear preferences in children, adolescents and adults. In agreement with the literature in general, they

2

PM-1 3½"x4" PHOTOGRAPHIC MICROCOPY TARGET
NBS 1010a ANSI/ISO #2 EQUIVALENT

reported that: (1) while there was an overall right ear advantage in both stutterers and nonstutterers, (2) the stutterers had a weaker right ear preference on the task; (3) and there was a higher proportion of stutterers than nonstutterers with atypical ear preferences. Most significantly, however, the authors reported that child stutterers had significantly smaller right ear preferences than the adolescent and adult stutterers. The authors related their findings to recovery from stuttering in children, suggesting that as one hemisphere emerges as dominant recovery from stuttering becomes more likely. This conclusion is clearly incompatible with the position taken here with respect to the cerebral dominance hypothesis. It is also incompatible with more recent evidence pertaining to the development of cerebral dominance. Converging evidence from a variety of sources indicates that hemispheric asymmetries are present from birth and do not increase in magnitude over time in normal individuals (Bryden and Allard, 1981; Dennis and Kohn, 1975; Dennis and Whitaker, 1976; Hiscock and Kinsbourne, 1977; 1980; Ingram, 1975; Kinsbourne, 1975; 1989; Molfese, Freeman and Palermo, 1975; Vargha-Khadem *et al.*, 1985; Witelson, 1970; Woods and Carey, 1978; Woods and Teuber, 1973).

As an alternative to the conclusions of Sommers *et al.* (1972), one might propose that recovery is in some way related to the development of the hemispheric attentional mechanisms. On the surface, this proposition appears unlikely to be correct. As with structural hemispheric asymmetries, attentional asymmetries can be demonstrated in young children and do not appear to increase with age (Geffen, 1978; Geffen and Sexton, 1978; Hiscock and Kinsbourne, 1977; 1980; Obrzut, Mondor and Uecker, 1993). On the other hand, selective attentional processes may be influenced by development independently of lateral asymmetries (Beckie and Hiscock, 1991; Geffen and Wale, 1979). Geffen (1978; Geffen and Wale, 1979), for example, has found that the ability to overcome the right ear advantage for consonant-vowel recognition improves with age in children between six and eight years of age. She argued that control of selective attention to one ear improves with

development, while structural asymmetries remain constant. Developmental changes in the ability to direct attention to one or the other ear may relate to the maturation of the corpus callosum and its role in the integration of the two hemispheres. Consistent with this idea, Molfese *et al.* (1975) reported that asymmetries in auditory evoked responses to speech and nonspeech stimuli decreased with age. They interpreted that finding in terms of increasing interaction between the cerebral hemispheres with maturation of the corpus callosum.

The development of the corpus callosum and its proposed roles in interhemispheric interaction (Molfese *et al.*, 1975) and the unification of attentional processes (Ellenberg and Sperry, 1979; Gazzaniga, 1987; Levy, 1985) certainly leave open the possibility that attentional mechanisms play a role in the expression of stuttering in children. It is interesting, in this context, to contrast the findings of Sommers *et al.* (1975) with those of Greiner and Fitzgerald (1992). Relative to the performance of fluent children, the bimanual coordination deficits noted by Greiner and Fitzgerald (1992) became more prominent with increasing age. The reverse pattern was evident in the dichotic listening data of Sommers *et al.* (1975). Among the stutterers, the youngest children had weaker (i.e. less normative) asymmetries than the adolescents and adults. Given the developmental invariance of both structural and attentional asymmetries in the general population, it is unlikely that dichotic listening asymmetries increase with age in people who stutter. A more reasonable interpretation of the data of Sommers *et al.* (1975) is that they reflect differences in the composition of the three age groups of stutterers with respect to transient and persistent stutterers. The youngest group would be expected to include a substantially greater proportion of transient stutterers. That same group demonstrated a weaker dichotic listening asymmetry than the other groups. This observation, in light of the data of Greiner and Fitzgerald (1992) is consistent with the idea that the proposed anomaly of interhemispheric relations may be sufficient to induce stuttering in young children in whom the speech-motor system is immature.

One question that has not been addressed relates to the variability in the age of recovery. The developmental model proposed here is not intended to provide a complete account of the factors that mediate recovery from childhood stuttering. It is likely that a variety of environmental and psychosocial factors interact with the mechanism proposed here to determine the developmental course of an individual's stuttering. It is fundamental to the original dual factor model that this kind of individual and temporal variability is able to influence the expression of stuttering. Applied to transient stuttering, the situation becomes even more dynamic and hence more complex. There are likely to be considerable variations across individuals in the time course of speech-motor development and the nature and degree of the attentional anomaly. Learned associations with sounds, words, places and situations could influence the expression of a child's stuttering. The influence of these associations could in turn influence the time and perhaps even extent of the recovery. The child's self-image and attitude toward his stuttering could play a similar role in expediting or delaying recovery. The essential feature of the developmental model proposed here is that it proposes changes in the underlying neural basis of the disorder which ultimately permit that recovery.

Summary: An extension to the model of Forster and Webster (1991) has been proposed in an attempt to account for the high rate of recovery from stuttering in childhood. This extension to the model has three major elements: (1) at the time of speech onset, the neural mechanisms that underlie the aspects of speech-motor control that are relevant to stuttering are immature in all children, and are thus fragile and poorly differentiated compared to those of adults, (2) because of the immaturity of the young speech-motor system in all children, a hemispheric attentional anomaly may be sufficient to trigger transient childhood stuttering, (3) the stuttering would be expected to improve with maturation of the speech motor system. The evidence for this model is largely indirect and speculative. The present research was intended to be a more direct test of the model.

Rationale of the Research

The research involved a series of four tasks intended to investigate both the attentional anomaly and speech-motor control. In order to address the issue of recovery, the same three groups of participants performed all four tasks: (1) adults who stutter ("stutterers," ST); (2) adults who stuttered as children but have recovered ("exstutterers," ES); (3) a control group of individuals who have never stuttered ("nonstutterers," NS). Of the four tasks, two were intended to assess the speech-motor fragility, and two were intended to assess the anomaly of interhemispheric relations. According to the model described here to account for transient childhood stuttering, the speech-motor tasks were expected to differentiate the persistent stutterers from the other two groups. The anomaly of interhemispheric relations, which was assessed in terms of the size and direction of the performance asymmetry on two divided visual field tasks, was expected to differentiate *both* the stutterers and ex-stutterers from the control group.

Stated differently, the following hypotheses were made with respect to the performance of the three groups on the tasks:

Speech-motor control tasks: $(NS = ES) > ST$

Divided visual field asymmetries: $NS > (ES = ST)$

The four tasks chosen for the research will be outlined briefly below.

IV. Summary of the Research and Hypotheses

Motor Tasks

Two of the tasks were intended to probe the speech-motor mechanisms that were implicated in stuttering. The tasks were selected to sample three speech-motor control functions which appear to be impaired in adults who stutter: organization of complex

sequential responses, initiation of complex sequential responses, and bimanual coordination. These functions are of special interest for a number of reasons that have already been reviewed.

First, it appears that stuttering as a speech disorder is at its root a disorder of sequencing or timing. Second, the tasks that have been chosen reflect aspects of speech-motor control that have been shown to be impaired in the manual (i.e. non-speech) performance of adults who stutter. Third, the functions being assessed all appear to be functions which are, at the cortical level mediated predominantly by the supplementary motor area, a region in which there is considerable overlap between speech and non-speech motor control. Finally, both manual sequencing and bimanual coordination have been shown to improve with age in children, with the former reaching a plateau at approximately 7 to 8 years of age (Denkla, 1973; 1974) and the latter continuing to improve until around 10 years of age (Fagard, 1987; Fagard and Pezé, 1992; Jeeves *et al.*, 1988).

One task was a variation of Webster's (1986a) Sequence Reproduction Task. This task assessed the subject's ability to rapidly initiate finger-tapping sequences. Participants were required to initiate, as quickly and as accurately as possible, finger-tapping sequences that were presented visually just prior to onset of the signal to initiate the sequences. Once initiated, the sequences were repeated, as rapidly and as accurately as possible, for five seconds following their initiation. The Sequence Reproduction Task assessed two of the speech-motor control functions summarized above. Response onset latencies on this task assessed the ability to rapidly initiate complex response sequences. Response execution time, and accuracy of initial responses (the first sequence performed following the signal to initiate tapping) provides a measure of the accuracy of sequential response organization.

The design used for the Sequence Reproduction Task extends the design employed by Webster (1986a) by including Sequence Length as an independent variable. This has the advantage of providing some additional information, relevant to the exclusion of the

cerebellum as a potential neural locus for the motor sequencing deficits observed in adults who stutter. Based upon previous research (Inhoff, Diener, Rafal and Ivry, 1989; Sternberg, Monsell, Knoll and Wright, 1978) it is expected that response onset latency should increase with increasing sequence length, and there is no *a priori* basis to expect this effect to differ among the groups. Sternberg *et al.*, (1978) reported that response onset latencies for sequential responses increase linearly with the length of the required sequence. Inhoff *et al.*, (1989) reported that cerebellar damage abolishes this sequence length effect. Thus, if the sequencing deficits observed in people who stutter are related to the functioning of the cerebellum, then the group differences observed on the Sequence Reproduction Task should be sensitive to sequence length.

With respect to accuracy and response onset latencies on the Sequence Reproduction Task, it was expected that the same pattern of results found by Webster (1986a) would be observed in the adults who stutter and the fluent controls. Stutterers were expected to demonstrate longer overall response initiation times than nonstutterers, and make more first sequence errors than nonstutterers. According to the hypothesis that transient childhood stuttering is a reflection of a developmentally immature but normal speech-motor system, the ex-stutterers were expected to perform similarly to normal controls with respect to the performance measures on the Sequence Reproduction Task.

The second motor task was a variation of Preilowski's (1972; 1975) bimanual crank-turning task (Forster, 1993). This task involved having participants turn two cranks to manipulate a "pen" that was displayed on a computer screen. Similar to the "Etch-a-Sketch" toy, each crank controlled movement in one axis of movement (up-down, or left-right). The task was to manipulate a cursor through a 2 cm x 15 cm track presented at two different angles chosen such that one hand (the "lead hand") was required to generate twice the output of the other ("nonlead") hand. The lead hand was varied across trials. After correct performance of 16 trials with visual feedback, participants performed eight trials in

which visual feedback was eliminated halfway through the track ("no-visual-feedback" trials).

Preilowski (1975) demonstrated the importance of the anterior corpus callosum on the bimanual crank-turning task. Since the SMA is known to have extensive interhemispheric connections via the corpus callosum (Rouiller *et al.*, 1994) and appears to be important for bimanual coordination, it is likely that the task relies heavily on interhemispheric connections between the left and right sides of the SMA. Preilowski (1975) found that performance of the task without visual feedback (i.e. based solely upon kinesthetic feedback) requires that the anterior corpus callosum be intact, whereas processes that rely on the posterior corpus callosum are sufficient to guide performance when visual feedback is available.

In the Bimanual Crank-turning Task, the degree to which the participant's actual trace deviates from a straight line through the centre of the track provides a measure of the overall accuracy of performance on the task. It is also possible to detect "reversal" errors on the task, as represented by the amount of excess output (i.e. crank movements in the wrong direction) generated. Based upon the bimanual handwriting data of Webster (1988) and Greiner *et al.* (1986; Greiner and Fitzgerald, 1992), persistent stutterers were expected to generate less accurate traces and more reversal errors than fluent controls and ex-stutterers.

However, the Bimanual Crank-Turning Task would appear to be more difficult than bimanual handwriting, and should thus pose more of a challenge to the neural mechanisms that mediate bimanual coordination. In addition, bimanual handwriting involves an extensively practiced and overlearned skill (handwriting), whereas the Bimanual Crank-Turning Task is essentially a novel task, excepting any childhood experience with the Etch-a-Sketch (or other similar devices). This distinction may be particularly relevant in the no-visual-feedback condition. By eliminating visual feedback midway through a trial,

participants were required to rely more heavily on kinesthetic feedback and hence anterior (presumably SMA) mechanisms (Goldberg, 1985; Preilowski, 1975). For this reason, the performance of the stutterers was expected to deteriorate even further under the no visual feedback condition, relative to control subjects and ex-stutterers.

Divided Visual Field Tasks

Two divided visual field tasks were used to test the hypothesis that both persistent and transient childhood stuttering are associated with an anomaly of interhemispheric relations. One was a lexical decision task, on which nonstutterers were expected to demonstrate a right visual field (left hemisphere) advantage (Beaumont, 1982; Hardyck *et al.*, 1985; Shanon, 1979). Two previous divided visual field studies (Moore, 1976; Hand and Haynes, 1983) have reported anomalous left visual field advantages for verbal tasks in stutterers. The study by Hand and Haynes (1983) involved a lexical decision task similar to the basic task to be used here.

The other divided visual field task was a dot enumeration task, on which nonstutterers were expected to demonstrate a left visual field (right hemisphere) advantage (Kimura, 1966; 1973). According to the extension to the dual factor model described here, both stutterers and exstutterers were expected to generate anomalous asymmetries on the two divided visual field tasks.

The divided visual field tasks contained two design elements intended to provide information relevant to the nature of the anomaly of interhemispheric relations in people who stutter. The first, to which I have already alluded, was the inclusion of the Dot Enumeration divided visual field task. The inclusion of this task constitutes the first attempt to investigate whether people who stutter would generate anomalous performance asymmetries on a task that did not depend on left hemisphere linguistic mechanisms. This allows the study to address the issue of the material-specificity of the anomaly of

interhemispheric relations. If the anomaly is specific to the information processing domain employed by the task, then the stutterers should demonstrate either no anomalous performance asymmetry (i.e. the anomaly is limited exclusively to the verbal/linguistic domain), or a weaker left visual field advantage (i.e. the anomaly manifests itself differently depending on the hemisphere specialized for the required task) on the Dot Enumeration Task. On the other hand, if the anomaly reflects a general attentional bias that is shifted to the left relative to the typical right visual field bias evident in fluent speakers, then one would expect the stutterers to demonstrate a *larger* left visual field advantage on the task.

In addition to the inclusion of the Dot Enumeration Task, there was a second attempt to isolate the nature of the anomaly of interhemispheric relations in people who stutter. Both divided visual field tasks employed a paradigm similar to that described by Mondor and Bryden (1992a) to manipulate the allocation of attention to the two visual fields. For the Lexical Decision Task, a dot precue was presented either simultaneous with ($SOA = 0$ ms), or 67 ms prior to ($SOA = 67$ ms) the onset of the target stimulus (word or nonword). The precue was presented directly above the location of the target stimulus in all cases. For the Dot Enumeration Task, a box that surrounded the random dot pattern was used as the precue, which was presented either simultaneously with ($SOA = 0$ ms) or 50 ms prior to ($SOA = 50$ ms) the onset of the dots. As described by Mondor and Bryden (1992a), it was predicted that there would be a reduction in the performance asymmetry on the lexical decision trials with $SOA = 67$ ms in the nonstutterers, as the precue was expected to minimize the influence of any preexposural attentional biases on the observed asymmetry. It was further expected that the nonstutterers' left visual field advantage on the Dot Enumeration Task should be enhanced, as the influence of the counter-acting right visual field attentional bias is minimized by the precue. Finally, it was predicted that, if the anomaly of interhemispheric relations in people who stutter reflects the influence of

attentional mechanisms, then the precue should attenuate or abolish any observed group differences in the Lexical Decision Task and the Dot Enumeration Task.

Summary of Tasks and Expected Results

Three experimental groups, nonstutterers, stutterers and exstutterers performed four different tasks.

Two of the tasks, a Sequence Reproduction Task and a Bimanual Crank-Turning Task were intended to probe the speech-motor mechanisms believed to be fragile in people who stutter. Since, according to the model of recovery being tested, transient childhood stuttering is *not* associated with a permanent speech-motor fragility, exstutterers were expected to perform similarly to nonstutterers on the two motor tasks.

The other two tasks were divided visual field tasks, intended to probe the anomaly of interhemispheric relations that has been identified in people who stutter. One was a Lexical Decision Task, and the other a Dot Enumeration Task. On the Lexical Decision Task, nonstutterers were expected to demonstrate a right visual field advantage, and stutterers were expected to demonstrate either a weaker or a reversed asymmetry, relative to the nonstutterers. Since, according to the model being tested, transient childhood stuttering is associated with the same anomaly of interhemispheric relations that has been identified in persistent adult stutterers, the exstutterers were expected to demonstrate the same anomalous performance as the nonstutterers on this task. On the Dot Enumeration Task, nonstutterers were expected to demonstrate a left visual field (right hemisphere) advantage. The size and direction of any visual field in the stutterers and exstutterers should depend on the nature of the anomaly of interhemispheric relations. If the anomaly of interhemispheric relations reflects a weaker or reversed attentional asymmetry, then stutterers and exstutterers would be expected to demonstrate a *stronger* left visual field advantage on the task.

A precue stimulus, intended to minimize any pre-exposural attentional biases, was expected to attenuate or eliminate any group differences attributable to the attentional bias. Thus, if the anomaly of interhemispheric relations reflects an anomalous pre-exposural attentional bias, the precue stimulus should have minimized any group differences on the divided visual field tasks.

The predicted patterns of performance on the four tasks are summarized below.

Summary of Hypotheses

Motor Tasks (expressions relate to overall "ability" on tasks)

Sequence Reproduction Task:	$(NS = ES) > ST$
Bimanual Crank Turning Task (with visual feedback):	$(NS = ES) > ST$
Bimanual Crank Turning Task (no visual feedback):	$(NS = ES) >> ST$

Divided Visual Field Tasks (expressions relate to size of expected normative visual field asymmetry*)

Lexical Decision Task (*right visual field advantage):	$NS > (ES = ST)$
Dot Enumeration Task (*left visual field advantage):	$NS > (ES = ST)^{\vee}$ or $NS = (ES = ST)^{\vee}$ or $NS < (ES = ST)^{\vee\vee}$

[∨] if anomaly of interhemispheric relations is specific to the information processing domain associated with the task.

^{∨∨} if anomaly of interhemispheric relations relates to a weaker or reversed attentional bias.

Note: additional hypotheses relating to performance on the divided visual field tasks were described above. These hypotheses pertain to the issue of the nature of the anomaly of interhemispheric relations as reflected in the effect of the dot precue on performance.

METHOD

Participants

Ninety-three right-handed participants were tested. Of these, 29 were nonstutterers, 31 were stutterers, 26 were exstutterers and 7 were of ambiguous classification (four who apparently recovered in adulthood, one in whom recovery was unclear, and two for whom it was unclear whether they had actually stuttered as children). Four participants (one NS, two ST, one ES) were excluded due to age (three too old, one too young). Four participants (one NS, 3 ST) were excluded due to neurological or asymmetrical visual impairments (those with corrected symmetrical visual impairments were not excluded). Three participants (one NS, one ST, one ES) were excluded because they performed one or more of the tasks incorrectly. Two were excluded because of extensive musical training (one NS, one ST), and one (NS) was excluded because of difficulty with English. The remaining participants included 24 in each of the three groups: nonstutterers, stutterers and exstutterers. Each group included 16 males and eight females.

Table 1 shows the mean ages and years of education and the percentage with a positive family history for stuttering in the three groups. The age of the included subjects ranged from 19 to 59 years. A one-way ANOVA indicated that the differences in mean age of the three groups were not statistically significant ($F(2,69) = .99$). Similarly, the groups did not differ significantly in terms of years of education ($F(2,69) = .51$).

As evident in Table 1, a positive family history for stuttering was much more frequent in the stutterers and exstutterers than in the nonstutterers. The groups did differ in terms of the frequency of the reported positive family history of stuttering. Positive family history was defined as at least one brother, sister, parent, grandparent or biological aunt or uncle who stuttered. Nonstutterers had the lowest incidence of a reported positive family

Table 1

Mean (and SD) Age and Education, and Percentage with a Reported Positive Family History for Stuttering in Nonstutterers, Stutterers and Exstutterers.

	Age	Years Education	Family History Stuttering (percent with)
Nonstutterers	33.3 (10.7)	16.3 (2.1)	8.3
Stutterers	37.4 (10.8)	16.6 (2.9)	66.7
Exstutterers	33.8 (11.7)	15.9 (1.2)	29.2

history for stuttering (8.3%), while stutterers had the highest incidence (66.7%).

Exstutterers fell between the other two groups with 29.2% reporting a positive family history for stuttering.

The mean ages of onset and recovery, and the mean self-reported Likert-scale severity of stuttering for the stutterers and exstutterers, is presented in Table 2. The mean reported age of onset was 5.6 years of age, for both stutterers ($SD = 2.2$ yr.) and exstutterers ($SD = 2.9$ yr.). The mean reported age of recovery was 14.6 years of age for the exstutterers, though it should be kept in mind that improvement and recovery was typically described as a gradual process that may have occurred over a period of several years.

Stuttering severity was assessed by questionnaires that asked participants to rate their current and past stuttering on a scale of one to seven, where one represented "very mild" and seven represented "very severe." Self-reported childhood stuttering severity was very similar in the stutterers ($M = 4.7$, $SD = 1.5$) and exstutterers ($M = 4.8$, $SD = 1.7$). Current (adult) stuttering severity for the adult stutterers was 3.5 ($SD = 1.5$).

Table 2

Mean (and SD) Onset, Recovery and Likert-Scale Severity in Stutterers and Exstutterers.

	Life History		Severity	
	Onset	Recovery	Child	Adult
Stutterers	5.6 (2.2)		4.7 (1.5)	3.5 (1.5)
Exstutterers	5.6 (2.9)	14.6 (3.7)	4.8 (1.7)	2.9 ^d (1.5)

"The instructions for exstutterers were "If you stutter at all now (e.g. when you are tired or under stress) please place an X on this scale to indicate how severe you feel your stuttering can be at its worst." The question cannot be properly compared with the scores for the stutterers, for whom the instructions were simply "Please place an X on this scale to indicate how severe you feel your stuttering to be."

Adult exstutterers were given a different version of the current (adult) severity scale, which asked them to rate the specific instances of stuttering if they still experienced them as adults. It is important to note that the lowest value on this scale represented "not at all," which means that the range of actual severity of stuttering instances was effectively limited to two through seven. The mean self-reported severity of stuttering in exstutterers was 2.9 (SD = 1.5). Thus, to compare exstutterers to stutterers, one could say that, when they experienced instances of stuttering, the exstutterers reported those instances as being relatively mild (2.9 on a scale which ranged from 2 to 7, or essentially 1.9 on a scale of 1 to 6), whereas the stutterers' overall perception of their stuttering was that it was of moderate severity.

Each participant was administered two different versions of the Perceptions of Stuttering Inventory (PSI) (Woolf, 1967; see Appendix A). The PSI is a 60-item questionnaire containing statements about speech and speech-related feelings and activities. The respondent is required to either check off an item to indicate that it is "Characteristic of me," or to leave it blank if it is not. The questionnaire contains three subscales, "Struggle," "Expectancy," and "Avoidance." The Struggle subscale includes items that describe speech or accessory activities indicative of the actual speech-related struggle (disfluencies, blocks, accessory facial activities, etc.). The Expectancy subscale contains items indicative of an expectancy on the part of the individual to stutter (i.e. in specific situations, on specific words or sounds, or in general). The Avoidance subscale contains items that represent various different strategies for avoiding sounds, words or situations associated with speaking. Each of the three subscales contained 20 items, and the score on each subscale is simply a score from zero to 20 to represent the number of corresponding items checked off. An overall score (the sum of all items checked off) was also computed.

The first of the two versions administered was the standard PSI, which focused on the individual's current speech. The second version was modified such that the individual was asked to answer the same questions but with respect to their speech in their preadolescent childhood. Both versions of the PSI were further modified for administration to nonstutterers. The nonstutterers' version of the PSI was called the "Perceptions of Speech Inventory," and included all 60 questions included in the standard PSI with only minor modification to eliminate specific references to stuttering.

The scores (number of items checked off out of a total possible of 20) for the three PSI subscales, Struggle, Expectancy and Avoidance, are presented in Table 3. The primary purpose of the PSI was to provide a test of the classification of exstutterers. Ideally, the exstutterers' scores should be similar to the stutterers' scores in the childhood version, and similar to the nonstutterers scores in the adulthood version. Based on these α

Table 3

Mean (and SD) Scores on Struggle, Expectancy and Avoidance Subscales of Perceptions of Stuttering Inventory for Nonstutterers, Stutterers and Exstutterers.

	Childhood			Adulthood		
	Struggle	Expect.	Avoid.	Struggle	Expect.	Avoid.
Nonstutterers	0.9 (1.6)	1.7 (2.4)	4.1 (3.6)	0.6 (1.2)	1.4 (1.6)	2.1 (2.1)
Stutterers	11.5 (5.3)	12.2 (5.6)	15.2 (4.1)	8.8 (4.4)	10.9 (4.9)	8.6 (5.5)
Exstutterers	10.0 (5.0)	11.5 (5.0)	10.8 (4.8)	3.3 (3.6)	5.8 (3.9)	3.1 (3.2)

priori hypotheses, and since a direct comparison of stutterers and nonstutterers on the PSI would be of little interest, two separate 2-way (Group by Subscale) ANOVAs were conducted on the PSI data: one comparing stutterers to exstutterers on the childhood version, and one comparing nonstutterers to exstutterers on the adulthood version.

Nonstutterers, as expected, checked off the fewest items on the PSI ($M = 6.7$ and 4.1 for childhood and adulthood, respectively). Most notably, the majority of the nonstutterers' positive responses were Avoidance items, and almost none were Struggle items (see Figure 1). Stutterers checked off the most items of the three groups, in both the childhood ($M = 38.8$) and adulthood ($M = 28.3$) versions. As expected, exstutterers' overall scores were similar to the stutterers in the childhood version ($M = 32.3$), though inspection of Table 2 will reveal that the two groups appear to be differentiated by the Avoidance subscale (for stutterers, $M = 15.2$, for exstutterers $M = 10.8$). Those observations are confirmed by the results of an ANOVA which included only stutterers and

Figure 1. Childhood-version PSI scores as a function of Subscale and Group for exstutterers and stutterers.

exstutterers: overall, the Group main effect was nonsignificant ($F(1,46) = 3.21, p = .08$) but a Group by Subscale interaction, illustrated in Figure 1, was indicated ($F(2, 92) = 4.75, p < .05$). Analysis of simple effects revealed that the stutterers and exstutterers differed only on the Avoidance subscale ($F(1,46) = 12.00, p < .005$).

In the adulthood version, the exstutterers responses were more similar to the nonstutterers than to the stutterers, but the exstutterers did check off significantly more items ($M = 12.25$) than the nonstutterers (4.04): $F(1,46) = 15.36, p < .0005$. However, as with the childhood version, there was a statistically significant Group x Subscale interaction: $F(2,92) = 12.28, p < .0005$. Inspection of Figure 2 reveals that the groups were most similar on the Avoidance scale, with exstutterers checking off an average of 1.0 more items than the nonstutterers, which was not a statistically significant difference: $F(1,46) = 1.81, p = .185$. Exstutterers checked off 2.7 more Struggle items and 4.4 more Expectancy items than the nonstutterers, both of which were statistically significant differences ($F(1,46) = 12.84, p < .005$, $F(1,46) = 26.42, p < .0005$, for Struggle and Expectancy, respectively).

All participants signed an informed consent form prior to performing the tasks, and all received either a \$40 honorarium, or experimental credit toward Psychology 49.100 at Carleton University, for completing the experiment. Participants were recruited from the university population, via direct appeals to the Psychology 49.100, and from the broader community, via public service announcements on radio, news stories in the radio, television and print media, and through the Communication Disorders Department of the Royal Ottawa Rehabilitation Centre in Ottawa. Participants were aware that the research concerned the issue of recovery from stuttering, but were naive as to the tasks they would be performing until the procedure for each task was explained.

Figure 2. Adulthood-version PSI scores as a function of Subscale and Group for all three groups.

Materials

In addition to the apparatus to be described below, the materials included the Perception of Stuttering Inventory (Wolff, 1967; see Appendix A), the Edinburgh Handedness Inventory (Oldfield, 1971; see Appendix B), and two Likert stuttering severity scales (See Appendix C). Both inventories were self-administered on a computer, while the two severity scales were administered on paper, with separate sheets for each scale (adult severity and childhood severity). All information gathered from the interview (including the values recorded on the two severity scales) was recorded by the experimenter directly to a computer database, illustrated in Appendix D.

Overview of Procedure

The testing session was approximately two and a half hours long. At the beginning of the sessions, participants were given an overview of the kind of tasks they would be performing, and were asked to read and sign the informed consent form (Appendix E). The interview then took place, during which the subject was asked about his or her stuttering (if he or she was a stutterer or exstutterer), family history for stuttering, handedness, neurological history, musical skill and other possible exclusionary criteria (motor skills, visual impairments, etc.). Information on the individual's stuttering included the age of onset and (if applicable) recovery, severity (via the Likert scales, the PSI and self-report), therapy received, factors associated with recovery, and situational variability. All responses were recorded by the experimenter directly into the database illustrated in Appendix D, with the exception of the PSI and the Edinburgh Handedness Inventory (these were self-administered directly onto the computer, and the responses were automatically entered into the database by the computer). Following the interview, the session followed the following order:

Lexical Decision Task -> Sequence Reproduction Task -> Bimanual Crank-Turning Task -> Dot Enumeration Task -> Debriefing

Following the last task, participants were debriefed verbally about the purpose of the research, and the purpose of each individual task. Participants were also provided with a written debriefing, which explained the purpose of the research, the rationale behind each of the tasks, and the hypotheses being tested (Appendix F). Following the debriefing, participants were paid a \$40 honorarium for their participation.

A written protocol, including the interview and the instructions for each task, is included in Appendix G.

Task 1: Sequence Reproduction Task

Apparatus

There were two main parts to the apparatus: the finger-tapping box and a computer. The finger-tapping apparatus consisted of a box 30 cm long, 30 cm deep and 20 cm high, with four telegraph keys mounted 3 cm apart. The finger-tapping box rested on a table 75 cm off the floor. Each telegraph key was interfaced with a Macintosh computer by means of direct wiring to four keys on a computer keyboard. Each tap of the telegraph key was thus recorded by computer as a different key stroke on the keyboard.

An external 14" Magnavox Professional Mac Color Display monitor (resolution, 640 x 480), placed directly behind the finger-tapping box, was used to present the sequences visually to the participants. Presentation of the sequences, and timing and recording of the responses were all controlled by a computer program on a Macintosh SE/30 computer to which the monitor and tapping box were connected. Parameters for each trial were entered by the experimenter prior to the onset of the series of trials, but were

only visible on the SE/30's internal monitor which was oriented to face the experimenter. Four circles (diameter = 1.5 cm) on the screen were arranged, 1.5 cm apart, to correspond to the four keys on the tapping box. A computer program presented specific sequences by briefly "flashing" (blackening) different circles (representing different telegraph keys) sequentially. During presentation of a given sequence, circles were flashed for 200 ms each, with an interval of 200 ms between successive flashes. 200 ms following the last flash in a sequence, a computer-generated tone sounded for 150 ms to indicate that the participant should begin tapping the sequence. A second tone sounded five seconds after the onset of the first tap to indicate the end of the trial. Participants' responses were recorded by the computer. The response onset latency (time from onset of the tone to the first tap) was recorded, and each successive tap (onset only) was recorded along with the time (in "ticks" of the computer's clock, with each tick representing 17 ms) at which it occurred. A separate computer program later used that data to generate a graphical 4-channel "chart recording," and the three dependent measures for each trial.

Procedure

The procedure used was based upon the Sequence Reproduction Task used by Webster (1986b). For this task, participants were asked to initiate tapping the sequence displayed on the computer screen as quickly as possible following the first tone, and to continue tapping the sequence over and over as quickly and as accurately as possible until they heard a second tone indicating the end of the five second trial. The task was demonstrated by the experimenter, and practiced twice by the participant before the test trials began. For each trial, the appearance of the array of circles on the screen indicated that the trial was about to begin. Two seconds later, the sequence was presented visually, followed by the tone (as described in the apparatus section).

There were 36 trials in total, all of which were performed by the right (dominant) hand. Of the 36 trials, there were 12 with three unique elements (e.g. 1-3-2), 12 with four unique elements (e.g. 1-3-4-2), and 12 with five elements (e.g. 2-1-3-4-2). For the sequences with five elements, one element was necessarily repeated. For the 4-element sequences, there were ${}_4P_4 = 24$ possible orders. For 3-element sequences, there were also ${}_4P_3 = 24$ possible orders, which essentially represents each of the 4-element sequences with the last element missing. There were $24 \times 4 = 96$ possible 5-element sequences with one repeated element. Twelve 4-element sequences were selected from the 24 available orders by imposing the criterion that no sequence began with the little finger or the ring finger. Twelve 3-element sequences were chosen by removing the last element from the twelve 4-element sequences. Twelve 5-element sequences were chosen by adding the ring finger to six of the 4-element sequences and the little finger to the other six 4-element sequences. The sequences used are presented in Appendix H. The 36 different trials were presented in the same random order for all participants.

This arrangement had the following advantages: (1) the number of trials being analyzed (12 of each length, 36 in total) was large enough, judging from previous research with the task (Webster, 1986a; Webster, 1989) to generate sufficient statistical power to detect group differences; (2) the first three elements of the sequences were the same across lengths, thus eliminating variability that could have confounded the comparison between sequences of different lengths. This design also allowed the findings reported by Webster (1986a) to be generalized across sequence lengths.

The dependent measures of interest were the Response Initiation Time (computed as the time from the onset of the tone to the onset of the first tap), the Sequence Execution Time for the first three elements of the first sequence (computed as the time from the onset of the first tap to the onset of the fourth tap), and the number of correct first sequences (only the first sequence tapped was considered; scored were the number of first sequences

correct out of a possible of 12 for each sequence length). Sequence Length (3, 4 or 5) was the only within group independent variable, and Fluency Group (Persistent Stutterer, Ex-Stutterer, Fluent Control) was the between group independent variable.

Task 2: Bimanual Crank-Turning Task

Apparatus

The task was performed on a computer-based version of Preilowski's (1972; 1975) X-Y recorder, and was similar in principle to the Etch-a-Sketch toy. The main portion of the apparatus consisted of two cranks mounted side by side (30 cm apart) on a metal chassis (60 cm x 60 cm x 30 cm). The cranks were interfaced with the inner workings of a Microsoft bus mouse which was connected to an IBM PC compatible 386 computer. Turning of the cranks would thus manipulate a cursor on a flat-screen monitor. Movement of the cursor left a trace of the cursor's path on the monitor. Each crank was capable of controlling either up-down or left-right movement of the cursor.

Prior to each trial, a computer program drew a track consisting of two parallel 15 cm lines placed 2 cm apart on the monitor. The track was presented at one of two different angles: 26.5° or 63.5°. The two angles were selected to require motor output from the two hands in a ratio of 2:1. The left crank always controlled movement in the up-down dimension and the right crank controlled left-right movement. Clockwise crank-turning moved the cursor either up or to the right, and counterclockwise moved it either down or to the left. Thus, on all trials, clockwise motion of both cranks was required. At the start of each trial, the "pen," represented by a small dot on the screen, was initially presented at the bottom of the track, one centimeter outside of its entrance. When the pen entered the track (in response to the participant turning the crank) the computer program recorded the pen's x and y positional coordinates and time coordinates (in ticks) approximately 60 times

per second (as long as the pen was moving) until the pen exited through the other end of the track or crossed the plane defined by the end of the track. At the end of the trial, the entire array of x, y, and time coordinates was saved to the computer's hard disk drive. A second computer program was able to read the data, graphically recreate the path drawn by the pen, and compute all the required dependent measures for each trial.

Procedure

Each trial began with the appearance of the track and the "pen" on the screen. Participants were told to begin the trial at their leisure, and that their data would not be recorded until the pen entered the track. They were instructed to try to move the pen through the center of the track as quickly as possible without leaving the track, and to try to keep both cranks turning at the same time, rather than alternating between the two cranks in a stepwise fashion. Participants were also told that when the pen did go outside of the track, the trial would be repeated.

Because of the orientation of the track in the two different track angle conditions, motor output from the two hands was required to be in a 2:1 ratio on all trials. In the "right lead" condition (track angle = 26.5°), the right hand was required to turn twice as quickly as the left hand. In the "left-lead" condition (track angle = 63.5°), the left hand was required to turn twice as quickly as the right hand.

Participants performed 16 correct trials (i.e. without leaving the track) which were organized into four blocks of four consecutive trials with the same lead-hand, as illustrated below. Participants were required to perform four correct trials (though not necessarily consecutively) in each block before moving on to the next block (correct performance was defined as a trace that does not go outside of the 2 cm-wide track). Trial order was counterbalanced across participants, with half of the participants beginning with the left-lead condition, and half beginning with the right-lead condition.

Trial orders: **LLLL RRRR LLLL RRRR** or **RRRR LLLL RRRR LLLL**

Following the 16 trials performed as described above, participants performed four additional trials with each track orientation, in which visual feedback was eliminated halfway through the track. This was accomplished simply by taping a rectangular piece of paper over the second half of the track, with placement guided by two small red marks placed at the center of the two lines that comprised the track. Participants were told to keep turning the cranks in the same manner in which they were turning them before the dot disappeared. These trials ended when the dot crossed the plane defined by the ends of the two lines that form the track - i.e. the "finish line" was no longer be limited to the inside of the track. A time limit of 30 seconds was imposed in case individual participants got "lost" in the no-visual-feedback area and failed to cross the finish line within that time frame. However, this was not employed in the present experiment: all participants completed all the trials within the 30 second limit. The no-visual-feedback trials were performed in one of the two following orders, counterbalanced across participants:

LLLLRRRR or **RRRRLLLL**.

All participants performed two practice trials with each track orientation before the test trials were administered.

Three dependent measures were used. There were two different measures of overall accuracy: 1) the total area between the trace and the optimal straight line through the centre of the track ("Integrated Error"), and 2) "Percent Excess Output," the ratio of the actual output from each crank over the minimal output that the task required, expressed as a percentage (larger values reflecting poorer performance). The third measure was the time taken from the moment the cursor entered the track to the moment the trial ended.

Data were collapsed across block and trial. Thus, the only within group independent variable was Lead Hand (Right or Left). Fluency Group was the only between group independent variable.

Tasks 3 and 4: Divided Visual Field Tasks

Apparatus

The divided visual field tasks were both performed using the same Macintosh SE/30 computer and external monitor as described for the Sequence Reproduction Task. All stimuli were presented on a white background on the Magnavox monitor. Responses were performed on a set of four keys mounted on a 45 cm x 22 cm piece of wood. Two buttons, one green and one red, were placed side by side, 6 cm apart, centered along the length of the piece of wood and 5 cm from the top edge. The colour of these buttons was switchable so that the either colour could be on either side. A white button (with a "<" painted on it) was placed 1 cm from the top edge, centered along the length. A long, narrow key (called the "spacebar," width = 3 cm, length = 10 cm) was placed 5 cm from the bottom edge, also centered along the length of the piece of wood. This arrangement of keys allowed the participants to comfortably rest both hands on the apparatus while placing their two index fingers on the green and red keys and both thumbs on the black key. Each key was interfaced with a Macintosh computer by means of direct wiring to the switches for four keys on a computer keyboard. Responses were thus recorded by the computer as computer keystrokes.

A computer program presented the stimuli, and timed and recorded the responses. The computer was able to time responses accurately within 17 ms (one 60th of a second).

There were two different stimulus sets, one for the Lexical Decision Task, and one for the Dot Enumeration Task. The lexical decision stimuli consisted of 40 three-letter

words and 40 pronounceable three-letter non-words. The words were selected from those used by Mondor and Bryden (1992a), and were varied according to frequency and imagery value, with equal numbers of high and low frequency and high and low imagery words (Coltheart, 1981). The stimulus set of words and non-words is presented in Appendix I. The precue for the Lexical Decision Task was a small blue dot (diameter = 3 mm) presented directly above the center letter of the word. The Dot Enumeration stimuli consisted of 160 different arrays of three to six dots (diameter = 2 mm) presented inside a 2 cm x 2 cm line frame, with the frame serving as the precue. No dot was presented less than 2 mm from the frame or from another dot. Aside from this constraint, the dots were located randomly anywhere inside the frame by the computer program. For both the Dot Enumeration and Lexical Decision Tasks, the entire set of stimuli was presented once in each visual half-field. For the Dot Enumeration Task, the stimulus set for each visual field was an exact mirror image of the stimulus set in the other visual field.

Participants viewed the monitor such that their eyes were situated approximately 50 cm from the monitor.

Procedure

The procedure for the divided visual field tasks was based upon the procedure described by Mondor and Bryden (1992a). For each of the two different divided visual field tasks, the participants were asked to categorize the stimuli into one of two different categories. For the Lexical Decision Task, the task was to identify whether the stimulus was a word or a non-word. For the Dot Enumeration Task, the task was to determine if the number of dots inside the frame was even or odd. In both cases, a forced choice response mechanism was used. The participants were asked to perform the task as quickly and as accurately as possible, but not to sacrifice accuracy for the sake of speed.

The participants were asked to rest their hands on the response apparatus with their two index fingers on the two response keys (the red key and the green key) and both thumbs on the spacebar. Each trial began with the participant pressing the spacebar with the two thumbs simultaneously. This action triggered a fixation sequence based upon one reported by Efron, Yund and Nichols (1987). One second after pressing the spacebar, a small minus sign appeared in the center of the monitor. After a random interval of between 1 and 2 seconds, the minus sign changed to an equal sign. The participants were required to press the spacebar with both fingers again within 500 ms of the appearance of the equal sign. If the response to the equal sign occurred within the 500 ms limit, the precue (either a dot or the frame) was presented. The target stimulus was presented either simultaneously with the precue ($SOA = 0$ ms) or just following (by 50 ms or 67 ms) the precue. The target stimulus was presented for 100 ms for the Lexical Decision Task and for 117 ms for the Dot Enumeration Task, and was centered at 4° of visual angle (3.1 cm) either to the left or to the right of the equal sign. The maximum time available for the participants to move their eyes (100 ms + 67 ms = 167 ms for the Lexical Decision Task, or 117 ms + 50 ms = 167 ms for the Dot Enumeration Task) was chosen to be less than that required to initiate an eye movement (Boer and Weijger, 1988). The participants were asked to press the green key if the stimulus was either a word (Lexical Decision Task) or an even number of dots (Dot Enumeration Task). They were asked to press the red key if the stimulus was not a word (Lexical Decision Task) or if the number of dots was not even (Dot Enumeration Task). The mapping of response key (red or green) to side (right or left) was counterbalanced across participants.

Following the participant's response, a prompt appeared, asking the participant to press the spacebar to initiate the next trial. If on any trial the participant pressed the spacebar in response to the equal sign either too quickly (either before it appeared, or 167

ms or less after it appeared) or too slowly (more than 500 ms after it appears) a dialog box appeared, asking the participant to press the spacebar to restart the trial.

Each of the 80 different stimuli were presented once to each visual field, for a total of 160 trials for each of the two different divided visual field tasks. Order of presentation of the stimuli was randomized for each task, with the constraint that the test stimuli were never presented to the same visual field in more than four consecutive trials.

Participants performed 32 practice trials with the Lexical Decision Task, and 16 practice trials with the Dot Enumeration Task (fewer than for the Lexical Decision Task because of the similarity between the two tasks, and the fact that the Lexical Decision Task was always performed first).

The dependent variables for the divided visual field tasks were (1) d' (accuracy) and (2) response latency (speed). Percent Correct was also recorded. The within-group independent variables were Visual Field (Left or Right) and SOA (0 or 67 ms). Fluency Group was the only between-group factor.

RESULTS

Overview of Analyses

Analyses for the four tasks will be reported separately below. Since the analyses most directly germane to the hypotheses being tested are the planned comparisons, the outcome of those tests will be discussed first, in the context of a general overview of the data. Following the planned comparisons, a set of more general multivariate (and, when appropriate, univariate) analyses of variance will explore the relationships between the grouping variable and the within-subject independent variables of secondary interest.

In conducting the MANOVAs and ANOVAs, design complexity and the number of statistical tests being conducted were sources of concern. Because of the potentially large number of dependent and independent variables, there was concern about the experimentwise error rate and the likelihood of obtaining complex higher-order effects of little or no interest to the conceptual issues being explored. To address the issue of experimentwise error, univariate tests have been reported only when the corresponding multivariate test was statistically significant. In addition, the MANOVAs reported here will include only independent variables of specific relevance to the main conceptual issues. In some cases data have been collapsed across conditions such as trials or blocks which, while necessary methodologically, are of limited conceptual interest.

Similarly, sex has not been included as a between subject independent variable in the analyses reported here. Both sexes were included in the sample because it was felt that doing so would broaden the availability of participants while at the same time allowing for a more representative sample of people who have stuttered. The proportion of males and females in the sample roughly corresponds to their representation (approximately 2 males :

1 female) in the population of adults who have stuttered at some point in their life (Ambrose, Yairi and Cox, 1993; Andrews *et al.*, 1983).

The decision to pool the data of males and females was based on a number of considerations. First, as mentioned above, one of the primary reasons for including both sexes in the sample was to broaden availability of participants and allow for a larger number of individuals ($N = 24$) in each of the fluency groups. That purpose would have been largely defeated by splitting each fluency group into two Fluency x Sex groups with $N = 16$ males and $N = 8$ females. Second, there were the same concerns mentioned above regarding design complexity and the number of statistical tests being conducted. Finally, past neuropsychological research with adults who stutter has revealed few instances where sex has interacted with fluency group. For example, Webster, (1988) included Sex as a factor in his analyses of bimanual handwriting and obtained no interactions between Group and Sex. Similarly, Blood and Blood (1989) found no differences between male and female stutterers on a dichotic listening task. A preliminary set of MANOVAs with the present data revealed no interactions that included both Group and Sex.

Sequence Reproduction Task

The Sequence Reproduction Task required participants to initiate tapping a novel sequence with their four fingers as quickly and accurately as possible following a tone. The response characteristics that are of interest are those that relate to the organization and initiation of the sequential response. Thus, the dependent measures for the Sequence Reproduction Task were Response Initiation Time (RIT), Sequence Execution Time (SET) and the probability of a Correct First Sequence (CFS). Response initiation time was defined as the time between the onset of the tone and the onset of the first tap. Sequence Execution Time was defined as the time from the onset of the first tap to the onset of the

fourth tap, regardless of sequence length. The latter two measures are represented on the typical event recording illustrated below.

The analyses of the sequence reproduction data contained two independent variables: Group (nonstutterers, stutterers, exstutterers), and Sequence Length (3, 4, or 5 elements). The scores for RIT and SET used in the analyses were the average of the values for the 12 trials performed at each sequence length. The CFS variable was computed as the proportion of the 12 trials at each sequence length in which the entire first sequence was tapped correctly.

Planned comparisons. Planned comparisons were based upon the *apriori* expectation that nonstutterers and exstutterers would constitute one homogeneous population, and would be distinct from (superior to) stutterers in performance on the Sequence Reproduction Task. Thus, two orthogonal contrasts were performed for each measure: the first contrasted stutterers with nonstutterers and exstutterers combined (predicted to be statistically significant) and the second contrasted nonstutterers with exstutterers (predicted to be nonsignificant).

Table 4 shows the mean RITs, SETs and CFS for the three groups collapsed across sequence length. As predicted, stutterers' RITs ($M = 834$ ms, $SD = 214$ ms) were slower than nonstutterers and exstutterers combined ($M = 726$ ms, $SD = 173$ ms) ($t(69) = 2.31$, $p < .02$). Also as predicted, nonstutterers ($M = 689$ ms, $SD = 175$ ms) did not differ

Table 4

Group Means (and *SD*) for the Three Dependent Measures Used in the Analysis of Sequence Reproduction Performance.

	Nonstutterers	Stutterers	Exstutterers
Response Initiation Time (ms)	689 (175)	834 (214)	764 (166)
Sequence Execution Time (ms)	753 (143)	879 (199)	726 (218)
First sequence accuracy (prob. correct)	.86 (.10)	.81 (.10)	.83 (.11)

significantly from exstutterers ($M = 764$ ms, $SD = 166$ ms), though the nonstutterers did tend to respond more quickly ($t(69) = 1.41$, $p = .16$).

Stutterers also had slower overall SETs ($M = 879$ ms, $SD = 199$ ms) than nonstutterers and exstutterers combined ($M = 739$ ms, $SD = 184$ ms) ($t(69) = 2.96$, $p < .005$), and nonstutterers ($M = 753$ ms, $SD = 143$ ms) did not differ from exstutterers ($M = 726$ ms, $SD = 218$ ms): $t(69) = .493$.

Finally, stutterers tended to make more errors on the first sequence (mean probability of a Correct First Sequence = .81, $SD = .10$) than the other two groups combined ($M = .85$, $SD = .11$), but this pattern failed to reach statistical significance ($t(69) = 1.28$, $p = .10$). Nonstutterers ($M = .86$, $SD = .10$) did not differ from stutterers ($M = .83$, $SD = .11$): $t(69) = .995$.

It should be noted, however, that the exstutterers RITs were in fact more similar to those of the stutterers (difference in RITs = 70 ms) than the nonstutterers (difference = 75 ms). It is thus possible that the observed group differences in RIT could be better explained by an alternative set of hypotheses, where exstutterers were grouped with

stutterers, or where exstutterers fell between nonstutterers and exstutterers. With this concern in mind, two *multivariate* contrasts, with all three dependent measures included, were performed. The first was based on the same *a priori* hypotheses used for the univariate contrasts reported above, and compared stutterers with exstutterers and nonstutterers combined. The second was a similar multivariate contrast, based on an alternative model that groups the exstutterers with the nonstutterers: [NS vs. (ST + ES)]. The contrast based on the *a priori* hypotheses was, as would have been predicted, statistically significant ($F(3, 67) = 3.03, p < .02$). The contrast based on the alternative model was not statistically significant ($F(3, 67) = 2.22, p = .093$). Two points regarding the second contrast should be made. First, a trend toward significance would be expected based on the *a priori* model, since half of the combined group was composed of stutterers, expected to be different from the nonstutterers. Second, the p value reported was unadjusted, in spite of the fact that the test was based on a post hoc model generated from a visual inspection of the data. This will also favour a trend toward statistical significance on this test. With these two factors in mind, one can with some confidence accept the position that, when all three measures are considered together, the *a priori* model is more consistent with the observed data than is the alternative model that exstutterers and stutterers are similar with respect to their performance on the Sequence Reproduction Task. However, on the basis of these data alone, the possibility that the group of exstutterers contained some misclassified stutterers cannot be ruled out completely.

MANOVA. Figures 3, 4 and 5 display the mean values of RIT, SET and CFS for the three groups as a function of Sequence Length. A two way (Group x Sequence Length) MANOVA with the three dependent measures indicated that there were main effects for Group ($F(6,134) = 2.43, p < .05$) and Sequence Length ($F(6,272) = 52.28, p < .0001$). Consistent with the planned contrasts reported above, univariate ANOVAs

Figure 3. Mean (and standard error) Response Initiation Time (RIT) in ms as a function of Group and Sequence Length in the Sequence Reproduction Task.

Figure 4. Mean (and standard error) Sequence Execution Time (SET) in ms as a function of Group and Sequence Length in the Sequence Reproduction Task.

Figure 5. Mean probability of a Correct First Sequence (and standard error) as a function of Group and Sequence Length in the Sequence Reproduction Task.

indicated that there were Group main effects for both RIT ($F(2,69) = 3.66, p < .05$) and SET ($F(2,69) = 4.49, p < .02$).

The Sequence Length main effect was evident in all three dependent measures. RIT ($F(2,138) = 33.63, p < .0001$), SET ($F(2,138) = 121.95, p < .0001$) and CFS ($F(2,138) = 85, p < .0001$). As illustrated in Figure 3, the expectation that Response Initiation Time would increase with sequence length was not supported: RITs actually decreased from sequences of length 3 to length 4 in all three groups. A post hoc comparison indicated that the decrease in RIT from length 3 to length 4 was statistically significant ($t(69) = 3.96, p < .0001$). In fact, there was a statistically significant quadratic trend in the RITs as a function of Sequence Length ($F(1,69) = 65.8, p < .0001$).

As illustrated in Figure 4, SET increased with Sequence Length. There was a linear trend ($F(1,69) = 200.58, p < .0001$) as well as a significant quadratic component ($F(1,69) = 23.85, p < .0001$), the latter reflecting a disproportionate increase in SET from length 4 to length 5. Similarly, there were significant linear ($F(1,69) = 112.15, p < .0001$) and quadratic ($F(1,69) = 34.36, p < .0001$) components to the CFS Sequence Length main effect (see Figure 5). Again, performance deteriorated as sequence length increased, with a disproportionate decrease from sequences of length 4 to sequences of length 5.

Summary: Consistent with the hypotheses: (1) stutterers' performance on the Sequence Reproduction Task was impaired relative to that of nonstutterers and exstutterers combined, regardless of sequence length, and (2) exstutterers did not differ reliably from nonstutterers.

Bimanual Crank-Turning

In the Bimanual Crank-turning Task, participants turned two cranks simultaneously, with their left and right hands, in order to manipulate a cursor through an

angled track on a computer monitor. Three measures of performance were obtained. Of the three measures, two were related to accuracy and one to the speed of performance.

The first accuracy measure, "Integrated Error" (IErr), was a measure of the accuracy of the trace produced by the participant; specifically, it was obtained by integrating to compute the area between the path followed by the participant's trace and the optimal path through the center of the track. The Integrated Error measure is illustrated in Figure 6.

The second accuracy measure, Percent Excess Output (PEO), reflects the amount of motor output generated by the two hands as manifested by the sum of all crank movements, regardless of direction. The measure was generated as follows. Crank turning was initially recorded by the computer as a series of x, y, and time coordinates which represented the position of the cursor across time. Coordinates were sampled every 16 ms provided cursor movement was detected. For each successive set of coordinates, the absolute change in position of the right and left cranks could be determined by calculating the absolute value of the difference between the successive values of x and y, respectively ($|x_{i+1} - x_i|$ and $|y_{i+1} - y_i|$). Summing these values over the entire series of coordinates gives values for the total amount of motor output of each hand (represented as centimetres of displacement of the cursor). The minimum required output for each hand (6.7 cm for the non-lead hand and 13.4 cm for the lead hand as determined by the angle of the track) was subtracted from the actual amount, and the difference ("Excess Output") was expressed as a percentage of the minimum required.

$$\text{PEO} = \frac{(\text{actual crank displacement} - \text{minimal crank displacement})}{\text{minimal crank displacement}} \times 100$$

Figure 6. Illustration of (A) a typical trace through the track and (B) a representation of the integrated area measure applied to the same trace.

For the purposes of the analyses reported here, Percent Excess Output was collapsed across the two hands to give a single value. For both dependent measures and the covariate, the raw values used in the analyses reported here were the average of the 8 trials in each Lead Hand condition for with-visual-feedback crank-turning or the 4 trials in each Lead Hand condition for no-visual-feedback crank-turning.

The third measure obtained was the natural logarithm of the time to complete the trace ($\ln(\text{Time})$). The time scores were log-transformed because the distribution of raw scores was positively skewed, and there were consequently a large number of outlier scores.

To minimize the number of dependent variables and to control for the possibility of differential speed-accuracy tradeoffs, $\ln(\text{Time})$ was treated as a "varying" covariate (i.e. there was a separate covariate for each level of each within-subject factor) in the planned comparisons and analyses of variance on the accuracy measures. The covariate was left in the analysis if the covariate was statistically significant for any of the main effects or interactions.

The main independent variable for the crank-turning task was Lead Hand. Lead Hand refers to the hand which was required to turn twice as fast as the other (non-lead) hand. Lead Hand corresponds directly to the angle of the track: the 63.5° track angle required twice as much output from the left hand as was required from the right hand, and thus represents the Left Lead condition. The 26.5° track angle required more output from the right hand and thus corresponds to the Right Lead condition. Stated differently, the track was presented with a slope $m = 2$ in the Left Lead condition, and a slope $m = 0.5$ in the Right Lead condition.

Planned comparisons. The hypotheses and corresponding planned comparisons for the Bimanual Crank-turning Task were the same as those described for the Sequence Reproduction Task. The first comparison contrasted stutterers with nonstutterers and

exstutterers combined (predicted to be statistically significant), and the second contrasted the nonstutterers with the exstutterers (predicted to be nonsignificant).

The group means for Integrated Error and Percent Excess Output for bimanual crank-turning when visual feedback was available are presented in Table 5. As predicted, stutterers generated less accurate traces (Integrated Error, $M = 4.29 \text{ cm}^2$, $SD = 0.88$) than nonstutterers and exstutterers combined ($M = 3.90 \text{ cm}^2$, $SD = 1.24$), though this effect was not statistically significant ($t(69) = 1.16$, $p = .125$). Also as predicted, exstutterers ($M = 3.78 \text{ cm}^2$, $SD = 1.13$) did not differ significantly from nonstutterers ($M = 4.02 \text{ cm}^2$, $SD = 1.36$): $t(69) = .89$.

Table 5

Group Means (and SD) for the Three Performance Measures Obtained in the Bimanual Crank Turning Task (With Visual Feedback).

	Nonstutterers	Stutterers	Exstutterers
Integrated Error (cm^2)	4.02 (1.36)	4.29 (0.88)	3.78 (1.13)
Percent Excess Output	2.79 (1.07)	4.24 (1.86)	3.29 (1.30)
Time (s)	11.1 (6.2)	9.5 (3.9)	10.2 (4.8)

Stutterers did, as predicted, generate significantly more excess output (Percent Excess Output, $M = 4.24\%$, $SD = 1.86$) than nonstutterers and exstutterers combined ($M = 3.04\%$, $SD = 1.21$): $t(68) = 2.81$, $p < .005$. Also as predicted, nonstutterers did not differ from exstutterers in terms of Percent Excess Output ($t(68) = .83$).

When visual feedback was not available (see Table 6), stutterers were both less accurate overall ($M = 5.09 \text{ cm}^2$, $SD = 1.89$) and generated more excess output ($M = 17.3\%$, $SD = 8.79$) than nonstutterers and exstutterers combined ($M = 3.64 \text{ cm}^2$, $SD = 1.52$ and $M = 12.42\%$, $SD = 4.52\%$ for Integrated Error and Percent Excess Output, respectively). Both of these differences were statistically significant; $t(69) = 3.56$, $p < .0005$ and $t(68) = 2.43$, $p < .01$, for Integrated Error and Percent excess output, respectively. As predicted, nonstutterers did not differ statistically from exstutterers on either measure ($t(69) = .19$ and $t(68) = 1.14$ for IErr and PEO, respectively).

Table 6

Group Means (and SD) for the Three Performance Measures Obtained in the Bimanual Crank Turning Task (No Visual Feedback).

	Nonstutterers	Stutterers	Exstutterers
Integrated Error (cm^2)	3.60 (1.43)	5.09 (1.89)	3.69 (1.64)
Percent Excess Output	11.1 (5.7)	17.3 (8.8)	13.7 (8.9)
Time (s)	6.17 (3.7)	4.82 (1.9)	7.11 (4.8)

Note. Data presented are from second half (NVF portion) of track only. To achieve a common unit for comparison with Table 5, the area and time measures presented here should be doubled.

To summarize, with the exception of the Integrated Error dependent measure when visual feedback was available, the results of the planned comparisons for the Bimanual

Crank-turning Task were consistent with the *apriori* hypotheses in all cases: the performance of the stutterers was impaired relative to the other two groups, and the exstutterers did not differ significantly from the nonstutterers.

MANCOVAs. In an effort to explore the role of the Lead Hand and Visual Feedback on performance in the three groups, three MANCOVAs were conducted. The first two MANCOVAs included Group as a between-subjects factor, and Lead Hand as a within-subjects factor. The dependent measures were the same as those discussed above in the context of the planned comparisons: Integrated Error and Percent Excess Output.

For a number of reasons, the two Visual Feedback conditions were treated as separate analyses, rather than as different levels of a Feedback factor in a single analysis. First, in the With-visual-feedback (WVF) trials, participants were required to stay within the track, while this requirement could not be maintained in the No-visual-feedback (NVF) condition. Second, for reasons discussed in the Methods section, there were twice as many trials (16) of WVF crank-turning than there were of NVF crank-turning (8 trials). Third, it was necessary in the NVF condition to compute the dependent measures based only on the second half (NVF portion) of the track, rather than the entire track. Finally, there was a great deal more variability in the scores under NVF conditions than there was under WVF conditions. While this does not constitute a violation of the assumptions underlying a within-subjects design with only two levels of the repeated measures factor, it does affect the outcome of the tests for certain effects (this will be discussed further below).

Nevertheless, a third MANCOVA was conducted which did include Visual Feedback as a within-subjects factor. This analysis, which was interpreted with the above-mentioned concerns in mind, was important because the role of Visual Feedback on performance was expected to differentiate the groups; i.e. a Group x Feedback interaction

was expected. The most serious of the concerns mentioned above should not preclude the interpretation of the Group x Feedback interaction.

1. MANCOVA: Crank-turning with Visual Feedback. Integrated Error and Percent Excess Output as a function of Group and Lead Hand for the With-visual-feedback trials are presented in Figures 7 and 8. There was a significant main effect for Group ($F(4,134) = 3.93, p < .01$). As discussed in the context of the planned comparisons, stutterers generated more excess output than the nonstutterers and exstutterers ($F(2,68) = 5.97, p < .005$), though the groups did not differ significantly in terms of overall accuracy (Integrated Error, $F(2,68) = 1.08$). The MANCOVA also indicated that there was a significant Lead Hand main effect, apparently attributable to the Integrated Error measure: all three groups performed more accurately in the Left Lead condition (Integrated Error for Left Lead, $M = 3.62 \text{ cm}^2, SD = 1.22$, for Right Lead, $M = 4.44 \text{ cm}^2, SD = 1.37$): $F(2,67) = 22.10, p < .0005$.

2. MANCOVA: Crank-turning Without Visual Feedback. Integrated Error and Percent Excess Output as a function of Group and Lead Hand for the NVF trials are presented in Figures 9 and 10. As with WVF crank-turning, there was a statistically significant main effect for Group ($F(4,134) = 3.48, p < .01$). In contrast to the WVF crank-turning analysis, however, there were statistically significant univariate Group main effects for both the Integrated Error ($F(2,68) = 5.47, p < .01$) and Percent Excess Output ($F(2,68) = 3.76, p < .05$) measures. Stutterers generated less accurate traces and more excess output than the other two groups.

3. MANCOVA: Feedback included as a factor. In the third MANCOVA, which included Visual Feedback (WVF, NVF) as a factor, the Integrated Error and time scores in the NVF condition were doubled to allow for comparison with the WVF scores (which, as

Figure 7. Mean Integrated Error area (and standard error) as a function of Group and Lead Hand in the With-visual-feedback condition of the Bimanual Crank-turning Task.

Figure 8. Mean Percent Excess Output (and standard error) as a function of Group and Lead Hand in the With-visual-feedback condition of the Bimanual Crank-turning Task.

Figure 9. Mean Integrated Error area (and standard error) as a function of Group and Lead Hand in the No-visual-feedback condition of the Bimanual Crank-turning Task.

Figure 10. Mean Percent Excess Output (and standard error) as a function of Group and Lead Hand in the No-visual-feedback condition of the Bimanual Crank-turning Task.

mentioned above, were computed over the entire 15 cm length of track rather than only the second half of the track).

The MANCOVA revealed three significant effects. In accordance with the analyses mentioned above, there was a statistically significant main effect for Group ($F(4, 134) = 3.55, p < .01$). A Visual Feedback main effect was evident ($F(4,67) = 76.29, p < .0005$), reflecting a marked deterioration in performance in all three groups when visual feedback was unavailable ($F(1,68) = 138.83, p < .0005$ and $F(1,68) = 146.03, p < .0005$, for Integrated Error and Percent Excess Output, respectively). Note that these main effects are confounded by fact that the requirement to keep one's trace within the track was not maintained in the NVF trials.

The same MANCOVA also indicated that there was a statistically significant interaction between Group and Visual Feedback ($F(2, 68) = 3.47, p < .01$). A similar Group x Feedback interaction was evident in the univariate Integrated Error ANCOVA ($F(2,68) = 5.24, p < .01$) and reflected the fact that the group difference in accuracy was only evident when visual feedback was unavailable.

One seemingly anomalous outcome of the analyses that included Visual Feedback as a factor relates to the Lead Hand effect that was observed in the WVF MANOVA. This strong effect ($p < .0005$) was not evident at all in the NVF MANOVA ($p = .872$) (and the Integrated Error ANOVA). One would thus have expected to find a significant Lead Hand x Feedback interaction in the analysis that included Visual Feedback as a factor. The fact that this was not observed ($F(2,67) = 1.33$) appears to be related to the substantially greater variability in the NVF data (overall $SD = 3.56$ cm) than in the WVF data (overall $SD = 1.14$ cm). In performing the within-subjects Lead Hand and Group by Lead Hand tests in the MANOVA, (Right Lead - Left Lead) difference scores are computed. The variability of those difference scores is predominantly influenced by the greater variability of the NVF condition, effectively masking the Lead Hand effect that was evident in the WVF condition.

Summary: Consistent with the hypotheses, stutterers were impaired relative to nonstutterers and exstutterers on the crank-turning task overall. While stutterers' traces were no less accurate on the task than the other two groups when visual feedback was available, they were 40% less accurate when visual feedback was not available. Furthermore, under both Visual Feedback conditions, they generated 40% more excess output than the other two groups combined.

Lexical Decision Task

In the Lexical Decision Task, participants were required to make a forced-choice word/non-word discrimination on stimuli presented tachistoscopically to one or the other visual field. A dot precue was presented above the target stimulus location either simultaneously with or 67 ms prior to presentation of the target stimulus. Thus, the independent variables with their associated levels were Visual Field (Left Visual Field = LVF, Right Visual Field = RVF), Stimulus Onset Asynchrony (SOA = 0 ms, SOA = 67 ms) and Group (nonstutterer, stutterer, exstutterer).

Two performance measures were obtained for the Lexical Decision Task. Accuracy was assessed by d' , and speed was assessed by response time (RT).

d' is a measure of accuracy derived from signal detection theory. It is calculated according to the expression:

$$d' = Z(\text{FA}) - Z(\text{HIT})$$

where HIT is the hit rate (proportion of "Word" responses when the target stimulus was in fact a word) and FA is the false alarm rate (proportion of "Word" responses when the target stimulus was in fact a nonword). The Z-score corresponding to a given proportion is taken from a standard function, represented in tabular form. The d' values for each participant

under each Visual Field x SOA condition were computed automatically by the stimulus presentation/data acquisition software.

d' a better measure of overall accuracy than Percent Correct because it accounts for the relative rates of hits and false alarms. Nevertheless, in order to provide a more intuitively interpretable measure of accuracy, Percent Correct scores are presented in the tables together with d' values.

The Percent Correct and Response Time scores reported here were averaged over the 40 trials conducted at each Visual Field x SOA combination (20 word, 20 non-word).

The instructions to the participants stressed that accuracy should be given priority over speed in responding. Participants were instructed to respond as quickly as possible without sacrificing accuracy. In the analyses to follow, d' was the only dependent measure used. Response time was treated as a varying covariate (i.e. there were separate covariates for each level of each within-subject factor). As with the crank-turning task analyses, covariates were left in the analysis if the covariate was significant for any of the main effects or interactions.

Planned Comparisons. Two orthogonal planned comparisons were conducted. The comparisons were based on the hypothesis that stutterers and exstutterers would form a homogeneous population with respect to their visual field performance asymmetry on the task, and that the nonstutterers would have a stronger right visual field advantage than the other two groups. Thus, planned comparisons were conducted on the (RVF - LVF) difference scores rather than on raw scores. The first comparison contrasted nonstutterers with stutterers and exstutterers combined (predicted to be statistically significant), and the second contrasted the stutterers with the exstutterers (predicted to be nonsignificant).

The mean values for Percent Correct, d' and RT for the three groups are presented in Table 7 as a function of Visual Field. Mean (Right - Left) difference scores for d' , Percent Correct and RT are presented in Table 8. As evident in Tables 7 and 8, all three

Table 7

Group Means (and *SD*) as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Lexical Decision Task Collapsed Across Stimulus Onset Asynchrony.

	Nonstutterers		Stutterers		Exstutterers	
	Left	Right	Left	Right	Left	Right
Percent Correct	73.1 (1.86)	79.1 (1.64)	73.3 (2.33)	74.6 (1.58)	75.9 (1.91)	78.6 (1.47)
<i>d'</i>	1.35 (.58)	1.85 (.70)	1.44 (.78)	1.55 (.62)	1.68 (.83)	1.82 (.67)
Response Time (ms)	942 (197)	917 (180)	1209 (377)	1190 (339)	1031 (266)	985 (215)

groups performed more accurately when the stimuli were presented to the right visual field (for Percent Correct collapsed across Group, $M = 77.4\%$, $SD = 8.0\%$ for RVF, $M = 74.1\%$, $SD = 10.2$ for LVF). Nonstutterers did, as predicted, have a significantly larger right visual field advantage (% correct difference scores, $M = 5.9\%$, $SD = 7.2$) than stutterers and exstutterers combined ($M = 2.0\%$, $SD = 8.2$): $t(69) = 2.66$, $p < .005$. Also as predicted, stutterers did not differ significantly from exstutterers: $t(69) = .19$.

ANCOVA. A three-way (Group $\times$ SOA $\times$ Visual Field) analysis of covariance, with Response Time as the covariate, indicated that there were main effects for Visual Field ($F(1,68) = 6.74$, $p < .05$) and SOA ($F(1,68) = 6.19$, $p < .05$), and an interaction between Group and Visual Field ($F(1,68) = 3.56$, $p < .05$).

The SOA main effect reflects greater accuracy when the SOA was 67 ms (Percent Correct $M = 77.8\%$, $SD = 9.5$ with SOA = 67 ms; $M = 73.7\%$, $SD = 8.5$, with SOA = 0 ms). The Visual Field main effect reflects the greater overall accuracy in the Right Visual

Table 8

Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Lexical Decision Task.

	Nonstutterers	Stutterers	Exstutterers
Percent Correct ^a	5.94 (7.21)	1.30 (9.06)	2.66 (7.43)
d' ^b	.496*** (.565)	.112 (.563)	.137 (.732)
Response Time (ms) ^c	25.7 (111.5)	19.0 (127.6)	46.8 (101.7)

^aTests of significance not computed for these data.

^bTests of significance refer to within-group Visual Field simple effect.

^cIncluded as covariate in comparisons with d' dependent variable.

*** $p < .0005$

Field (noted above), but, as indicated by the significant Group $\times$ Visual Field interaction, the performance asymmetry differed among the three groups.

Figure 11 illustrates the Group by Visual Field interaction. While all three groups displayed greater accuracy in the right visual field, the asymmetry was substantially larger in the nonstutterers than in the other two groups. In fact, analysis of simple effects revealed that the asymmetry was statistically significant only in the nonstutterers ($F(1,68) = 13.65$, $p < .0005$). Neither the stutterers ($F(1,68) = .36$) nor the exstutterers ($F(1,68) = .10$) had a statistically significant Right Visual Field advantage.

interhemispheric relations can be demonstrated with nonverbal target stimuli in both stutterers and exstutterers. This suggests that the anomaly is not specific to the speech/language domain, and may involve more general laterality mechanisms.

(b). Attentional Manipulation

In both the Lexical Decision Task and the Dot Enumeration Task, the target stimuli were presented together with a lateralized attentional precue that always corresponded to the side of the target stimulus. The precue was presented either just before ($SOA = 67$ ms for Lexical Decision Task, $SOA = 50$ ms for Dot Enumeration Task) or simultaneous with ($SOA = 0$ ms) the target stimulus. Mondor and Bryden have shown, using a lexical decision divided visual field task (Mondor and Bryden, 1992a) and various dichotic listening tasks (Mondor and Bryden, 1991; 1992b; 1992c; Mondor, 1993), that the size of the perceptual asymmetry can be reduced or eliminated by preceding the target stimulus with an attentional precue such as the dot and box used here, and that this effect is dependent on the magnitude of the stimulus onset asynchrony.

The present results did not replicate those of Mondor and Bryden (1992a). The effect should have manifested itself, at least within the nonstutterers, as a Visual Field $\times$ SOA interaction, with a larger Visual Field asymmetry under $SOA = 0$ ms than under $SOA = 67$ ms (Lexical Decision) or $SOA = 50$ ms (Dot Enumeration). This was not observed in either task. The reason for this failure to replicate the findings of Mondor and Bryden (1992a) is unclear. It should be noted that in the present experiment, only one value of SOA was used, whereas Mondor and Bryden (1992a) used a range of different SOA values and examined the more general relationship between SOA and visual field asymmetry. They reported an increase in the degree to which the asymmetry was diminished with increasing SOA. With that in mind, the largest possible SOA was chosen for the present study. The magnitude of the SOA is limited by the fact that overall SOA + target stimulus

The expected SOA x Visual Field and Group x SOA x Visual Field interactions were not observed. In fact, nonstutterers actually had a larger mean asymmetry at the SOA of 67 ms (mean d' difference = 0.640) than they did at SOA = 0 ms (difference = 0.351).

For the sake of comparison with previously reported research, the data for individual subjects were coded into the nominal categories of "RVF advantage" and "LVF advantage" according to the visual field performance asymmetry, and a series of chi-square analyses were conducted. These data are presented in Table 9. The first two analyses were analogous to the planned contrasts: nonstutterers were compared to stutterers and exstutterers combined, and stutterers were compared to exstutterers. The first analysis indicated that LVF advantages were more frequent among the stutterers and exstutterers combined (21/48 or 43.75%) than among the nonstutterers (4/24 or 16.67%): $\chi^2(1) = 5.18$, $p < .05$. Stutterers (11/24 or 45.8%) did not differ significantly from exstutterers (10/24 or 41.7%): $\chi^2(1) = .085$.

Table 9

Total Number of Individuals in the Three Groups with Left Visual Field (LVF) and Right Visual Field (RVF) Advantages on the Lexical Decision Task.

	Nonstutterers	Stutterers	Exstutterers
LVF advantage	4	11	10
RVF advantage	20	13	14

The third chi-square analysis included only nonstutterers and stutterers and was included for direct comparison with past research that has reported a higher frequency of anomalous asymmetries in stutterers than nonstutterers. The same finding was obtained in the present data: LVF advantages were more frequent in the stutterers (11/24 or 45.8%) than in the nonstutterers (4/24 or 16.7%): $\chi^2(1) = 4.75, p < .05$.

Summary: Consistent with the main hypothesis, stutterers and exstutterers were similar in having a weaker asymmetry (actually no asymmetry) on the Lexical Decision Task than nonstutterers, who demonstrated the expected right visual field advantage. However, the dot precue manipulation did not affect the direction or size of the asymmetry in any of the three groups.

Dot Enumeration Task

The same performance measures used in the Lexical Decision Task were used in the Dot Enumeration Task: d' (presented together with Percent Correct) to assess accuracy, and response time (RT), once again treated as a covariate. The Percent Correct and Response Time scores reported here were the averages of the 40 trials conducted at each Visual Field x SOA combination (10 each with 3 dots, 4 dots, 5 dots and 6 dots). d' was calculated as described in the Lexical Decision Task section. For the Dot Enumeration Task, a hit was defined as identification of the number of dots as "Even" when there was in fact an even number of dots, and a false alarm was defined as identification of the number of dots as "Even" when that was in fact an odd number of dots.

The independent variables were also similar to those used in the Lexical Decision Task: Visual Field (RVF, LVF) and SOA (0 ms, 50 ms), with the one difference being the magnitude of the SOA in the asynchronous condition (50 ms rather than 67 ms).

It was clear during testing that participants found the Dot Enumeration Task considerably more difficult than the Lexical Decision Task. Several participants completed the task with below-chance accuracy (indeed, some had below 50% accuracy). Data from participants whose accuracy was not significantly better than chance were excluded from the analysis. Based on the binomial distribution with a one-tailed probability level of .05, the criterion for inclusion in the analysis was established at 90 out of 160 trials (56.25%) correct. The data from three nonstutterers, two stutterers and six exstutterers were excluded on the basis of this criterion.

Planned Comparisons. Table 10 shows the mean Percent Correct, d' and response time scores for the three groups as a function of Visual Field. Mean (Right - Left) difference scores collapsed across SOAs for d' , Percent Correct and RT are presented in Table 11. In contrast to the Lexical Decision Data, the only notable performance asymmetry was a weak RVF advantage in the nonstutterers (Percent Correct difference score $M = 2.38\%$, $SD = 7.56$; d' difference score $M = .257$, $SD = .561$). Stutterers and exstutterers had a combined Percent Correct asymmetry of 0.3% ($SD = 8.4\%$) and a combined d' asymmetry of .000 ($SD = .583$); the two groups did not differ significantly from one another in that respect: $t(59) = .15$. The difference between nonstutterers and the combined group of stutterers and exstutterers approached statistical significance: $t(59) = 1.65$, $p = .052$.

However, a right visual field advantage in the nonstutterers was not expected, and it was hypothesized that the stutterers and exstutterers would demonstrate a stronger *left* visual field advantage than the nonstutterers. Thus, while the difference observed was at the hypothesized tail of the distribution (i.e. a more leftward or less rightward asymmetry for the stutterers and exstutterers than for the nonstutterers), it must be noted that the asymmetries in the three groups were shifted to the right relative to what was expected.

Table 10

Group Means as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Dot Enumeration Task Collapsed Across Stimulus Onset Asynchrony.

	Nonstutterers (<i>N</i> = 21)		Stutterers (<i>N</i> = 22)		Exstutterers (<i>N</i> = 18)	
	Left	Right	Left	Right	Left	Right
Percent Correct	66.9 (8.7)	69.3 (7.6)	67.4 (8.4)	67.6 (7.7)	69.2 (9.5)	69.7 (8.3)
<i>d'</i>	1.08 (.67)	1.34 (.62)	1.13 (.59)	1.14 (.58)	1.27 (.71)	1.25 (.60)
Response Time (ms)	1137 (248)	1125 (277)	1386 (436)	1391 (499)	1345 (456)	1306 (353)

Note. Data of 11 participants were excluded due to below-chance performance on the Dot Enumeration Task.

For reasons relating to the poor overall accuracy (to be discussed below), a second set of contrasts was conducted, using only trials with SOA = 50 ms. Mean Percent Correct, *d'* and response time scores with SOA = 50 ms for the three groups are presented in Table 12. The mean (Right - Left) difference scores for Percent Correct, *d'* and response time with SOA = 50 ms are presented in Table 13.

When only trials with SOA = 50 ms were considered, there was a notable increase in accuracy (to be discussed below in the context of the ANCOVA) as compared to the overall data set reported above. In addition, the nonstutterers demonstrated a more substantial right visual field advantage (Percent Correct difference score $M = 6.19\%$, $SD = 9.86$; *d'* difference score $M = 5.06$, $SD = .771$) than was evident when both SOAs were considered. However, as with the overall data set, the performance asymmetry in the

Table 11

Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Dot Enumeration Task.

	Nonstutterers (<i>N</i> = 21)	Stutterers (<i>N</i> = 22)	Exstutterers (<i>N</i> = 18)
Percent Correct ^a	2.38 (7.56)	0.17 (8.08)	0.49 (8.96)
<i>d'</i> ^b	.257* (.561)	.012 (.560)	-.015 (.625)
Response Time (ms) ^c	11 (91)	.5 (154)	39 (171)

Note. Data of 11 participants were excluded due to below-chance performance on the Dot Enumeration Task.

^aTests of significance not computed for these data.

^bTests of significance refer to within-group Visual Field simple effect.

^cIncluded as covariate in comparisons with *d'* dependent variable.

**p* = .052

combined group of stutterers and exstutterers was negligible (Percent Correct difference score $M = -.31\%$, $SD = 9.44$; *d'* difference score $M = .022$, $SD = .734$). The difference between the nonstutterers and the combined group of stutterers and exstutterers was statistically significant ($t(59) = 2.66$, $p < .01$), while the stutterers did not differ from the exstutterers ($t(59) = .83$).

ANOVA (Entire data set): As response time was not a significant covariate, it was excluded and the data were analyzed as a standard analysis of variance. A 3-way (Group x SOA by Visual Field) ANOVA revealed only one main effect, a strong main effect for SOA ($F(1, 58) = 61.04$, $p < .0001$). Overall, participants were much more accurate with the

Table 12

Group Means as a Function of Group and Visual Field for the Three Performance Measures Obtained in the Dot Enumeration Task with SOA = 50 ms.

	Nonstutterers (<i>N</i> = 21)		Stutterers (<i>N</i> = 22)		Exstutterers (<i>N</i> = 18)	
	Left	Right	Left	Right	Left	Right
Percent Correct	69.6 (11.8)	75.8 (10.8)	71.9 (9.1)	72.6 (11.3)	74.3 (13.2)	72.8 (10.1)
<i>d'</i>	1.31 (.94)	1.80 (.86)	1.40 (.71)	1.47 (.92)	1.64 (.99)	1.51 (.78)
Response Time (ms)	1123 (263)	1113 (288)	1361 (408)	1372 (472)	1335 (500)	1308 (391)

Note. Dot enumeration data restricted to trials with SOA = 50 ms due to poor accuracy on trials with SOA = 0 ms. Data of 11 participants were excluded due to below-chance performance on the Dot Enumeration Task.

SOA of 50 ms (Percent Correct $M = 72.8\%$, $SD = 9.8$) than with the SOA of 0 ms ($M = 63.7\%$, $SD = 6.6$). In signal detection terms, participants were nearly twice as sensitive to the "even" signal with the SOA of 50 ms ($d' M = 1.52$, $SD = .77$) than with the SOA of 0 ms ($d' M = 0.88$, $SD = .45$).

In spite of the fact that data from individuals with below-chance accuracy overall were excluded, accuracy on the task, particularly with the SOA of 0 ms, was low. The overall mean of 63.7% correct with SOA = 0 ms was within less than one standard deviation (6.6%) from the cutoff point for above chance performance (for 80 trials with one-tailed $p = .05$, 47 correct or 58.75%). In addition, as illustrated by Figure 12, there was no appreciable asymmetry in any group at the SOA of 0 ms. In light of the above concerns, and since SOA did not interact significantly with the other independent variables,

Table 13

Mean (Right - Left) Visual Field Performance Asymmetries (and SD) for the Three Performance Measures Obtained in the Dot Enumeration Task with SOA = 50 ms.

	Nonstutterers (<i>N</i> = 21)	Stutterers (<i>N</i> = 22)	Exstutterers (<i>N</i> = 18)
Percent Correct ^a	6.19 (9.86)	0.68 (10.01)	-1.53 (8.83)
<i>d'</i> ^b	.506*** (.771)	.067 (.771)	-.130 (.692)
Response Time (ms) ^c	-10 (116)	11 (189)	-27 (220)

Note. Data of 11 participants were excluded due to below-chance performance on the Dot Enumeration Task.

^aTests of significance not computed for these data.

^bTests of significance refer to within-group Visual Field simple effect.

^cIncluded as covariate in comparisons with *d'* dependent variable.

****p* < .005

it seemed appropriate to exclude the trials with SOA = 0 ms and reanalyze the data without the SOA independent variable.

For the sake of consistency, the analyses reported below were conducted on data from the same subset of participants described above (those with at least 90 correct out of the entire set of 160 trials). However, the results of the planned comparisons (discussed above) and the ANOVA were essentially the same when the exclusionary criterion was

Figure 12. Mean (and standard error) visual field asymmetry in accuracy (d') as a function of Group and Stimulus Onset Asynchrony (SOA) in Dot Enumeration Task. LVF = Left Visual Field, RVF = Right Visual Field.

based solely on the 80 trials with SOA = 50 ms (47/80 trials correct, which would have resulted in the exclusion of 8 nonstutterers, 9 stutterers and 10 exstutterers).

ANCOVA (SOA = 50 ms only): The second analysis, a 2-way Group by Visual Field ANOVA, revealed one statistically significant effect, an interaction between Group and Visual Field ($F(2,58) = 3.77, p < .05$). To reiterate, this Group x Visual Field effect was also the only statistically significant effect when the more stringent exclusionary criterion, based on the 80 SOA = 50 ms trials, was used ($F(2,42) = 3.99, p < .05$).

Analysis of simple effects indicated that there was a statistically significant RVF advantage in nonstutterers (Percent Correct in RVF, $M = 75.8\%$, $SD = 10.8$, in LVF, $M = 69.6\%$, $SD = 11.8$: $F(1,58) = 9.60, p < .005$), and no visual field asymmetry in either the stutterers ($F(1, 58) = 0.18$) or the exstutterers ($F(1,58) = .54$).

Summary: Because of the poor overall accuracy on the Dot Enumeration Task (particularly in the SOA = 0 ms condition), the results of the Dot Enumeration Task should be interpreted with caution. When the entire data set is considered, accuracy was poor and there were no statistically significant Group-related effects. However, when the analyses were limited to trials with SOA = 50 ms, the results were similar to those of the Lexical Decision Task. Nonstutterers had a right visual field advantage on the task, while the other two groups had no reliable asymmetry in either direction. These results are consistent the general predictions that nonstutterers would have a more leftward (or less rightward) asymmetry, and that stutterers and exstutterers would perform similarly on the task. However, contrary to the expectation, a left visual field advantage was not obtained in any of the three groups. This is an additional reason that caution must be used in the interpretation of the results of the Dot Enumeration Task.

Summary of Results

The outcome of all the planned comparisons for all four tasks are summarized in Tables 14 (motor tasks) and 15 (divided visual field tasks). As is evident from those tables, the predictions generated by the hypotheses were supported in most cases.

In general, the performance of the exstutterers on the motor tasks was indistinguishable from the nonstutterers, while the stutterers were impaired relative to the other two groups.

Table 14

Outcome of Planned Comparisons (*p* values) for Motor Tasks. *p* values are one-tailed for comparisons on the left, two-tailed for comparisons on the right.

	<i>p</i> values	
	(NS + ES) vs. ST	NS vs. ES
Sequence Reproduction		
Response Initiation Time	.012	.162
Response Execution Time	.002	.624
First sequence errors	.102	.323
With Visual Feedback Crank Turning		
Integrated Error	.125	.374
Excess Output	.003	.409
No Visual Feedback Crank Turning		
Integrated Error	.0005	.846
Excess Output	.009	.259

On the divided visual field tasks, the exstutterers performed similarly to the stutterers in that they lacked performance asymmetries on the tasks. The nonstutterers had significant RVF advantages in the Lexical Decision Task and in the Dot Enumeration Task when only trials with SOA = 50 ms were considered. However, the attentional

Table 15

Outcome of Planned Comparisons (*p* values) for Divided Visual Field Tasks. *p* values are one-tailed for comparisons on the left, two-tailed for comparisons on the right.

	<i>p</i> values	
	NS vs. (ST + ES)	ST vs. ES
Lexical Decision d' (R - L)	.005	.847
Dot Enumeration d' (R - L)	.052	.885
^a Dot Enumeration SOA = 50 ms d' (R - L)	.005	.410

^aDot enumeration data restricted to trials with SOA = 50 ms due to poor accuracy on trials with SOA = 0 ms.

manipulation failed to influence performance asymmetries as expected: there were no statistically significant SOA x Side or Group x SOA x Side interactions in either the Lexical Decision or Dot Enumeration Data.

DISCUSSION

According to the dual factor model, two distinct neuropsychological factors interact with one another to give rise to the expression of stuttering in adults. Those two factors are: 1) a speech-motor fragility (believed to be related to the functioning of the SMA), and 2) some anomaly in interhemispheric relations, the exact nature of which is unclear.

There were two main purposes to the research. One purpose of the research was to replicate and generalize the evidence supporting the model outlined above. A second purpose was to test a series of hypotheses relating to neuropsychological factors that may be associated with recovery, or failure to recover, from stuttering. The two purposes are interrelated, since the design chosen to test the recovery model is predicated on a comparison of stutterers and exstutterers within the framework of the dual factor model. However, for the sake of clarity, the Discussion section is divided into two major subsections, each of which pertains primarily to one of the two major purposes described above.

1. Generalizing and Extending the Dual Factor Model in Adults.

One purpose of the research was to replicate and expand upon evidence previously advanced in support of the dual factor model of stuttering in adults. This was accomplished in a number of different ways. First, the research necessarily involved either replication or generalization of previous findings upon which the dual factor model is based. Second, the research offered an opportunity to directly address the issue of the separability of the two factors in the dual factor model. Third, the inclusion of the Dot Enumeration Task, and the attentional manipulation in both the Lexical Decision and Dot Enumeration Tasks, were attempts to gain further insight into the nature of the anomaly of

interhemispheric relations in adults who stutter. Each of these three issues will be discussed separately below.

(1). Replication and Generalization of Previous Findings.

Each of the tasks chosen for this experiment was based, either directly or indirectly, on tasks previously used to investigate stuttering in persistent adult stutterers. The results of the four tasks, as they relate to results of previous findings with similar or related tasks, will be discussed below. Note that the following discussion will be limited, for the most part, to the performance of nonstutterers and stutterers.

(a). Sequence Reproduction Task

The Sequence Reproduction Task reported here was very similar to the task used and reported by Webster (1986a). The version reported here had two major differences. First, it was a computer-based implementation, with the stimuli presented via graphics on the computer monitor rather than an LED display panel. Second, Sequence Length was included as a factor in the task reported here, whereas Webster (1986a) only analyzed and reported data for sequences of length four.

The findings reported here did replicate those of Webster (1986a), in that stutterers were slower in initiating the sequences, and tended to make more First Sequence Errors, than nonstutterers. One notable difference between the results reported here and those of Webster (1986a) is the statistically significant Sequence Execution Time main effect obtained here. Webster (1986a) reported no group differences in Sequence Execution Time. The reason for this discrepancy is not clear. Perhaps the most likely explanation is that there were differences in the instructions given to the participants, leading to a greater emphasis on accuracy in the present experiment. If the people who have an inherent difficulty in the organization of sequential responses, this could manifest itself in one of two, different ways: more errors, or longer sequence execution times. Webster (1986a)

found that stutterers generated significantly more first sequence errors than nonstutterers, but that their Sequence Execution Times did not differ. The opposite was observed here, suggesting that the stutterers placed greater emphasis on the accuracy of their tapping at the expense of Sequence Execution Time. The flexibility of these effects across experiments, and their presumed sensitivity to the experimenter's instructions, is consistent with the nature of the motor sequencing deficit being described here. People who stutter are *capable*, of accurately sequencing their responses on finger-tapping tasks, given that they dedicate sufficient processing resources to the task, and given that other factors do not interfere with performance (as is believed to be the case with speech in instances of stuttering).

Notwithstanding the discrepancy described above, the present results did replicate the most significant findings of Webster (1986a). Stutterers were slower than nonstutterers in initiating the response sequences required of the task, and were less efficient in executing those sequences (tending to make more errors and, in the present study, taking longer to do so). In addition to replicating those findings, the present results generalized them to a range of sequence lengths (three to five elements) and demonstrated that, within that range, the group difference is not related to sequence length. This observation is relevant to the issue of the neuroanatomical localization of the sequencing deficits observed on the task. According to a model of cerebellar function described by Inhoff *et al.* (1989), if the locus of the motor deficit were the cerebellum, one would expect that the group difference in the Response Initiation Times would have been related to Sequence Length. The present results showed that, while stutterers were slower overall, the relationship between RIT and Sequence Length in stutterers paralleled that of nonstutterers (and exstutterers) almost exactly.

The model of cerebellar function described by Inhoff *et al.* (1989) was based on the "Sequence Length Effect" reported by Sternberg *et al.* (1978). Sternberg *et al.* reported that

reaction times to initiate finger tapping sequences increased linearly as a function of sequence length. Inhoff *et al.* (1989) noted that the sequence length effect is abolished by cerebellar damage, and argued that the translation of a programmed sequence of responses into an action is a function of cerebellar structures.

It should be noted that the RITs obtained here were not a linear function of Sequence Length, and that there was in fact a small but statistically reliable decrease in RIT from sequences of length three to sequences of length four. This was likely due to methodological differences between the task reported here and those of Inhoff *et al.* (1989), and Sternberg *et al.* (1978).

The most important methodological differences were (1) the fact that trials of different sequence lengths were blocked in the other studies, whereas in the present study they were randomized, and participants did not know what the sequence length for each impending trial would be; and (2) the fact that in the other studies, the sequences were practiced prior to the initiation of experimental trials, whereas here they were not.

The randomized presentation of stimuli of different sequence lengths does introduce a confound that complicates the interpretation of effects related to the Sequence Length independent variable. As the last element in a five-element sequence was being presented, the participant knew that it was the last element, because he or she knew that no sequence had more than five elements. At the other extreme, as the third element in a three-element sequence was being presented, the participant could reasonably expect that there would be more elements coming (i.e. in 2/3 of all trials). The hesitation that such knowledge might cause could explain the drop in RIT from sequences of length three to sequences of length four. More generally, the fact that the degree of uncertainty would decrease with Sequence Length could explain the predominantly quadratic, rather than linear, relationship between Sequence Length and RIT, as illustrated in the diagram below.

Figure 13. Hypothetical influence of uncertainty about number of elements in sequence on Response Initiation Time in Sequence Reproduction Task.

The randomization of sequence lengths and the use of novel, unpracticed sequence lengths was necessary in order to remain consistent with previous tasks (Webster, 1986a, in particular) and with the predominant underlying suspicion that previously observed group differences on the task are related to the functioning of the SMA. These methodological considerations would be expected to minimize (though not necessarily eliminate) the relative cerebellar contribution to the motor component of the task. Furthermore, the novel, randomized nature of the stimuli in the present task also introduced a mnemonic component to task, particularly for the longer sequence lengths. Nevertheless, while all of the above factors may constitute additional sources of variability, they do not preclude the demonstration of a cerebellar contribution to the task. If the observed group difference was cerebellar in origin, then, according to the model of cerebellar function described by Inhoff *et al.* (1989), one would expect that difference to be sensitive to sequence length.

(b). Bimanual Crank-turning Task

The Bimanual Crank-Turning Task was created specifically for the present experiment (i.e. was not a replication) but was designed with a view to generalizing deficits observed on bimanual handwriting tasks (Greiner and Fitzgerald, 1992; Greiner *et al.* , 1986; Webster, 1988) to a task with more specific and well-documented demands on brain mechanisms related to bimanual coordination.

The results support the notion that adults who stutter do, as a group, have a kinesthetically-based bimanual coordination deficit. Under both visual feedback conditions, stutterers generated 40% more Excess Output than nonstutterers, and under no-visual-feedback conditions, they generated both 40% more excess output and 40% more Integrated Error. Overall, performance of the stutterers was substantially poorer than both the nonstutterers and exstutterers.

The above results indicate that stutterers' performance on the task was more reliant on visual feedback than nonstutterers. While stutterers generated 40% more Integrated Error than nonstutterers when visual feedback was unavailable, there was no group difference in overall accuracy when visual feedback was available. This suggests the possibility that the deficit is related to the kinesthetically-guided medial motor system (Goldberg, 1985), providing added support to the growing body of evidence that stuttering is associated with a focal anomaly in the functioning of the SMA. As discussed in the Introduction, Preilowski (1972; 1975) demonstrated very elegantly the association of the anterior motor systems (particularly the SMA) with kinesthetically-guided bimanual coordination and the association of the posterior corpus callosum with visual control of bimanual coordination.

In the studies of bimanual handwriting, a dependent measure of particular interest was the number of mirror reversals generated by the nondominant hand (Greiner and Fitzgerald, 1992; Greiner *et al.* , 1986; Webster, 1988). Mirror reversals are of special

interest because of their relationship to motor overflow and associated movements. The Bimanual Crank-Turning Task offers the opportunity to examine the tendency to make mirror reversals in a novel task without the overlearned laterality inherent in handwriting. Mirror reversal errors in bimanual crank-turning correspond directly to the Percent Excess Output dependent measure. In the present context, mirror reversals can be thought of as instances in which the participant moves either crank in the direction opposite to the required (clockwise) direction. Mirror reversals are the only errors that increase the value of the Percent Excess Output measure.

Overall accuracy is not necessarily directly related to generation of reversal errors, and thus the two dependent measures involve fundamentally different types of errors. It is possible for the path of the cursor to deviate substantially from the optimal path through the centre of the track, without any reversal errors being generated. This kind of error occurs when the ratio of the rate of rotation of the two cranks deviates from the required ratio (in this case 2:1), determined by the slope of the track. Such deviations in the timing of the two cranks will cause the cursor to deviate from the optimal path, thus increasing the Integrated Error. Thus, one may view the Integrated Error dependent measure as representative of *timing errors*, and the Percent Excess Output dependent measure as representative of *reversal errors*. In the strictest sense, reversal errors may be considered a special subset of timing errors, rather than being mutually exclusive of them¹.

It is interesting to examine the role of visual feedback in the context of the distinction provided above. When visual feedback was available, stutterers were differentiated from nonstutterers by their tendency to make more reversal errors, but not

¹ Any timing error may be viewed as an instance where one crank is being turned too slowly relative to the other. When the rate of turning of the too-slow crank drops below zero and becomes negative (relative to the required direction of turning), a reversal error has been generated.

more timing errors. Given that reversal errors may be viewed as a special case of timing errors, we may infer the following regarding the present results: while stutterers did not make more errors overall than nonstutterers when visual feedback was available, a greater proportion of the errors they did make were reversal errors. Stated differently, the presence of visual feedback allowed stutterers to control the number, *but not the kind*, of errors they made. Since associated movements and the tendency to make reversal errors have been linked to callosal development in children (associated movements: Cohen *et al.*, 1967; Connolly and Stratton, 1968; Lazarus and Todor, 1987; 1991; Wolff *et al.*, 1983), and SMA damage in adults (Chan and Ross, 1988) and non-human primates (Brinkman, 1981; 1984), the specific tendency of people who stutter to make reversal errors constitutes new evidence in support of the ideas that the speech-motor deficit in stuttering is either directly or indirectly related to the functioning of the anterior callosum and/or the SMA, and that the bimanual coordination deficit in adult stutterers involves neural mechanisms known to undergo maturation in normal children.

In summary, the data provide further evidence of a deficit of bimanual coordination in people who stutter, and added three lines of evidence that suggest SMA involvement in stuttering: (1) stutterers were impaired overall relative to both nonstutterers and exstutterers on the Bimanual Crank-Turning Task, a task which is believed to place significant demands on the anterior callosum and SMA; (2) stutterers' performance deteriorated, relative to nonstutterers and exstutterers, when visual feedback was removed, suggesting that the observed bimanual coordination deficit involves the kinesthetically-guided medial motor system (Goldberg, 1985) of which the SMA is an integral component; and (3), analysis of the kinds of errors made by the three groups indicates that stutterers had a specific tendency to make more reversal errors on the Bimanual Crank-Turning Task relative to the other groups. The latter point is of particular interest because a tendency to make mirror reversal

errors has been associated with both the development of the corpus callosum in children, and with damage to the SMA in adult humans and non-human primates.

(c). Divided Visual Field Lexical Decision Task

As summarized in the Introduction, a variety of studies have indicated that people who stutter are more likely to demonstrate either no asymmetry or a reversed asymmetry on linguistic divided visual field and dichotic listening tasks (Blood, 1985; Blood and Blood, 1989; Davenport, 1979; Hand and Haynes, 1983; Rosenfield and Goodglass, 1980; Quinn, 1972; Sommers, Brady and Moore, 1975). The results of the Lexical Decision Task are in agreement with those findings, both in terms of the overall size of the asymmetry in the groups, and in terms of the number of individuals in the groups who demonstrated reversed asymmetries. As a group, nonstutterers had a statistically significant performance asymmetry favouring the right visual field, whereas stutterers (and exstutterers) did not favour either visual field. The results of several studies that have reported similar findings were discussed in the Introduction.

It is also noteworthy that stutterers had slower overall reaction times than both nonstutterers and exstutterers on the Lexical Decision Task. This is in agreement with findings reported in the one other study involving stutters that employed a lexical decision task.

(d). Dot Enumeration Task

Past investigations that have used divided visual field and dichotic listening paradigms to investigate interhemispheric relations in people who stutter have, without exception, involved the use of verbal or linguistic stimuli (letters, consonant-vowel-consonant trigrams, words etc.). The Dot Enumeration Task used here was, in part, intended to address the issue of whether the anomalous performance asymmetries observed in people who stutter are limited to the verbal/linguistic stimulus class or whether they would also be observed with nonverbal stimuli such as the random dot patterns used here.

Overall performance on the Dot Enumeration Task was extremely poor, compared to the Lexical Decision Task (66% accuracy versus 76% accuracy, respectively). Even when trials with $SOA = 0$ ms were eliminated, and the data of participants whose performance was not significantly above chance were excluded, overall accuracy was below that which was observed on the Lexical Decision Task. There are a number of probable explanations for this. First, the stimuli for the Dot Enumeration Task consisted of random patterns of dots enclosed within a small square frame, and as such were novel, in contrast to the Word stimuli used in the Lexical Decision Task. Second, the task was probably simply more difficult, in terms of its perceptual and cognitive demands on the participants, than the Lexical Decision Task. Third, participant fatigue could well have contributed to the poor performance on the task, given that it was the last of the four tasks performed, and demanded a substantial degree of concentration from the participants over a period of 15 to 20 minutes.

While the observed asymmetry in the nonstutterers' performance was not in the expected direction, the group difference did closely parallel the one observed on the Lexical Decision Task. Both stutterers and exstutterers lacked the visual field advantage that was observed in nonstutterers on both tasks. This suggests that the anomaly of interhemispheric relations in people who stutter is not limited to verbal stimuli. That assertion is only reasonable inasmuch as (1) the performance of the nonstutterers in the present task can be considered normative, and (2) the stimuli used can be considered nonverbal. Unfortunately, the results were ambiguous in their support of those two assumptions, because the expected left visual field advantage in the nonstutterers' performance was not observed. On the contrary, when only trials with $SOA = 50$ ms were considered, nonstutterers had a right visual field advantage on the Dot Enumeration Task.

Within the framework of the two assumptions given above, there are a number of possible explanations for the right visual field advantage obtained by the nonstutterers in the Dot Enumeration Task.

First, it is possible that either the data do not represent a normative sample of nonstutterers, or the results do not reflect a normative representation of their performance. Given the results of the Lexical Decision Task, this is unlikely to be the case, though it cannot be ruled out.

Second, it is possible that the second assumption is invalid; i.e. it is possible that the task was essentially verbal in nature and was more dependent on left hemisphere than on right hemisphere structural mechanisms. While it has been reported that dot enumeration tasks yield fairly strong right visual field advantages in fluent individuals (Kimura, 1966; 1973), there was one major difference between the response mode employed by the previous studies and the one used here. In order to maintain consistency with the Lexical Decision Task, the present task involved an even/odd forced choice, rather than free recall of the number of dots. It is possible that people use a verbal strategy to represent the concepts of "even" and "odd," and that the process of making that even/odd decision predominated over the visuo-spatial component (i.e. the representation of the pattern and number of dots) of the task in determining the performance asymmetry. However, it must be considered that the process of making the even/odd decision, given that one has determined the absolute number of dots, is an inherently easy one, particularly in light of the fact that participants knew that there could only be three, four, five or six dots. On the other hand, it is clear from the overall level of performance on the task that perceiving and counting the dots, which were presented in random arrangements and for only 116 ms, was not easy for the participants. Thus, it is difficult, from a purely rational basis, to accept that the even/odd decision predominated over the visuospatial component of the task and thus accounted for the right visual field advantage that was observed.

A third possibility is that, in terms of its demands on lateralized structural mechanisms, the task was indeed either ambiguous or "right hemisphere/left visual field dominant" and that *other* factors that favoured the right visual field predominated. The two most obvious candidates would be pre-exposural attentional asymmetries and cognitive set. These two possibilities will be discussed below.

One possible factor that could have affected the direction of the asymmetry in the Dot Enumeration Task is attention. As discussed in the Introduction, there is a substantial body of evidence of an inherent preexposural attentional bias favouring the right visual field in fluent right-handed people (Lambert, Beard and Thompson, 1988; Leiderman, 1986; Levinthal, 1988; Mondor and Bryden, 1991; 1992a; 1992b; 1992c). As will be discussed in a subsequent section, the attempt to isolate that attentional bias by way of the stimulus precue (the box around the dots) was unsuccessful. This does not, however, preclude the possibility that preexposural attentional factors contributed to the performance asymmetry. Rather, it simply limits our ability to identify the *extent* to which attentional factors contributed to the observed performance asymmetry.

Mondor and Bryden (1992c) have speculated that there may be a relationship between task difficulty and the extent to which the inherent attentional bias favouring the right visual field influences perceptual asymmetries. Using a dichotic listening task involving identification of emotional content, they reported that an observed left ear advantage was reversed to a right ear advantage when the difficulty of the task was increased. They suggest that "when subjects are faced with a difficult dichotic task, there is a general tendency for right-handed subjects to bias their attention toward the right ear" (p. 393). This idea provides an appealing explanation of the present results, given the poor overall accuracy on the task.

Cognitive set is another possible explanation for the left visual field advantage in the performance of the nonstutterers on the Dot Enumeration Task. In the Introduction, the

role of cognitive set in divided visual field asymmetries was discussed, and evidence was cited which suggests that both the size and direction of attentional asymmetries can potentially be influenced by factors which affect the individual's cognitive set (Berlucchi *et al.*, 1974; Dee and Hannay, 1973; Hellige, 1978; Hellige and Cox, 1976; Kallman, 1978; Kinsbourne, 1970; Klein *et al.*, 1976). Of particular relevance to the present results is evidence that blocking trials of one stimulus class can induce a cognitive set that will exert an influence on performance of subsequent trials with a different stimulus class (e.g. Klein *et al.*, 1976). In the present experiment, all participants performed 160 trials of a Lexical Decision Task prior to performing the Dot Enumeration Task. Order of administration of the two tasks was deliberately not counterbalanced because it was considered more important to eliminate a potential source of variability in the performance on the more important Lexical Decision Task. However, measures were taken to minimize the potential for cognitive set carryover from the Lexical Decision Task to the Dot Enumeration Task. The Lexical Decision Task was the first of the four tasks administered, and the Dot Enumeration Task was the last. This meant that there was a pause of at least one hour between the two tasks, and that participants were thoroughly distracted by the two motor tasks during that pause. Furthermore, participants were kept naive of the precise nature of the tasks until the instructions were given, immediately before they were performed. Thus, they did not know that they would be performing another task similar to the Lexical Decision Task until they were given the instructions for the Dot Enumeration Task. This was done in order to encourage the participants to forget about the Lexical Decision Task and concentrate on the tasks they performed (Sequence Reproduction, BMCT) prior to the Dot Enumeration Task.

In summary, there are a number of possible explanations for the lack of a left visual field advantage on the Dot Enumeration Task. However, notwithstanding that limitation, the results do reveal an anomalous perceptual asymmetry (relative to the performance of the

nonstutterers) in both stutterers and exstutterers on a predominantly nonverbal task. At the least, the results suggest that the anomaly of interhemispheric relations in stutterers is unlikely to be limited to structural linguistic mechanisms.

(2). Separability of the Factors

An important issue with the Dual Factor model of stuttering is the question of the distinctiveness or separability of the two hypothesized factors that interact to give rise to stuttering. An inherent assumption of the model is that the two factors are distinct, and that stuttering reflects an interaction between anomalies within different neuropsychological systems. Given that one of the factors involves relatively low-level mechanisms of motor control, and that the other factor appears to involve high-level processes relating to interhemispheric interaction, the idea that the factors are separate seems reasonable on the surface. However, it is possible that the two factors represent different functional manifestations (i.e. in different systems) of a common underlying structural anomaly. In particular, it is difficult to ignore the possible common role of the corpus callosum in the two factors. As discussed previously, the two halves of the SMA have major reciprocal connections with one another via the corpus callosum (Rouiller *et al.*, 1994), and this may be of particular importance to bimanual coordination or, more generally, bilateral coordination. The SMA appears to play an important role in translating unilateral instructions onto bilaterally-paired speech structures via the corpus callosum. On the other hand, there is substantial evidence that the corpus callosum is also involved in interhemispheric interaction and the regulation of lateralized attentional processes (e.g. Bowers and Heilman, 1980; Dimond, 1976; 1979). Thus, an anomaly in the corpus callosum could potentially underlie both a fragility of speech-motor control and an anomaly of interhemispheric relations.

The present research offers a unique opportunity to empirically test the hypothesis that the two factors in the dual factor model are in fact distinct, rather than simply being different manifestations of a common underlying anomaly. The notion of separability of the factors is inherent in the model from which the predicted group differences on the two different classes of tasks were generated. According to that model, one of the conditions, the anomaly of interhemispheric relations, is present in both persistent and transient childhood stutterers, while the other condition, the fragility of speech-motor control is present only in persistent stutterers. The fact that the data supported those predictions provides a simple but clear illustration of the separability, or dissociability, of the two factors.

Furthermore, the fact that the exstutterers, as a group, exhibited the same anomalous performance on the divided visual field tasks as the stutterers demonstrates that simply possessing the anomaly of interhemispheric relations is not sufficient to cause stuttering in adults. This is an important contribution to the literature on interhemispheric relations in people who stutter, much of which continues to subscribe to the assumption that there is a direct causal relationship between some form of anomalous interhemispheric relations and stuttering (e.g. Moore, 1990; Nass, Schreter and Heier, 1994).

(3). Nature of the Anomaly of Interhemispheric Relations

The third way in which this research is relevant to the dual factor model of persistent stuttering in adults, was an attempt to gain insight into the nature of the anomaly of interhemispheric relations. As discussed in the Introduction, there are a number of possible explanations for the anomalous results that have been obtained with neuropsychological, neuroradiological and electrophysiological indices of interhemispheric relations in adults who stutter. The most frequently cited explanation of those results are the structural explanations. These explanations are variations of the cerebral dominance

model as originally described by Orton and Travis, and they generally refer to either a lack of, a reversal of, or a diminished gradient of cerebral dominance for speech and language. Alternatively, these findings could reflect an anomaly in the normally asymmetric distribution of attention between the two cerebral hemispheres. Such a hemispheric attentional anomaly could reflect a context-independent basis, such as a reduced or reversed gradient of hemispheric attentional asymmetry, or a less stable or more labile hemispheric asymmetry. Alternatively, a hemispheric attentional anomaly could involve context-dependent mechanisms, such as a diminished gradient of *structural* hemispheric specialization, or an anomaly in the higher level frontal lobe mechanisms responsible for the selective activation of appropriate cortical regions corresponding to a particular mental activity (Stuss and Benson, 1986). Another possible explanation for the frequently observed anomalies of interhemispheric relations would be that they are simply artifacts of the interaction between the individual's mental state (demand characteristics, emotions) and the task demands.

It was hoped that the research would provide insight into the anomaly of interhemispheric relations in two ways. First, the Dot Enumeration Task was included along with the Lexical Decision Task in order to address the issue of the context-specificity of the anomaly of interhemispheric relations. Second, the attentional precue was included to test the hypothesis that the anomalous performance asymmetries observed in people who stutter reflect an anomaly in the context-independent lateralized attentional mechanisms that underlie the inherent rightward attentional bias in fluent speakers.

(a). Inclusion of Dot Enumeration Task:

The results of the Dot Enumeration Task were discussed in the previous section. In general, interpretation of those results is problematic because of the poor overall accuracy on the task, and the unexpected asymmetry favouring the right visual field in the nonstutterers. However, as discussed earlier, the results do suggest that an anomaly of

interhemispheric relations can be demonstrated with nonverbal target stimuli in both stutterers and exstutterers. This suggests that the anomaly is not specific to the speech/language domain, and may involve more general laterality mechanisms.

(b). Attentional Manipulation

In both the Lexical Decision Task and the Dot Enumeration Task, the target stimuli were presented together with a lateralized attentional precue that always corresponded to the side of the target stimulus. The precue was presented either just before ($SOA = 67$ ms for Lexical Decision Task, $SOA = 50$ ms for Dot Enumeration Task) or simultaneous with ($SOA = 0$ ms) the target stimulus. Mondor and Bryden have shown, using a lexical decision divided visual field task (Mondor and Bryden, 1992a) and various dichotic listening tasks (Mondor and Bryden, 1991; 1992b; 1992c; Mondor, 1993), that the size of the perceptual asymmetry can be reduced or eliminated by preceding the target stimulus with an attentional precue such as the dot and box used here, and that this effect is dependent on the magnitude of the stimulus onset asynchrony.

The present results did not replicate those of Mondor and Bryden (1992a). The effect should have manifested itself, at least within the nonstutterers, as a Visual Field $\times$ SOA interaction, with a larger Visual Field asymmetry under $SOA = 0$ ms than under $SOA = 67$ ms (Lexical Decision) or $SOA = 50$ ms (Dot Enumeration). This was not observed in either task. The reason for this failure to replicate the findings of Mondor and Bryden (1992a) is unclear. It should be noted that in the present experiment, only one value of SOA was used, whereas Mondor and Bryden (1992a) used a range of different SOA values and examined the more general relationship between SOA and visual field asymmetry. They reported an increase in the degree to which the asymmetry was diminished with increasing SOA. With that in mind, the largest possible SOA was chosen for the present study. The magnitude of the SOA is limited by the fact that overall SOA + target stimulus

presentation time must be less than the minimal time (approximately 180 ms; Boer and Weijger, 1988) required to initiate an eye movement.

Because of the failure to replicate the findings of Mondor and Bryden (1992a), one is unable to draw any meaningful inferences from the present study regarding the possibility that the anomaly of interhemispheric relations in people who stutter is related to lateralized hemispheric attentional mechanisms.

II. Recovery from Stuttering.

Extending the Dual Factor Model to Account for Recovery

It was argued in the Introduction that the anomaly of interhemispheric relations is likely to be common to all children who stutter, while the speech-motor fragility is probably limited to those children whose stuttering will persist into adulthood. Furthermore, it was argued that because hemispheric asymmetries, both structural and attentional, are developmentally invariant, the anomaly of interhemispheric relations should still be demonstrable in adults who stuttered as children but who have since recovered. Since the neural mechanisms underlying speech-motor control do undergo maturation throughout childhood, it was suggested that this process of maturation may underlie the recovery from stuttering in those children who do eventually recover. Based on this reasoning the following hypotheses were made: 1) stutterers would be differentiated from fluent controls both on the tasks sensitive to the anomaly of interhemispheric relations *and* on tasks designed to probe the fragility of speech-motor control; 2) exstutterers would be differentiated from the controls (i.e. they would perform similarly to the stutterers) only on tasks designed to probe the anomaly of interhemispheric relations. These hypotheses are summarized symbolically below.

Speech-motor control tasks: (NS = ES) > ST

Divided visual field asymmetries: NS > (ES = ST)

Overview of Findings

For both of the motor tasks (Sequence Reproduction and Bimanual Crank-Turning) the results supported the hypotheses stated above. In both cases, the performance of stutterers on the tasks was impaired relative to nonstutterers and exstutterers combined, and the performance of exstutterers was not distinguishable from that of the nonstutterers. The results on the Sequence Reproduction task were somewhat less unambiguous in this regard, as the exstutterers' Response Initiation Times fell almost exactly halfway between those of the nonstutterers and stutterers. Clearly, these data alone could provide equally clear support to an alternative model that placed exstutterers' ability to rapidly initiate complex sequences at a midpoint on a continuum between that of nonstutterers and stutterers (or even a model in which exstutterers were considered to be equivalent in this regard to stutterers). However, any ambiguity inherent in this result is largely mitigated by a number of other factors: (1) the Sequence Execution Time data, on which the exstutterers were in fact slightly *faster* than the nonstutterers; (2) the fact that when RIT and SET are combined, the exstutterers' overall times (764 ms + 726 ms = 1490 ms) were clearly more similar to the nonstutterers' (689 ms + 753 ms = 1442 ms) than they were to the stutterers' (834 ms + 879 ms = 1713 ms) times; (3) the outcome of the multivariate planned comparisons (which supported the *a priori* model); and (4) the fairly clear support for the *a priori* hypotheses provided by the data from the Bimanual Crank-Turning Task.

The results of the DVF tasks also supported the *a priori* hypotheses. Stutterers lacked an asymmetry on the Lexical Decision Task, whereas the nonstutterers had the expected advantage favouring the right visual field. Importantly, for the purposes of the recovery model, exstutterers were similar to stutterers in that they lacked a performance

asymmetry on the Lexical Decision Task. The results of the Dot Enumeration Task were less clear, because the observation of a right visual field advantage on the task was contrary to what was expected based upon the nature of the task and past research (Kimura, 1966: 1973). Nevertheless, even on the Dot Enumeration Task, it is noteworthy that the exstutterers' performance was similar to that of the stutterers in that both groups lacked any asymmetry on the task.

It is particularly interesting to note that, on the divided visual field tasks, the performance of the exstutterers was similar to that of the stutterers in terms of the lateral performance asymmetry, but was similar to the nonstutterers in terms of overall reaction times. For both tasks, the stutterers had substantially slower overall reaction times than both nonstutterers and exstutterers.

Taken together, the results support the hypotheses that (1) an anomaly of interhemispheric relations is a predisposing factor to stuttering in childhood, and (2) the failure to recover during childhood is associated with a permanent deficit of speech-motor control that exists over and above any fragility inherent in the immature brain.

While the results suggest that the anomaly of interhemispheric relations is not sufficient to cause stuttering in adults, they do not address the possibility that a more pronounced deficit of speech-motor control may, in some cases, be sufficient to cause stuttering.

Similarly, it is not clear whether the anomaly of interhemispheric relations is sufficient to cause transient childhood stuttering, or if other internal or external factors are involved. Even if it could be shown that the anomaly of interhemispheric relations is present in all children who stutter, it does not logically follow that all children with the same pattern of interhemispheric relations will stutter. If there are such children who do not stutter, then it remains to ask what psychological, environmental and/or neurological

factors characterize the difference between those children and the children who do experience transient childhood stuttering.

Clinical Implications of the Recovery Model

An understanding of the neuropsychological mechanisms associated with recovery and failure to recover from stuttering could have valuable implications for the identification and treatment of children who stutter. In particular, the results of the present research suggest at least two ways in which such knowledge could aid clinicians in both diagnosis and prognosis.

First, if a developmentally invariant anomaly of interhemispheric relations could be shown to be a predisposing factor for childhood stuttering, then a test that could identify that condition in preschool-aged children would offer a means of early identification of children at risk for stuttering.

Second, the fragility of speech-motor control could potentially be used as a means of identifying children whose stuttering is unlikely to spontaneously recover. As discussed in the Introduction, motor sequencing and bimanual coordination continue to develop throughout childhood. The question of whether or not the speech-motor control fragility that persists into adulthood differs quantitatively or qualitatively from the normal immature state cannot be answered by the present research. Only a longitudinal design, in which individuals were followed throughout childhood and adolescence, could properly address that question.

Stated differently, the present study relates only to the endpoint of development with respect to the speech-motor control mechanisms of interest, and not to the developmental course taken to reach that endpoint. However, if future research indicated that a speech-motor fragility in young persistent stutterers was distinguishable from the normative (immature) state, then that condition could be used to identify those children whose stuttering would not be expected to recover spontaneously.

Classification of Exstutterers

The classification of adults into the categories of "stutterer" and "exstutterer" is fundamental to the model for recovery from stuttering being investigated here. The rationale behind that classification was discussed in the Introduction. In short, it is based on the observation that many, and perhaps most, children who stutter appear to spontaneously "grow out of it" before reaching adulthood. The term "spontaneous" is only intended to convey the idea that these recoveries do not appear to be the result of formal treatment. Thus, for the purposes of this study, exstutterers were defined simply as individuals who report having stuttered as children but who report that they no longer stutter as adults.

However, it is important to note that there is controversy surrounding the issue of recovery from stuttering, and there are those (e.g. Ingham, 1983) who deny that spontaneous recovery occurs at all. The responses to the questionnaires administered to the participants offer some information relevant to that issue. Ideally, one would hope that the scores on the PSI would correspond perfectly with the predictions that are inherent in the stutterer/exstutterer classification. Specifically, one would expect the scores of exstutterers to be indistinguishable from those of the stutterers on the childhood version of the PSI, and indistinguishable from those of the nonstutterers on the adult version.

In fact, while the exstutterers' scores were similar to the stutterers on the childhood version of the PSI, they were significantly higher than the nonstutterers on the adult version. The two comparisons will be discussed in turn below.

There was no significant difference between the exstutterers and the stutterers on the overall childhood-version PSI scores. However, there was an interaction between Group and Subscale, which indicated that the exstutterers did differ significantly from the stutterers on the Avoidance subscale. The Avoidance scale is unique in that, of the three, it is the most removed from the actual speech-motor component of stuttering. The fact that

the exstutterers recalled substantially less avoidance behaviour in their childhoods than the nonstutterers could potentially have considerable clinical implications. While it would be dangerous to imply causality from a correlational finding involving a retrospective self-report questionnaire such as the childhood-version PSI, it nevertheless highlights the importance of psychological issues in stuttering, particularly in children. However, of primary importance to the present study, the lack of significant group differences in the overall PSI score and in the Struggle and Expectancy subscale scores does provide a degree of validation to the retrospective self-classification of individuals as exstutterers.

On the adult-version PSI, the exstutterers did have significantly higher PSI scores than the nonstutterers. However, this group difference must be interpreted within the context of a number of factors. First, as illustrated by Figure 2, the exstutterers were more similar to the nonstutterers than the stutterers on the adult version of the PSI. Second, the difference between the exstutterers and nonstutterers was small, amounting to approximately three out of twenty responses on the Struggle subscale and four out of twenty responses on the Expectancy subscale. Third, both past research (e.g. Shearer and Williams, 1965) and the Likert severity scale reported here indicate that exstutterers do retain a tendency to "stutter" (or possibly to simply be more sensitive to their own speech disfluencies) when tired or under stress.

In spite of the mitigating factors outlined above, it is possible that individuals who were stutterers "neuropsychologically," were classified as exstutterers. The differences observed between the exstutterers and the other groups on the PSI suggest that if any misclassifications did occur, they probably involved well-managed stutterers being classified as exstutterers. This would be more of a concern had the results of the four tasks not supported the *a priori* hypotheses. On the motor tasks, misclassification of stutterers as exstutterers would have militated against obtaining the results that were both predicted and observed. Yet there were no significant group differences between the nonstutterers and

exstutterers on any of the measures of motor performance. In fact, on some measures (Integrated Error in WVF crank-turning, SET on the Sequence Reproduction Task), the overall performance of the exstutterers tended to be superior to that of the nonstutterers. In light of these observations, it is unlikely that the sample of exstutterers contained a substantial number of misclassified stutterers.

The other potential misclassification involving exstutterers would be classification of nonstutterers as exstutterers. It is possible, for example, that the operational definition of "stutterer" being used here is not appropriate when applied retrospectively to one's childhood, based upon the memory of the individual or his or her parents. This concern is particularly important because of the high incidence of developmental disfluency in preschool-aged children. Developmental disfluency is considered by most speech pathologists as distinct from true stuttering, though the criteria by which the two forms of disfluency are distinguished are not clear. In the present study, the selection criterion was deliberately intended to address these concerns, by excluding those whose age of onset and recovery from stuttering were too young to distinguish them with confidence from simple developmental disfluency. This is the reason that the mean ages for onset (5.6 years) and recovery (14.6 years) from stuttering in the present sample are higher than the mean ages typically reported in unselected samples (Andrew and Harris, 1964; Andrews *et al.*, 1983).

Furthermore, misclassification of nonstutterers as exstutterers would have militated against the predicted and observed findings on the divided visual field tasks. On both the Lexical Decision and Dot Enumeration Tasks, the performance of the exstutterers was similar to the stutterers, and different from the nonstutterers.

In summary, the most important validation of the classification of individuals into "stutterers" and "exstutterers" is their performance on the two different classes of experimental tasks. An important issue in the present context is whether or not

“exstutterers” are indeed recovered stutterers, or if they are simply individuals who have learned to manage their stuttering, perhaps to the point where it is essentially automatic to do so. In the latter case, the term “exstutterer” would have value only as a phenomenological description of the individual’s current speech fluency relative to an earlier state. The results of the experimental tasks do support the underlying assumption of this research that the classification has a more fundamental basis in qualitative neuropsychological differences between adult stutterers and exstutterers.

III. Synthesis: The Dual Factor Model in Persistent and Transient Childhood Stuttering

At this point, it is possible to reevaluate the Dual Factor Model as it applies to persistent stuttering in adults, and to extend it to include an account of spontaneous recovery from stuttering. In doing so, the model will be summarized in terms of five key elements distributed over four levels: (1) neuropsychological factors including (a) a speech-motor fragility and (b), the anomaly of interhemispheric relations; (2) psychological factors; (3) developmental factors; and (4) the interaction between the above four elements.

Neuropsychological Factors

Speech-Motor Fragility

The key aspects of the speech-motor fragility in stuttering are a deficit in the sequencing and timing of complex response sequences, and, in particular, difficulty with the organization and initiation of those complex response sequences. The present results reinforce the notion that stuttering is associated with those general motor deficits, but that, in adults, they are only present in persistent stutterers and are not evident in those who have recovered from stuttering in childhood. The results also support previous findings suggestive of a mild bimanual coordination deficit in adults who stutter, particularly when bimanual coordination relies heavily on kinesthetic sensory feedback. These findings,

taken together with the above, suggest that the SMA may be the locus of the speech-motor fragility evident in stuttering.

For the purposes of the model, the speech-motor fragility is considered to be associated with momentary breakdowns in the fluent sequencing of the multiple movement components inherent in speech. However, it is not believed to be sufficient, in most cases, to cause full blown stuttering, which typically involves more profound speech blockages and loss of control than a relatively mild speech-motor fragility could explain.

Interhemispheric Relations

The second condition of the dual factor model is an anomaly of interhemispheric relations. The nature of the anomaly is not clear, but it is most typically manifested experimentally as an unusual lateral asymmetry on divided visual field and dichotic listening tasks, or as elevated right hemispheric activation in electrophysiological or neuroradiological studies. While the mechanisms remains unclear, our preferred explanation of the anomaly of interhemispheric relations is that it manifests itself functionally by altering the balance of activation between the left and right hemispheres.

According to the model being proposed, the anomaly of interhemispheric relations is present in both persistent adult stutterers and transient childhood stutterers.

Psychological Factors

As discussed in the Introduction, it is a long-established characteristic of the syndrome of stuttering that situational and psychological factors can have a profound influence on the speech of the person who stutters. Typically, situations that evoke anxiety or stress can either trigger speech difficulties or exacerbate existing ones.

The syndrome of stuttering extends well beyond the confines of neuropsychological anomalies involving the speech-motor system or interhemispheric relations. It extends beyond the speech disfluencies that characterize the act of stuttering. It has the potential to influence, not merely the ability to speak fluently, but to *communicate*, to engage in social

intercourse, and to function successfully within society. People who stutter often manage their condition by way of maladaptive coping mechanisms that may include various forms of avoidance, ranging from avoiding the use of specific speech sounds or words, to avoidance of certain social situations and, in the worst scenario, social contact in general (Van Riper, 1982). These strategies are often reinforced by the attitudes that people who stutter encounter within society (Carlisle, 1985). The negative experiences associated with stuttering may lead to overt fears of the sounds, words and situations associated with those experiences. Van Riper (1982) reports that two common characteristics of people who stutter, and children in particular, are low self esteem and a negatively distorted self-image.

All of the psychosocial aspects of stuttering mentioned above will clearly have an impact on the emotional state of the person who stutters. Since the speech fluency of people who stutter may be influenced by their emotional state (Caruso *et al.*, 1994; Trotter and Bergmann, 1957; Van Riper, 1937; 1982), those psychosocial factors are themselves capable of exerting considerable influence over the expression of stuttering. In the context of the dual factor model, the most likely explanation for the association between speech fluency and negative emotions is that they exert their influence via the anomaly of interhemispheric relations by influencing patterns of hemispheric activation.

Developmental Factors

It is proposed that recovery from stuttering is under the ultimate influence of developmental factors. More specifically, the relative immaturity of the corpus callosum and SMA in young children, and the process of maturation of those structures throughout childhood, is proposed to underlie the developmental variability in speech fluency that characterizes transient childhood stuttering. According to this idea, the immaturity of the corpus callosum and the SMA in young children carries with it an inherent fragility in the neural mechanisms that underlie the organization and initiation of complex sequential response sequences. This situation is seen as analogous to the permanent speech-motor

fragility that exists throughout adulthood in persistent stuttering. However, in transient childhood stuttering the speech-motor fragility resolves itself eventually as the neural mechanisms mature, permitting a gradual improvement in speech fluency.

Interaction Between the Above Factors

The dual factor model proposes that the two neuropsychological factors interact with one another in a positive-feedback relationship (i.e. a "neural amplification system") to give rise to the more profound characteristics of stuttering that cannot be explained in terms of a mild fragility of motor sequencing. Because the act of stuttering is a negative emotional experience, it would be expected to be associated with activation of the right hemisphere (Ahern and Schwartz, 1985; Davidson and Fox, 1982; Fox and Davidson, 1988). The anomaly of interhemispheric relations may predispose the individual to greater, or more prolonged, than normal right hemisphere activation in such situations. Excessive right hemisphere activation could, in turn, influence the speech system in one of at least two different ways. First, excessive activity in either cerebral hemisphere could serve as a source of neural interference with a fragile speech-motor system that is predisposed to momentary breakdowns. Alternatively, right hemisphere activation could exert an indirect influence on speech by drawing limited attentional resources away from speech-motor control mechanisms associated with stuttering. In either case, the result would be a worsening of the initial trigger stimulus itself (the speech-motor disfluency), and an ensuing "snowball effect," which could amplify a minor disfluency to the point where it becomes a major speech blockage. The power of this "neural amplification system" (see Figure 14) concept is its ability to explain the utter loss of control of a fragile speech apparatus that in other respects, and on other occasions, functions normally. The speech-motor fragility described here, and by Webster (1990c), does not appear to be sufficiently severe to account for the more severe aspects of stuttering.

Neural Amplification System

Figure 14. Illustration of the neural amplification system that characterizes the relationship between the fragility of speech-motor control and the anomaly of interhemispheric relations in stuttering.

Factors that influence emotional arousal and right hemisphere activation would be expected to exert a parallel influence on speech fluency. Thus, whereas the two neuropsychological factors (speech-motor fragility and the anomaly of interhemispheric relations) affect speech-fluency in a proximate way, emotional and psychosocial factors exert a higher-level modulating influence that determines patterns of situational variability in the expression of stuttering.

Development would, in those who will ultimately recover, exert an even higher level influence on the overall life history of the disorder, by placing chronological constraints on the expression of the stuttering.

The "neural amplification system" illustrated in Figure 14 represents an organ-level explanation of the impairment of speech production associated with stuttering. In a proximate sense, one may view the act of stuttering as being the result of an interaction between speech-motor and hemispheric attentional factors, as represented by Figure 14 and the oval at the bottom of Figure 15. At the person-level, a fairly complex model emerges, in which the *syndrome* of stuttering is proposed to be the result of an interaction between neuropsychological, developmental and psychosocial influences (see Figure 15). Neuropsychological factors are under the influence of developmental and psychosocial influences, they in turn can have reciprocal consequences on the higher levels of functioning, and it is at the interaction between those levels that the situational and developmental variability inherent in stuttering is mediated.

The model provides some insight into the means by which different clinical therapies exert their influence on overall fluency. The most effective therapies for the treatment of stuttering are those which treat the speech-motor aspect of the disorder as the primary characteristic and focus directly on improving fluency (Andrews, Guitar and Howie, 1980; Andrews *et al.*, 1983). Those therapies (e.g. Precision Fluency Shaping Program, R.L. Webster, p. 303) typically involve the application of learning principles to

3

OF/DE

3

PM-1 3½"x4" PHOTOGRAPHIC MICROCOPY TARGET
NBS 1010a ANSI/ISO #2 EQUIVALENT

Figure 15. Hierarchical model illustrating the relationship between neuropsychological, psychosocial/affective, and developmental factors in the expression and modulation of stuttering.

alter the speech-response complex in an effort to generate fluent sounding speech. As discussed earlier, these treatment approaches do not lead to a cure for stuttering. Rather, it is likely that the techniques learned in speech therapy serve to impose a more rigorous structure and greater focus on the speech mechanism. This might lead to greater activation of the appropriate speech-motor mechanisms and greater resiliency of those mechanisms to neural interference (Webster, 1990c).

While it is clear that treatments for stuttering can lead to significant improvement in fluency, the resiliency of these gains in the environment beyond the clinic, particularly over long periods of time, is in question. The gains achieved by these therapies are not always permanent, and appear to deteriorate over time (Boberg, Howie and Woods, 1979; Mowrer, 1975). Alluding to the issue of right hemispheric activation, Webster and Poulos (1989) suggest that a critical component in the transfer and long-term maintenance of fluency gains achieved in the clinic is an effort to teach people who stutter how to manage the psychological aspects of their disorder. By controlling potential sources of interference with efforts to control speech, such as anxiety and "inappropriate self-talk," gains in fluency are more likely to be transferred from the clinic to the "real world," and to be better maintained (Webster and Poulos, 1989).

Summary and Conclusions

1. Adults who stutter have deficits in bimanual coordination and in the organization and initiation of complex response sequences. On the bimanual coordination task, their performance deteriorated further when visual feedback was removed. When visual feedback was available, they were able to monitor and control the number of errors they made, but they did make more reversal errors under these conditions. Taken together, these findings support the contention that stuttering that persists into adulthood is associated with a mild deficit in the functioning of the supplementary motor area.

2. Both adults who stutter, and adults who stuttered only as children, have an anomaly of interhemispheric relations that manifests itself experimentally as a greater likelihood of generating anomalous performance asymmetries on tasks such as the divided visual field tasks employed here.

3. Since the anomalous performance asymmetry was also observed in stutterers and exstutterers when a Dot Enumeration Task was used, the anomaly of interhemispheric relations appears not to be limited to verbal or linguistic processing, but may involve more general mechanisms of interhemispheric relations.

4. However, beyond the observation stated above, the results did not allow for an improved understanding of the anomaly of interhemispheric relations.

5. Since the anomaly of interhemispheric relations was observed in recovered stutterers, it is unlikely that there is a simple and direct causal relationship between that condition and stuttering. This is an important observation inasmuch as it relates to the cerebral dominance hypothesis and related theories which propose a direct causal relationship between stuttering and some anomaly of hemispheric specialization or activation.

6. The fact that there was evidence of an anomaly of interhemispheric relations in both stutterers and exstutterers, but that only stutterers demonstrated a fragility of speech-motor control, supports the hypothesis that recovery from stuttering may be associated with maturation of speech-motor mechanisms.

7. The two factors in the Dual Factor Model appear to have distinct neuropsychological bases, since only one of them (the fragility of speech-motor control) was observed in recovered stutterers, whereas both were evident in persistent stutterers.

8. The fact that the dissociation between the two factors described above was observed in the two groups suggests that the classification of individuals as "stutterers" and "exstutterers" is a neuropsychologically meaningful one.

REFERENCES

- Abbs, J.H. and Gracco, V.L. (1983). Sensorimotor actions in the control of multi-movement speech gestures. Trends in Neurosciences, 6, 391-395.
- Ahern, G.L. and Schwartz, G.E. (1985). Differential lateralization for positive and negative emotion in the human brain: E.E.G. spectral analysis. Neuropsychologia, 23, 745-755.
- Ambrose, N.G., Yairi, E. and Cox, N. (1993). Genetic aspects of early childhood stuttering. Journal of Speech and Hearing Research, 36, 701-706
- Andrews, G. and Harris, M. (1964). The syndrome of stuttering. Clinics in Developmental Medicine, 17.
- Andrews, G., Craig, A., Feyer, A., Hoddinott, S., Howie, P. and Neilson, M. (1983). Stuttering: A review of research findings and theories circa 1982. Journal of Speech and Hearing Disorders, 48, 226-246.
- Andrews, G., Guitar, B. and Howie, P. (1980). Meta-analysis of the effects of stuttering treatment. Journal of Speech and Hearing Disorders, 45, 287-307.
- Andrews, G., Morris-Yates, A., Howie, P., and Martin, N.G. (1991). Genetic factors in stuttering confirmed. Archives of General Psychiatry, 48, 1034-1035.
- Andrews, G., Quinn, P.T., and Sorby, W.A. (1972). Stuttering: an investigation into cerebral dominance for speech. Journal of Neurology, Neurosurgery, and Psychiatry, 35, 414-418.

- Annett, M. (1978). Family handedness in three generations predicted by the right shift theory. Annals of Human Genetics, 42, 479-491.
- Ballard, P.B. (1912). Sinistrality and Speech. Journal of Experimental Pediatrics, 1, 298-310.
- Basser, L.S. (1962). Hemiplegia of early onset and the faculty of speech with special reference to the effects of hemispherectomy. Brain, 85, 427-460.
- Beaumont, J.G. (1982). Divided Visual Field Studies Of Cerebral Organization. New York: Academic Press.
- Beaumont, J.G. and Colley, M. (1980). Attentional bias and visual field asymmetry. Cortex, 16, 391-396.
- Beckie, J. and Hiscock, M. (1991). Focused attention in children: Overcoming the right-ear advantage. Paper presented at Canadian Psychological Association Annual Convention, Calgary.
- Berlucchi, G., Brizzolara, D., Marzi, C.A., Rizzolata, G. and Umilatà, C. (1974). Can lateral asymmetries in attention explain interfield differences in visual perception? Cortex, 10, 1177-185.
- Bishop, D.V.M. (1984). Using non-preferred hand skill to investigate pathological left-handedness in an unselected population. Developmental Medicine and Child Neurology, 26, 214-226.
- Bishop, D.V.M. (1990). Handedness and Developmental Disorder. Hillsdale: Lawrence Erlbaum Associates.

- Blood, G.W. (1985). Laterality differences in child stutterers: Heterogeneity, severity levels, and statistical treatments. Journal of Speech and Hearing Disorders, 50, 66-72.
- Blood, G.W. and Blood, I.M. (1986). Relationship between specific disfluency variables and dichotic listening in stutterers. Perceptual and Motor Skills, 62, 337-338.
- Blood, G.W. and Blood, I.M. (1989). Laterality preferences in adult female and male stutterers. Journal of Fluency Disorders, 14, 1-10.
- Blood, G.W., Blood, I.M. and Newton, K.R. (1986). Effect of directed attention on cerebral asymmetries in stuttering adults. Perceptual and Motor Skills, 62, 351-355.
- Bloodstein, O. (1960a). The development of stuttering: I. Changes in nine basic features. Journal of Speech and Hearing Disorders, 25, 219-237.
- Bloodstein, O. (1960a). The development of stuttering: I. Developmental Phases. Journal of Speech and Hearing Disorders, 25, 366-376.
- Bloodstein, O. (1970). Stuttering and normal nonfluency: A continuity hypothesis. British Journal of Disorders of Communication, 5, 30-39.
- Bloodstein, O. (1981). A Handbook on Stuttering. Chicago: National Easter Seal Society.
- Bloodstein, O. (1984). Stuttering as an anticipatory struggle disorder. In R.F. Curlee and W.H. Perkins (Eds.), Nature and Treatment of Stuttering. San Diego: College-Hill Press.
- Bloodstein, O. (1990). On Pluttering, skivering and flogging: A commentary. Journal of Speech and Hearing Disorders, 55, 392-393.

- Boberg, E., Howie, P. and Woods, L. (1979). Maintenance of fluency: A review. Journal of Fluency Disorders, 4, 93-116.
- Boer, L.C. and Weijger, E.C.M. (1988). Eye movements and stages of processing. Acta Psychologica, 67, 3-18.
- Boles, D.B. (1979). Laterally biased attention with concurrent verbal load: Multiple failures to replicate. Neuropsychologia, 17, 353-361.
- Boles, D.B. (1987). Reaction time asymmetry through bilateral versus unilateral stimulus presentation. Brain and Cognition, 6, 321-333.
- Boles, D.B. (1990). What bilateral displays do. Brain and Cognition, 12, 205-228.
- Boliek, C.A., Obrzut, J.E. and Shaw, D. (1988). The effects of hemispatial and asymmetrically focused attention on dichotic listening with normal and learning-disabled children. Neuropsychologia, 26, 417-433.
- Bowers, D. and Heilman, K. (1980). Material specific hemispheric activation. Neuropsychologia, 18, 309-319.
- Bradshaw, J.L. and Nettleton, N.C. (1981). The nature of hemispheric specialization in man. Behavioral and Brain Science, 4, 51-91.
- Bradshaw, J.L., Burden, V. and Nettleton, N.C. (1986). Dichotic and dichaptic techniques. Neuropsychologia, 26, 79-90.
- Brain, R.W. (1945). Speech and handedness. The Lancet, 837-842.
- Brebner, J., Shepard, M. and Cairney, P. (1972). Spatial relationships and the motor system. Acta Psychologica, 36, 1-15.

- Brickner, R.M. (1940). A human cortical area producing repetitive phenomena when stimulated. Journal of Neurophysiology, 3, 128-130.
- Brinkman, C. (1981). Lesions of the supplementary motor area interfere with a monkey's performance on a bimanual coordination task. Neuroscience Letters, 27, 267-270.
- Brinkman, C. (1984). Supplementary motor area of the monkey's cerebral cortex: Short- and long-term deficits after unilateral ablation, and the effects of subsequent callosal section. Journal of Neuroscience, 4, 918-929.
- Bryden, M.P. (1965). Tachistoscopic recognition, handedness and cerebral dominance. Neuropsychologia, 3, 1-8.
- Bryden, M.P. (1978). Strategy effects in the assessment of hemispheric asymmetries. In G. Underwood (Ed.), Strategies of Information Processing. New York: Academic Press.
- Bryden, M.P. (1982). Laterality: Functional asymmetry in the intact brain. New York: Academic Press.
- Bryden, M.P. and Allard, F.A. (1981). Do auditory perceptual asymmetries develop? Cortex, 17, 313-318.
- Bryden, M.P. and Mondor, T.A. (1991). Attentional factors in visual field asymmetries. Canadian Journal of Psychology, 45, 427-447.
- Bryden, M.P. and Rainey, C.A. (1963). Left-right differences in tachistoscopic recognition. Journal of Experimental Psychology, 65, 568-571.

- Bryden, M.P., Munhall, K. and Allard, F. (1983). Attentional biases and the right-ear effect in dichotic listening. Brain and Language, 18, 236-248 .
- Bryngelson, B. (1931). Treatment of stuttering. Journal of Expression, 5, 19-26.
- Bryngelson, B. (1935). Sidedness as an etiological factor in stuttering. Journal of Genetic Psychology, 48, 204-217.
- Bryngelson, B. (1940). A study of laterality of stutterers and normal speakers. The Journal of Social Psychology, 11, 151-155.
- Bryngelson, B. and Rutherford, B. (1937). A comparative study of laterality of stutterers and non-stutterers. Journal of Speech Disorders, 2, 15-16.
- Burt, C. (1937). The Backward Child. London: University of London Press.
- Carlisle, J.A. (1985). Tangled Tongue: Living With a Stutter. Toronto: University of Toronto Press.
- Caruso, A.J., Abbs, J.H. and Gracco, V.L. (1988). Kinematic analysis of multiple movement coordination during speech in stutterers. Brain, 111, 439-455.
- Caruso, A.J., Chodzko-Zajko, W.J., Bidinger, D.A. and Sommers, R.K. (1994). Adults who stutter: Responses to cognitive stress. Journal of Speech and Hearing Research, 37, 746-754.
- Caruso, A.J., Conture, E.G. and Colton, R.H. (1988). Selected temporal parameters of coordination associated with stuttering in children. Journal of Fluency Disorders, 13, 57-82.

- Chan, J.L. and Ross, E.D. (1988). Left-handed mirror writing following right anterior cerebral artery infarction: Evidence for non-mirror transformation of motor programs by right supplementary area. Neurology, 38, 59-63.
- Cohen, G. (1982). Theoretical interpretations. In J.G. Beaumont (Ed.), Divided Visual Field Studies Of Cerebral Organization. New York: Academic Press.
- Cohen, H.J., Taft, L.T., Mahadeviah, M.S. and Birch, H.G. (1967). Developmental changes in overflow in normal and aberrantly functioning children. The Journal of Pediatrics, 71, 39-47.
- Colby, K.M. and Parkinson, C. (1977). Handedness in autistic children. Journal of Autism and Childhood Schizophrenia, 7, 3-9.
- Coltheart, M. (1981). The MRC Psycholinguistic Database. Quarterly Journal of Experimental Psychology, 33A, 497-505.
- Connolly, K. and Stratton, P. (1968). Developmental changes in associated movements. Developmental Medicine and Child Neurology, 10, 49-56.
- Conture, E.G., Colton, R.H. and Gleason, J.R. (1988). Selected temporal aspects of coordination during fluent speech of young stutterers. Journal of Speech and Hearing Research, 31, 640-653.
- Cooper, E.B. (1972). Recovery from stuttering in a junior and senior high school population. Journal Of Speech And Hearing Research, 15, 632-638.
- Cooper, E.B. and Wells, M.T. (1974). Prevalence of and recovery from speech disorders in a group of freshmen at the University of Alabama. ASHA, 16, 359-360.

- Coriat, I.H. (1927). The oral-erotic components of stammering. International Journal of Psychoanalysis, 8, 56-69.
- Cox, M.D. (1982). The stutterer and stuttering: Neuropsychological correlates. Journal of Fluency Disorders, 7, 1129-140.
- Curry, F.K.W. and Gregory, H.H. (1969). The performance of stutterers on dichotic listening tasks thought to reflect cerebral dominance. Journal of Speech and Hearing Research, 12, 78-82.
- Daniels, E.M. (1940). An analysis of the relation between handedness and stuttering with special reference to the Orton-Travis theory of cerebral dominance. Journal of Speech Disorders, 5, 309-326.
- Davenport, R.W. (1979). Dichotic listening in four severity levels of stuttering. ASHA Abstracts, 769.
- Davidson, R.J. and Fox, N.A. (1982). Asymmetrical brain activity discriminates between positive and negative affective stimuli in human infants. Science, 218, 1235-1237.
- Dearborn, W.F. (1933). Structural factors which condition special disability in reading. Proceedings of the American Association for Mental Deficiency, 38, 266-283.
- Dee, H.L. and Hannay, H.J. (1973). Asymmetry in perception: Attention versus other determinants. Acta Psychologica, 37, 241-247.
- Deecke, L. (1987). Bereitschaftspotential as an indicator of movement preparation in supplementary motor area and motor cortex. In R. Porter (Ed.) Motor Areas of the Cerebral Cortex. Ciba Foundation Symposium 132. Chichester: Wiley.

- Deecke, L. Kornhuber, L.L., Lang, W. and Schreiber, H. (1985). Timing function of the frontal cortex in sequential motor and learning tasks. Human Neurobiology, 4, 143-154.
- Deiber, M.P., Passingham, R.E., Colebatch, J.G., Friston, K.J., Nixon, P.D. and Frackowiak, R.S.J. (1991). Cortical areas and the selection of movement: A study with positron emission tomography. Experimental Brain Research, 84, 393-402.
- Denkla, M.B. (1973). Development of speed in repetitive and successive finger-movements in normal children. Developmental Medicine and Child Neurology, 15, 635-645.
- Denkla, M.B. (1974). Development of motor co-ordination in normal children. Developmental Medicine and Child Neurology, 16, 729-741.
- Dennis, M. (1976). Impaired sensory and motor differentiation with corpus callosum agenesis: A lack of callosal inhibition during ontogeny. Neuropsychologia, 14, 455-469.
- Dennis, M. and Kohn, B. (1975). Comprehension of syntax in infantile hemiplegics after cerebral hemidecortication: Left-hemisphere superiority. Brain and Language, 2, 472-482.
- Dennis, M. and Whitaker, H. (1976). Language acquisition following hemidecortication: Linguistic superiority of the left over the right hemisphere. Brain and Language, 3, 404-433.
- Dickson, S. (1971). Incipient stuttering and spontaneous remission of stuttered speech. Journal of Communication Disorders, 4, 99-110.

- Dickstein, P.W. and Tallal, P. (1987). Attentional capabilities of reading-impaired children during dichotic presentation of phonetic and complex non-phonetic sounds. Cortex, 23, 237-249.
- Dimond, S.J. (1976). Depletion of attentional capacity after total commissurotomy in man. Brain, 99, 347-356.
- Dimond, S.J. (1979). Performance by split-brain humans on lateralized vigilance tasks. Cortex, 15, 43-50.
- Efron, R. (1990). The Decline and Fall of Hemispheric Specialization. Hillsdale: Lawrence Erlbaum.
- Efron, R., Yund, E.W. and Nichols, D.R. (1987). Scanning the visual field without eye movements – a sex difference. Neuropsychologia, 25, 637-644.
- Ellenberg, L. and Sperry, R.W. (1979). Capacity for holding sustained attention following commissurotomy. Cortex, 15, 421-438.
- Eriksen, C.W. and St. James, J.D. (1986). Visual attention within and around the field of focal attention: A zoom lens model. Perception and Psychophysics, 40, 225-240.
- Etcoff, N.L. (1986). The neuropsychology of emotional expression. In G. Goldstein, and R.E. Tarter (Eds.) Advances in Clinical Neuropsychology. New York: Plenum Press.
- Fagard, J. (1987). Bimanual stereotypes: Bimanual coordination in children as a function of movements and relative velocity. Journal of Motor Behavior, 19, 355-366.
- Fagard, J. and Pezé, A. (1992). Coupling and Lateralization in bimanual coordination at 7, 8, and 9 years of age. Developmental Neuropsychology, 8, 69-85.

- Fagar1, J., Morioka, M. and Wolff, P.H. (1985). Early stages in the acquisition of a bimanual motor skill. Neuropsychologia, 23, 535-543.
- Felton, R.H., Wood, F.B., Brown, I.S., Campbell, S.K. and Harter, M.R. (1987). Separate verbal memory and naming deficits in attention deficits in attention deficit disorder and reading disability. Brain and Language, 31, 171-184.
- Fog, E. and Fog, E. (1963). Cerebral inhibition examined by associated movements. In Bax, M., and MacKeith, R.C. (Eds.), Minimal Cerebral Dysfunction: Clinics in Developmental Medicine (no. 10). London: Heinemann Medical.
- Forster, D.C. (1993). Bimanual Coordination in Left and Right-Handers: Evidence of Stronger Interhemispheric Coupling in Left-Handers. Unpublished manuscript.
- Forster, D.C. and Webster, W.G. (1991). Concurrent task interference in stutterers: Dissociating hemispheric specialization and activation. Canadian Journal of Psychology, In Press.
- Fox, N.A. and Davidson, R.J. (1988). Patterns of brain electrical activity during facial signs of emotion in 10-month-old infants. Developmental Psychology, 24, 230-236.
- Freund, H.J. (1984). Premotor areas in man. Trends in Neurosciences, 7, 481-483.
- Fried, I., Katz, A., McCarthy, G., Sass, K.J., Williamson, P., Spencer, S.S. and Spencer, D.D. (1991). Functional organization of human supplementary motor cortex studied by electrical stimulation. The Journal of Neuroscience, 11, 3656-3666.
- Galaburda, A.M. and Kemper, T.L. (1979). Cytoarchitectonic abnormalities in developmental dyslexia: A case study. Annals of Neurology, 39, 333-336.

- Galaburda, A.M., Sherman, G.F., Rosen, G.D. Aboitiz, F. and Geschwind, N. (1985). Developmental Dyslexia: Four consecutive patients with cortical anomalies. Annals of Neurology, 18, 222-233.
- Gardner, E.B. and Branski, D.M. (1976). Unilateral cerebral activation and perception of gaps: A signal detection analysis. Neuropsychologia, 14, 43-53.
- Gawryszewski, L.G., Riggio, L., Rizzolatti, G. and Umiltà. (1987). Movements of attention in the three spatial dimensions and the meaning of "neutral" cues. Neuropsychologia, 25, 19-29.
- Gazzaniga, M.S. (1987). Perceptual and attentional processes following callosal section in humans. Neuropsychologia, 25, 119-133.
- Geffen G. (1978). The development of the right ear advantage in dichotic listening with focused attention. Cortex, 14, 169-177.
- Geffen, G. and Sexton, M.A. (1978). The development of auditory strategies of attention. Developmental Psychology, 14, 11-17.
- Geffen, G. and Wale, J. (1979). Development of selective listening and hemispheric asymmetry. Developmental Psychology, 15, 138-146.
- Gianotti, G. (1972). Emotional behavior and hemispheric side of the lesion. Cortex, 8, 41-55.
- Goldberg, G. (1985). Supplementary motor area structure and function: Review and hypotheses. The Behavioral and Brain Sciences, 8, 567-617.

- Goldman-Rakic, P.S., Isseroff, A., Schwartz, M.L. and Bugbee, N.M. (1983). The neurobiology of cognitive development. In P. Mussen (Ed.) Handbook of Child Psychology: Biology and Infancy Development. New York: Wiley.
- Goodale, M.A. (1988). Hemispheric differences in motor control. Behavioural and Brain Sciences, 30, 203-214.
- Goodglass, H. and Barton, M. (1963). Handedness and differential perception of verbal stimuli in left and right visual fields. Perceptual and Motor Skills, 17, 851-854.
- Goodglass, H. and Calderon, M. (1977). Parallel processing of verbal and musical stimuli in right and left hemispheres. Neuropsychologia, 15, 397-407.
- Gracco, V.L. (1988). Timing factors in coordination of speech movements. The Journal of Neuroscience, 8, 4628-4639.
- Gracco, V.L. and Abbs, J.H. (1983). Variant and invariant characteristics of speech movements. Experimental Brain Research, 65, 156-166.
- Grafton, S.T. (1994). Cortical control of movement. Annals of Neurology, 36, 3-4.
- Greiner, J.R., Fitzgerald, H.E., and Cooke, P.A. (1986). Bimanual hand writing in right-handed and left-handed stutterers and non-stutterers. Neuropsychologia, 24, 441- 47.
- Greiner, J.R. and Fitzgerald, H.E. (1992). Bimanual handwriting reveals delayed interhemispheric integration in childhood stuttering. Developmental Neuropsychology, 8, 395-411.
- Hand, C.R. and Haynes, W.O. (1983). Linguistic processing and reaction time differences in stutterers and nonstutterers. Journal of Speech and Hearing Research, 26, 181-185.

- Hardyck, C. Chiarello, C., Dronkers, N.F. and Simpson, G.V. (1985). Orienting attention within visual fields: How efficient is interhemispheric transfer? Journal of Experimental Psychology, 11, 650-666.
- Hebb, D.O. (1949). The Organization of Behavior. New York: Wiley.
- Heilman, K.M. and Van Den Abell, T. (1979). Right hemisphere dominance for mediating cerebral activation. Neuropsychologia, 17, 315-321.
- Heilman, K.M. and Van Den Abell, T. (1980). Right hemisphere dominance for attention: The mechanism underlying hemispheric asymmetries of inattention (neglect). Neurology, 30, 327-330.
- Heilman, R.K., Scholes, R., and Watson, R.T. (1975). Auditory affective agnosia: Disturbed comprehension of affective speech. Journal of Neurology, Neurosurgery, and Psychiatry, 38, 69-72.
- Hellige, J.B. (1978). Visual laterality patterns for pure- versus fixed-list presentation. Journal of Experimental Psychology: Human Perception and Performance, 1, 121-131.
- Hellige, J.B. and Cox, P.J. (1976). Effects of concurrent verbal memory on recognition of stimuli from the left and right visual fields. Journal of Experimental Psychology, 2, 210-221.
- Heron, W. (1957). Perception as a function of retinal locus and attention. American Journal of Psychology, 70, 38-48.

- Hines, D., Sutker, L.W., Satz, P. and Altman, A. (1976). Recall of letters from the left and right visual half-fields under unilateral and bilateral presentation. Perceptual and Motor Skills, 42, 531-539.
- Hiscock, M. and Kinsbourne, M. (1977). Selective listening asymmetry in preschool children. Developmental Psychology, 13, 217-224.
- Hiscock, M. and Kinsbourne, M. (1980). Asymmetries of selective listening and attention switching in children. Developmental Psychology, 16, 70-82
- Hiscock, M. and Stewart, C. (1984). The effect of asymmetrically focused attention upon subsequent ear differences in dichotic listening. Neuropsychologia, 22, 337-351.
- Howie, P. (1981). Concordance for stuttering in monozygotic and dizygotic twin pairs. Journal of Speech and Hearing Research, 24, 317-321.
- Humphries, P., Kaufmann, W.E. and Galaburda, A.M. (1991). Developmental dyslexia in women: Neuropathological findings in three cases. Manuscript submitted for publication.
- Ikeda, A., Lüders, H.O., Shibasaki, H., Collura, T.F., Burgess, R.C., Morriss, H.M. and Hamano, T. (1995). Movement-related potentials associated with bilateral simultaneous and unilateral movements recorded from human supplementary motor area. Electroencephalography and Clinical Neurophysiology, 95, 323-334.
- Ingham, R.J. (1983). Spontaneous remission of stuttering: When will the emperor realize he has no clothes on? In D. Prins and R.J. Ingham (Eds.) Treatment of Stuttering in Early Childhood. San Diego: College-Hill Press.

- Ingham, R.J. (1990). commentary on Perkins (1990) and Moore and Perkins (1990): On the valid role of reliability in identifying "What is Stuttering?" Journal of Speech and Hearing Disorders, 55, 394-397.
- Ingram, D. (1975). Cerebral speech lateralization in young children. Neuropsychologia, 13, 103-105.
- Inhoff, A.W. Diener, H.C., Rafal, R.D. and Ivry, R. (1989). The role of cerebellar structures in the execution of serial movements. Brain, 112, 565-581.
- Jeeves, M.A., Silver, P.H. and Milne, A.B. (1988). Role of the corpus callosum in the development of a bimanual motor skill. Developmental Neuropsychology, 4, 305-323.
- Johnson, W. (1959). The Onset of Stuttering. Minneapolis: University of Minneapolis Press.
- Jonas, S. (1981). The supplementary motor region and speech emission. Journal of Communication Disorders, 14, 349-373.
- Jones, R.K. (1966). Observations on stammering after localized cerebral injury. Journal of Neurology, Neurosurgery, and Psychiatry, 29, 192-195.
- Jones, R.K. (1967). Dyspraxic ambiphasia: a neurophysiologic theory of stammering. Transactions of the American Neurological Association, 92, 197-201.
- Jonides, J. and Yantis, S. (1988). Uniqueness of abrupt visual onset in capturing attention. Perception and Psychophysics, 43, 346-354.
- Joullian, A.L., Viswanath, N.S. and Rosenfield, D.B. (1995). Motor processes underlying incipient and chronic stuttering. Annals of Neurology, 38, 285.

- Kahneman, D. (1973). Attention and Effort. Englewood Cliffs: Prentice Hall.
- Kallman, H.J. (1978). Can expectancy explain reaction time ear asymmetries? Neuropsychologia, 16, 225-228.
- Kent, R.D. (1984). Stuttering as a temporal programming disorder. In Curlee, R.F. and Perkins, W.H., (Eds.), Nature and Treatment of Stuttering: New Directions. San Diego: College-Hill Press.
- Kershner, J. and Graham, N.A. (1995). Attentional control over language lateralization in dyslexic children: deficit or delay?. Neuropsychologia, 33, 39-51.
- Kershner, J. and Morton, L.L. (1990). Directed attention dichotic listening in reading disabled children: A test of four models of maladaptive lateralization. Neuropsychologia, 28, 181-198.
- Kershner, J., Henninger, P. and Cooke, W. (1984). Writing induces a right hemisphere linguistic advantage in dysphonetic dyslexic children: Implications for attention and capacity models of laterality. Brain and Language, 21, 104-122.
- Kershner, J., Thomae, R. and Callaway, R. (1977). Nonverbal fixation control in young children induces a left-field advantage in digit recall. Neuropsychologia, 15, 569-576.
- Kidd, K.K. (1984). Stuttering as a genetic disorder. In Curlee, R.F. and Perkins, W.H., (Eds.), Nature and Treatment of Stuttering: New Directions. San Diego: College-Hill Press.
- Kidd, K.K., Kidd, J.R. and Records, M.A. (1978). The possible causes of the sex ratio in stuttering and its implications. Journal of Fluency Disorders, 3, 13-23.

- Kidd, K.K., Heimbuch, R.C. and Records, M.A. (1981). Vertical transmission of susceptibility to stuttering with sex-modified expression. Proceedings of the National Academy of Sciences, 78, 606-610.
- Kimura, D. (1961). Cerebral dominance and the perception of verbal stimuli. Canadian Journal of Psychology, 15, 166-171.
- Kimura, D. (1963). Speech lateralization in young children as determined by an auditory test. Journal of Comparative and Physiological Psychology, 56, 899-902.
- Kimura, D. (1964). Left-right differences in the perception of melodies. Quarterly Journal of Experimental Psychology, 16, 355-358.
- Kimura, D. (1966). Dual functional asymmetry in the brain in visual perception. Neuropsychologia, 4, 275-285.
- Kimura, D. (1973). The asymmetry of the human brain. Scientific American, 228, 70-78.
- Kimura, D. (1977). Acquisition of a motor skill after left-hemisphere damage. Brain, 100, 527-542.
- Kinsbourne, M. (1970). The cerebral basis of lateral asymmetries in attention. Acta Psychologica, 33, 193-201.
- Kinsbourne, M. (1975). The ontogeny of cerebral dominance. Annals of the New York Academy of Sciences, 263, 244-250.
- Kinsbourne, M. (1989). Mechanisms and development of hemisphere specialization in children. In C.R. Reynolds and E. Fletcher-Janzen (Eds.) Handbook of Clinical Neuropsychology. Plenum Press, New York.

- Kinsbourne, M. and Cook, J. (1971). Generalized and lateralized effects of concurrent verbalization on a unimanual skill. Quarterly Journal of Experimental Psychology, 23, 341-345.
- Kinsbourne, M. and Hicks, R.E. (1978). Functional cerebral space: A model for overflow, transfer and interference in human performance. In J. Requin (Ed.) Attention and Performance VII. Hillsdale: Erlbaum.
- Klein, D., Moscovitch, M. and Vigna, C. (1976). Attentional mechanisms and perceptual asymmetries in tachistoscopic recognition of words and faces. Neuropsychologia, 14, 55-66.
- Kolb, B.K. and Fantie, B. (1989). Mechanisms and development of hemisphere specialization in children. In C.R. Reynolds and E. Fletcher-Janzen (Eds.) Handbook of Clinical Neuropsychology. Plenum Press, New York.
- Lambert, A.J., Beard, C.T. and Thompson, R.J. (1988). Selective attention, visual laterality, awareness and perceiving the meaning of parafoveally presented words. The Quarterly Journal of Experimental Psychology, 40, 615-652.
- Lang, W., Obrig, H., Lindinger, G., Cheyne, D. and Deecke, L. (1990). Supplementary motor area activation while tapping bimanually different rhythms in musicians. Experimental Brain Research, 79, 504-514.
- Laplane, D. Talairach, V., Meininger, V., Bancaud, J. and Orgogozo, J.M. (1977). Clinical consequences of corticectomies involving the supplementary motor area in man. Journal of the Neurological Sciences, 34, 301-314.

- Larsen, B., Skinhøj, E. and Lassen, N.A. (1978). Variations in regional cortical blood flow in the right and left hemispheres during automatic speech. Brain, 101, 193-209.
- Lazarus, J.C. and Todor, J.I. (1987). Age differences in the magnitude of associated movement. Developmental Medicine and Child Neurology, 29, 726-733.
- Lazarus, J.C. and Todor, J.I. (1991). The role of attention in the regulation of associated movement in children. Developmental Medicine and Child Neurology, 33, 32-39.
- Leventhal, G. (1988). Cerebral dominance and attentional bias in word recognition. Perceptual And Motor Skills, 66, 791-800.
- Levy, J. (1985). Interhemispheric collaboration: Single-mindedness in the Asymmetric Brain. Hemispheric function and collaboration in the child. New York: Academic Press.
- Lezak, M.D. (1983). Neuropsychological Assessment. New York: Oxford University Press.
- Liederman, J. (1986). Determinants of the enhancement of the right visual field advantage by bilateral vs. unilateral stimuli. Cortex, 22, 553-565.
- Luessenhop, A.J., Boggs, J.S., Laborwit, L.J. and Walle, E.L. (1973). Cerebral dominance in stutterers determined by Wada testing. Neurology, 23, 1190-1192.
- Luria, A.R. (1970). Traumatic Aphasia. The Hague: Mouton.
- MacKay, D.G. and MacDonald, M.C. (1984). Stuttering as a sequencing and timing disorder. In Curlee, R.F. and Perkins, W.H., (Eds.), Nature and Treatment of Stuttering: New Directions. San Diego: College-Hill Press.

- Martin, R. and Haroldson, S. (1986). Stuttering as involuntary loss of speech control: Barking up a new tree. Journal of Speech and Hearing Disorders, 51, 187-191.
- McClean, M.D. (1990). Neuromotor aspects of stuttering: Levels of impairment and disability. ASHA Reports, 18, 64-71.
- McKeever, W.F. and Huling, M.D. (1971). Lateral dominance in tachistoscopic word recognition performances obtained with simultaneous bilateral input. Neuropsychologia, 9, 15-20.
- Mesulam, M.M. (1981). A cortical network for directed attention and unilateral neglect. Archives of Neurology, 10, 309-325.
- Molfese, D.L., Freeman, R.B. and Palermo, D.S. (1975). The ontogeny of brain lateralization for speech and non-speech stimuli. Brain and Language, 2, 356, 368.
- Mondor, T.A. and Bryden, M.P. (1991). The influence of attention on the dichotic REA. Neuropsychologia, 29, 1179-1190.
- Mondor, T.A. and Bryden, M.P. (1992a). On the relation between visual spatial attention and visual field asymmetries. Quarterly Journal of Experimental Psychology, 44, 529-555.
- Mondor, T.A. and Bryden, M.P. (1992b). Orienting of auditory spatial attention: Effects of a lateralized tone cue. Neuropsychologia, 30, 743-752.
- Mondor, T.A. and Bryden, M.P. (1992c). On the relation between auditory spatial attention and auditory perceptual asymmetries. Perception and Psychophysics, 52, 393-402.

- Moore, S.E. and Perkins, W.H. (1990). Validity and reliability of judgments of authentic and simulated stuttering. Journal of Speech and Hearing Disorders, 55, 383-391.
- Moore, W.H. (1976). Bilateral tachistoscopic word perception of stutterers and normal subjects. Brain and Language, 3, 434-442.
- Moore, W.H. (1984). Central nervous system characteristics of stutterers. In R.F. Curlee and W.H. Perkins (Eds.), Nature and Treatment of Stuttering: New Directions. San Diego: College-Hill.
- Moore, W.H. (1986). Hemispheric alpha asymmetries of stutterers and nonstutterers for the recall and recognition of words and connected reading passages: some relationships to severity of stuttering. Journal of Fluency Disorders, 11, 71-89.
- Moore, W.H. (1990). Pathophysiology of stuttering: Cerebral activation differences in stutterers vs. nonstutterers. ASHA Reports, 18, 72-80.
- Moore, W.H. and Haynes, W.O. (1980). Alpha-hemispheric asymmetry and stuttering: Some support for a segmentation dysfunction hypothesis. Journal of Speech and Hearing Research, 23, 229-247.
- Moore, W.H., Craven, D.C., and Faber, M.M. (1982). Hemispheric alpha asymmetries of words with positive, negative, and neutral arousal values preceding tasks of recall and recognition: electrophysiological and behavioural results from stuttering males and nonstuttering males and females. Brain and Language, 17, 211-224.
- Morris, H.H., Dinner, D.S., Luders, H., Wyllie, E. and Kramer, R. (1988). Supplementary motor area seizures: Clinical and electroencephalographic findings. Neurology, 38, 1075-1082.

- Moscovitch, M. (1986). Afferent and efferent models of visual perceptual asymmetries: Theoretical and empirical implications. Neuropsychologia, 24, 91-144.
- Mowrer, D. (1975). Technical Research Report S-1: Reduction of Stuttering Behavior. Tempe: University of Arizona.
- Nass, R., Schreter, B. and Heier, L. (1994). Acquired stuttering after a second stroke in a two-year-old. Developmental Medicine and Child Neurology, 36, 73-78.
- Neafsey, E.J. (1985). Preparation yes, intention no. The Behavioral and Brain Sciences, 8, 594-595.
- O'Leary, D.S. (1980). A developmental study of interhemispheric transfer in children aged five to ten. Child Development, 51, 743-750.
- Obrzut, J.E., Conrad, P.F., Bryden, M.P. and Boliek, C.A. (1988). Cued dichotic listening with right-handed, left-handed, bilingual and learning-disabled children. Neuropsychologia, 26, 119-131.
- Obrzut, J.E., Mondor, T.A. and Uecker, A. (1993). The influence of attention on the dichotic REA with normal and learning disabled children. Neuropsychologia, 31, 1411-1416.
- Oldfield, R.C. (1971). The assessment and analysis of handedness: The Edinburgh Inventory. Neuropsychologia, 9, 97-113.
- Ojemann, R.H. (1931). Studies in handedness: III. Relation of handedness to speech. Journal of Educational Psychology, 22, 120-126.

Orton, S.T. (1925). A physiological theory of reading disability and stuttering in children. New England Journal of Medicine. 199, 1045-1052.

Orton, S.T. (1937). Reading, writing and speech problems in children. New York: Norton.

Panelli, C.A., McFarlane, S.C. and Shipley, K.G. (1978). Implications of evaluating and intervening with incipient stutterers. Journal of Fluency Disorders. 3, 41-50.

Pascual-Leone, A., Cohen, L.G., Wassermann, E.M. and Hallett, M. (1994). The role of the supplementary motor area (SMA) in the coordination of bimanual movements. Neurology. 44 (Suppl. 2). A329.

Passler, M.A., Isaac, W. and Hynd, G.W. (1985). Neuropsychological development of behavior attributed to frontal lobe functioning in children. Developmental Neuropsychology. 1, 349-370.

Penfield, W. and Rasmussen, T. (1950). The Cerebral Cortex Of Man: A Clinical Study Of Localization Of Function. New York: MacMillen.

Penfield, W. and Welch, K. (1951). The supplementary motor area of the cerebral cortex: A clinical and experimental study. American Medical Association Archives of Neurology and Psychiatry. 66, 289-317.

Pennington, B.F., Smith, S.D., Kimberling, W.J., Green, P.A. and Haith, M.M. (1987). Left-handedness and immune disorders in familial dyslexics. Archives of Neurology. 44, 634-639.

Perkins, W.H. (1983). The problem of definition: Commentary on "Stuttering." Journal of Speech and Hearing Disorders. 48, 246-249.

- Perkins, W.H. (1984). Stuttering as a categorical event: Barking up the wrong tree – Reply to Wingate. Journal of Speech and Hearing Disorders, 49, 431-434.
- Perkins, W.H. (1986). More bite for a bark: Epilogue to Martin and Haroldson's Letter. Journal of Speech and Hearing Disorders, 51, 190-191.
- Perkins, W.H. (1990a). What is stuttering? Journal of Speech and Hearing Disorders, 55, 370-382.
- Perkins, W.H. (1990b). Gratitude, good intentions and red herrings: A response to commentaries. Journal of Speech and Hearing Disorders, 55, 402-404.
- Peters (1976). Unilateral control of bilaterally symmetrical movement as a factor in lateralization of speech. Perceptual and Motor Skills, 42, 841-842.
- Peters, M. (1983). Differentiation and lateral specialization in motor development. In G. Young, S.J. Segalowitz, C.M. Corter and S.E. Trehub (Eds.), Manual Specialization and the Developing Brain. New York: Academic Press.
- Peters, M. (1987). A non-trivial motor performance difference between right-handers and left-handers: Attention as an intervening variable in the expression of handedness. Canadian Journal of Psychology, 41, 91-99.
- Peters, M. (1988). Footedness: Asymmetries in foot preference and skill and neuropsychological assessment of foot movement. Psychological Bulletin, 103, 179-192.
- Petersen, S.E., Fox, P.T., Posner, M.I., Mintum, M. and Raichle, M.E. (1988). Positron emission tomographic studies of the cortical anatomy of single-word processing. Nature, 331, 585-589.

- Pinsky, S.D. and McAdam, D.W. (1980). Electrophysiological and dichotic indices of cerebral laterality in stutterers. Brain and Language, 11, 374-397.
- Ponsford, R.E., Brown, W.S., Marsh, J.T. and Travis, L.E. (1975). Evoked correlates of cerebral dominance for speech in stutterers and non-stutterers. E.E.G. Journal, 39, 43.
- Pool, K., Devous, M.D., Freeman, F.J., Watson, B.C. and Finitzo, T. (1991). Regional cerebral blood flow in developmental stutterers. Archives of Neurology, 48, 509-512.
- Pool, K., Freeman, F.J., and Finitzo, T. (1987). Brain electrical activity mapping: Applications to vocal motor control disorders. In H.F.M. Peters and W. Hulstijn (Eds.) Speech Motor Dynamics in Stuttering. New York: Springer-Verlag.
- Porfert, A.R. and Rosenfield, D.B. (1978). Prevalence of stuttering. Journal of Neurology, Neurosurgery and Psychiatry, 41, 954-956.
- Posner, M.I., Snyder, C.R.R. and Davidson, B.J. (1980). Attention and the detection of signals. Journal of Experimental Psychology: General, 109, 160-174.
- Preilowski, B.F. (1972). Possible contribution of the anterior forebrain commissures to bilateral motor coordination. Neuropsychologia, 10, 267-277.
- Preilowski, B.F. (1975). Bimanual motor interaction: Perceptual-motor performance of partial and complete "split-brain" patients. In, K.T. Zülch, O. Creutzfeld and G.C. Galbraith (Eds.), Cerebral Localization. Berlin: Springer.
- Quinan, C. (1921). Sinistrality in relation to high blood pressure and defects of speech. Archives of internal medicine, 27, 255-261.

- Quinn, P.T. (1972). Stuttering: Cerebral dominance and the dichotic word test. The Medical Journal of Australia, 2, 639-643.
- Qvarström, M.J., Jaroma, S.M. and Laine, M.T. (1994). Changes in the peripheral speech mechanism of children from the age of 7 to 10 years. Folia Phoniatria Logopaedia, 46, 193-202.
- Rankin, J.M., Aram, D.M. and Horwitz, D.B. (1981). Language ability in right and left hemiplegic children. Brain and Language, 14, 292-306.
- Rao, S.M., Binder, J.R., Bandettini, P.A., Hammeke, T.A., Yetkin, F.Z., Jesmanowicz, A., Lisk, L.M., Morris, G.L., Mueller, W.M., Estkowski, L.D., Wong, E.C., Houghton, V.M. and Hyde, J.S.. (1993). Functional magnetic resonance imaging of complex human movements. Neurology, 43, 2311-2318.
- Rastatter, M.P. and Dell, C. (1987). Vocal reaction times of stuttering subjects to tachistoscopically presented concrete and abstract words: a closer look at cerebral dominance and language processing. Journal of Speech and Hearing Research, 30, 306-310.
- Records, M.A., Heimbuch, R.C., and Kidd, K.K. (1977). Handedness and stuttering: a dead horse? Journal of Fluency Disorders, 2, 271-282.
- Remington, R. and Pierce, L. (1984). Moving attention: Evidence for time-invariant shifts of visual selective attention. Perception and Psychophysics, 35, 393-399.
- Robbins, J. and Klee, T. (1987). Clinical assessment of oropharyngeal motor development in young children. Journal of Speech and Hearing Disorders, 52, 271-277.

- Roland, P.E. (1984a). Metabolic measurements of the working frontal cortex in man. Trends in Neurosciences, 7, 430-435.
- Roland, P.E. (1984b). Organization of motor control by the normal human brain. Human Neurobiology, 2, 205-216.
- Roland, P.E., Skinhøj, B., Lassen, N.A. and Skinhøj, E. (1980). Supplementary motor area and other cortical areas in organization of voluntary movements in man. Journal of Neurophysiology, 43, 118-136.
- Roland, P.E., Skinhøj, E., Larsen, B. and Lassen, N.A. (1977). The role of different cortical areas in the organization of voluntary movements in man. A regional cerebral blood flow study. Acta Neurologica Scandinavica, Suppl. 64, 542-543.
- Romo, R. and Schultz, W. (1987). Neuronal activity preceding self-initiated or externally timed arm movements in area 6 of monkey cortex. Experimental Brain Research, 67, 656-662.
- Rosenfield, D.B. and Goodglass, H. (1980). Dichotic listening tests of cerebral dominance in stutterers. Brain and Language, 11, 170-180.
- Ross, E.D. (1981). The aprosodias. Archives of Neurology, 38, 561-569.
- Rostomily, R.C., Berger, M.S., Ojemann, G.A. and Lettich, E. (1991). Postoperative deficits and functional recovery following removal of tumors involving the dominant hemisphere supplementary motor area. Journal of Neurosurgery, 75, 62-68.
- Rouiller, E.M., Babalian, A., Kazennikov, O., Moret, V., Yu, X.-H., and Wiesendanger, M. (1994). Transcallosal connections of the distal forelimb representations of the

primary and supplementary motor cortical areas in macaque monkeys. Experimental Brain Research, 102, 227-243.

Rutter, M., Tizard, K., and Whitmore, K. (1970). Education, Health and Behaviour. Longman: London.

Salami, A. (1978). Commissural transmission time: Maturational changes in humans. Science, 200, 1409-1411.

Schell, G.R. and Strick, P.L. (1984). The origin of thalamic inputs to the arcuate premotor and supplementary motor areas. The Journal of Neuroscience, 4, 539-560.

Schweinberger, S.R., Sommer, W., Stiller, R. (1993). Event-related potential (ERP) indicators of cerebral lateralization in a divided visual field study. Journal of Clinical and Experimental Neuropsychology, 15, 393.

Seider, R.A., Gladstien, K.L. and Kidd, K.K. (1983). Recovery and persistence of stuttering among relatives of stutterers. Journal of Speech and Hearing Disorders, 48, 394-402

Seitz, K.S. and McKeever, W.F. (1984). Unilateral versus bilateral presentation methods in the reaction time paradigm. Brain and Cognition, 3, 413-425.

Seth, G. (1957). Psychomotor control in stammering and normal subjects: An experimental study. British Journal of Psychology, 49, 139-143.

Shaiman, S. (1989). Kinematic and electromyographic responses to perturbation of the jaw. Journal of the Acoustical Society of America, 86, 78-88.

- Shames, G.H. and Beams, H.L. (1956). Incidence of stuttering in older age groups. Journal of Speech and Hearing Disorders, 21, 313-316.
- Shanon, B. (1979). Lateralization effects in lexical decision tasks. Brain and Language, 8, 380-387.
- Sharkey, S.G. and Folkins, J.W. (1985). Variability of lip and jaw movements in children and adults. Journal of Speech and Hearing Research, 28, 8-15.
- Sheehan, J.G. (1958). Conflict theory of stuttering. In J. Eisenson (Ed.), Stuttering: A Symposium. New York: Harper and Row.
- Sheehan, J.G. (1979). Current issues on stuttering and recovery. In H.H. Gregory (Ed.), Controversies About Stuttering Therapy. Baltimore: University Park Press.
- Sheehan, J.G. and Martyn, M.M. (1966). Spontaneous recovery from stuttering. Journal of Speech and Hearing Research, 9, 121-135.
- Sheehan, J.G. and Martyn, M.M. (1970). Stuttering and its disappearance. Journal of Speech and Hearing Research, 13, 279-289.
- Shibasaki, H., Sadato, H., Lyshdow, H., Yonekura, Y., Honda, M., Nagamine, T., Suwazono, S., Magata, Y., Ikeda, A., Miyazaki, M., Fukuyama, H., Asato, R. and Kinishi, J. (1993). Both primary motor cortex and supplementary motor area play an important role in complex finger movement. Brain, 116, 1387-1398.
- Shucard, D.W., Cummins, K.R., and McGee, M.G. (1984). Event-related brain potentials in normal and disabled readers. Brain and Language, 21, 318-334.

- Shute, G.E. and Huertas, V. (1990). Developmental variability of frontal lobe function. Developmental Neuropsychology, 6, 1-11.
- Sidtis, J.J. (1982). Predicting brain organization from dichotic listening performance: Cortical and subcortical functional asymmetries contribute to perceptual asymmetries. Brain and Language, 17, 287-300.
- Slorach, N. and Noehr, B. (1973). Dichotic listening in stuttering and dyslalic children. Cortex, 9, 295-300.
- Smith, A. (1990). Toward a comprehensive theory of stuttering: A commentary. Journal of Speech and Hearing Disorders, 55, 398-401.
- Sommers, R.K., Brady, W.A. and Moore, W.H. (1975). Dichotic ear preferences of stuttering children and adults. Perceptual and Motor Skills, 41, 931-938.
- Spellacy, F. and Blumstein, S. (1970). The influence of language set on ear preference in phoneme recognition. Cortex, 6, 430-439.
- Sperry, R.W. (1974). Lateralized specialization in the surgically separated hemispheres. In F.O. Schmitt and F.G. Worden (Eds.), Neuroscience 3rd Study Program. Cambridge: MIT Press.
- Springer, S.P. and Eisenson, J. (1977). Hemispheric specialization for speech in language-disordered children. Neuropsychologia, 15, 287-293.
- Sternberg, S., Monsell, S., Knoll, R.L. and Wright, C.E. (1978). The latency and duration of rapid movement sequences: Comparisons of speech and typewriting. In G.E. Stelmach (Ed.), Information Processing in Motor Control and Learning. New York: Academic Press.

- Stuss, D. T. and Benson, D.F. (1986). The Frontal Lobes. New York: Raven Press.
- stutterers. Neuropsychologia, 13, 19-26.
- Sussman, H.M. and MacNeilage, P.F. (1975). Hemispheric specialization for speech production and perception in stutterers. Neuropsychologia, 13, 19-26.
- Tanji, J. (1994). The supplementary motor area in the cerebral cortex. Neuroscience Research, 19, 251-268.
- Tanji, J., Okano, K. and Sato, K. (1988). Neuronal activity in cortical motor areas related to ipsilateral, contralateral and bilateral digit movements of the monkey. Journal of Neurophysiology, 60, 325-343.
- Tanji, J., Taniguchi, K. and Saga, T. (1980). Supplementary motor area: Neuronal response to motor instructions. Journal of Neurophysiology, 43, 60-68.
- The Council of Europe (1989). The Use Of the International Classification of Impairments, Disabilities and Handicaps in Rehabilitation.
- Tokuno, H., Kimura, M. and Tanji, J. (1992). Pallidal inputs to thalamocortical neurons projecting to the supplementary motor area: An anterograde and retrograde double labeling study in the macaque monkey. Experimental Brain Research, 90, 635-638.
- Travis, L.E. (1931). Speech Pathology. New York: D. Appleton.
- Travis, L.E. (1934). Dissociation of the homologous muscle function in stuttering. Archives of Neurology and Psychiatry, 31, 127-133.
- Travis, L.E. (1978). The cerebral dominance theory of stuttering: 1931-1978. Journal of Speech and Hearing Research, 43, 278-281.

- Travis, L.E. and Lindsley, D.B. (1933). An action current study of handedness in relation to stuttering. Journal of Experimental Psychology, 16, 258-270.
- Trotter, W.D. and Bergmann, M.F. (1957). Stutterers' and nonstutterers' reactions to speech situations. Journal of Speech and Hearing Disorders, 22, 40-45.
- Van Dusen, C.R. (1939). A laterality study of non-stutterers and stutterers. Journal of Speech Disorders, 4, 261-265.
- Van Lieshout, P.H.H.M., Hulstijn, W. and Peters, H.F.M. (1996). Speech production in people who stutter: Testing the motor plan assembly hypothesis. Journal of Speech and Hearing Research, 39, 76-92.
- Van Riper, C. (1937). The effect of penalty upon frequency of stuttering spasms. Journal of Genetic Psychology, 50, 193-195.
- Van Riper, C. (1982). The Nature of Stuttering. Englewood Cliffs: Prentice Hall.
- Vargha-Khadem, F., O'Gorman, A.M. and Watters, G.V. (1985). Aphasia and handedness in relation to hemispheric side, age at injury, and severity of cerebral lesion during childhood. Brain, 96, 641-646.
- Verfaellie, M., Bowers, D. and Heilman, K.M. (1988). Hemispheric asymmetries in mediating intention, but not selective attention. Neuropsychologia, 26, 521-531.
- Wada, J. and Rasmussen, T. (1960). Intracarotid injection of sodium amytal for the lateralization of cerebral speech dominance: Experimental and clinical observations. Journal of Neurology, 17, 266-282.

- Watkin, K.L. and Fromm, D. (1984). Labial coordination in children. Journal of the Acoustical Society of America, 75, 629-632.
- Watson, B.C. and Alfonso, P.J. (1987). Physiological bases of acoustic LRT in nonstutterers, mild stutterers, and severe stutterers. Journal of Speech and Hearing Research, 30, 434-447.
- Watson, B.C., Freeman, F.J., Devous, M.D., Chapman, S.B., Finitzo, T. and Pool, K.D. (1994). Linguistic performance and regional cerebral blood flow in persons who stutter. Journal of Speech and Hearing Research, 37, 1221-1228.
- Watson, B.C., Pool, K.D., Devous, M.D., Freeman, F.J. and Finitzo, T. (1992). Brain blood flow related to acoustic laryngeal reaction time in adult developmental stutterers. Journal of Speech and Hearing Research, 35, 555-561.
- Webster, R.L. (1980). Evolution of a target-based behavioral therapy for stuttering. Journal of Fluency Disorders, 5, 303-320.
- Webster, W.G. (1985). Neuropsychological mechanisms of stuttering -- I. Representation of sequential response mechanisms. Neuropsychologia, 23, 263-267.
- Webster, W.G. (1986a). Response sequence organization and reproduction by stutterers. Neuropsychologia, 24, 813-821.
- Webster, W.G. (1986b). Neuropsychological models of stuttering -- II. Interhemispheric interference. Neuropsychologia, 24, 737-741.
- Webster, W.G. (1987). Rapid letter transcription performance by stutterers. Neuropsychologia, 25, 845-847.

- Webster, W.G. (1988). Neural mechanisms underlying stuttering: Evidence from bimanual handwriting performance. Brain and Language, 33, 226-244.
- Webster, W.G. (1989). Sequence reproduction deficits in stutterers tested under nonspeeded response conditions. Journal of Fluency Disorders, 14, 79-86.
- Webster, W.G. (1990a). Evidence in bimanual finger tapping of an attentional component to stuttering. Behavioural Brain Research, 37, 93-100.
- Webster, W.G. (1990b). Concurrent cognitive processing and letter sequence transcription deficits in stutterers. Canadian Journal of Psychology, 44, 1-13.
- Webster, W.G. (1990c). "Hurried hands and tangled tongues: Implications of current research for the management of stuttering." In E. Boberg (Ed.), Proceedings of the Third International Banff Conference on Stuttering: The Neuropsychology of Stuttering. Edmonton: University of Alberta Press.
- Webster, W.G. and Poulos, M. (1987). Handedness distribution among adults who stutter. Cortex, 23, 705-708.
- Webster, W.G. and Poulos, M. (1989). Facilitating Fluency: Transfer Strategies for Adult Stuttering Treatment Programs. Tucson: Communication Skill Builders.
- Westby, C.E. (1978). Language performance of stuttering and nonstuttering children. Journal of Communication Disorders, 12, 133-145.
- Wiesendanger, M. (1986). Recent developments in studies of supplementary motor area of primates. Reviews in Physiology, Biochemistry and Pharmacology, 103, 1-59.

- Williams, D.E. (1955). Masseter muscle action current potentials in stuttered and non-stuttered speech. Journal of Speech and Hearing Disorders, 20, 242-261.
- Wingate, M.E. (1964a). Recovery from stuttering. Journal of Speech and Hearing Disorders, 29, 312-321.
- Wingate, M.E. (1964b). A standard definition of stuttering. Journal of Speech and Hearing Disorders, 29, 484-489.
- Wingate, M.E. (1984). Definition is the problem. Journal of Speech and Hearing Disorders, 49, 429-431.
- Witelson, S.F. (1985). The brain connection: The corpus callosum is larger in left-handers. Science, 229, 665-668.
- Witelson, S.F. and Kigar, D.L. (1988). Anatomical development of the corpus callosum in humans: A review with reference to sex and cognition. In D.L. Molfese and S.J. Segalowitz (Eds.), Brain Lateralization in Children: Developmental perspectives. New York: Guilford
- Wohlert, A.B. (1993). Event-related brain potentials preceding speech and nonspeech oral movements of varying complexity. Journal of Speech and Hearing Research, 36, 897-905.
- Wolff, P.H., Gunnoe, C.E. and Cohen, C. (1983). Associated movements as a measure of developmental age. Developmental Medicine and Child Neurology, 25, 417-429.
- Wood, F., Stump, D., McKeehan, A., Sheldon, S., and Proctor, J. (1980). Patterns of regional cerebral blood flow during attempted reading aloud by stutterers both on and

- off haloperidol medication: evidence for inadequate left frontal activation during stuttering. Brain and Language, 9, 141-144.
- Woods, B.T. and Carey, S. (1979). Language deficits after apparent clinical recovery from childhood aphasia. Annals of Neurology, 6, 405-409.
- Woods, B.T. and Teuber, H. (1973). Early onset of complementary specialization of cerebral hemispheres in man. Transactions of the American Neurological Association, 98, 113-117.
- Woolf, G. (1967). The assessment of stuttering as struggle, avoidance and expectancy. British Journal of Disorders of Communication, 2, 158-177.
- World Health Organization (1980). International Classification of Impairments, Disabilities and Handicaps (ICIDH). A Manual of Classification Relating to Consequences of Diseases. Geneva: W.H.O..
- Yairi, E. and Clifton, N.F. (1972). Disfluent speech behavior of preschool children, high school seniors and geriatric persons. Journal of Speech and Hearing Research, 15, 714-719.
- Yairi, E. (1982). Longitudinal studies of disfluencies in two-year-old children. Journal of Speech and Hearing Research, 25, 155-160.
- Yakolev, P.I. and Lecours, A.-R. (1967). The myelogenetic cycles of regional maturation of the brain. In Minkowski, A. (Ed.), Development of the Brain in Early Life. Philadelphia: F.A. Davis and Company.
- Zaidel, E. (1983). Advances and retreats in laterality research. Behavioral and Brain Sciences, 6, 523-528.

- Zatorre, R.J. (1989). Perceptual asymmetry on the dichotic fused words test and cerebral speech lateralization determined by the carotid sodium amytal test. Neuropsychologia, 27, 1207-1209.
- Zimmermann, G.N. (1980). Articulatory dynamics of fluent utterances of stutterers and nonstutterers. Journal of Speech and Hearing Research, 23, 95-107.
- Zimmerman G.N. (1984). Articulatory dynamics of stutterers. In Curlee, R.F. and Perkins, W.H., (Eds.), Nature and Treatment of Stuttering: New Directions. San Diego: College-Hill Press.

APPENDICES

Appendix A: Perceptions of Stuttering Inventory

Appendix A(i): Screenshot of instructions provided in computer-based implementation of Perceptions of Stuttering Inventory.

Standard Version (refers to current speech)

**Perceptions of
Stuttering Inventory**

Name: Joe Participant
Date: February 10, 1996

Directions

Here are sixty statements about stuttering. Some of these may be characteristic of your stuttering. Read each item carefully and respond as in the example below.

**Characteristic
of me**
☐ Repeating sounds

Put an 'x' in the box under "Characteristic of me" (move the cursor to the box and click the mouse button) if 'repeating sounds' is part of your stuttering, if it is **not characteristic**, leave the box blank.

"Characteristic of me" refers only to what you do **now**, not to what was true of your stuttering in the past and which you no longer do, and not what you think you should or should not be doing. Even if the behaviour described occurs only occasionally or only in some speaking situations, if you regard it as characteristic of your stuttering, check the box under "Characteristic of me." Be accurate in your judgements.

⏶

Begin

Appendix A (ii): PSI-modified: Retrospective (Childhood) Version

**Perceptions of
Stuttering Inventory****Name.** Joe Participant**Date** February 10, 1996**Directions**

Here are sixty statements about stuttering. Some of these may have been characteristic of **your** childhood stuttering. Read each item carefully and respond as in the example below.

**Characteristic
of me**

☐ Repeating sounds

Put an 'x' in the box under "**Characteristic of me**" (move the cursor to the box and click the mouse button) if 'repeating sounds' was part of your childhood stuttering, if it was **not** characteristic, leave the box blank.

"**Characteristic of me**" refers here to what you did **as a child**, not to what is only true of your speech now, and not what you think you should or should not have been doing. Even if the behaviour described occurred only occasionally or only in some speaking situations, if you regard it as characteristic of your childhood stuttering, check the box under "**Characteristic of me.**" Be accurate in your judgements.

Begin

Appendix A(ii): Sample screenshot of questionnaire items on computer-based implementation of Perceptions of Stuttering Inventory.

Characteristic of me	Joe Participant	Page: 1
☒ 1. Avoiding talking to people in authority (e.g., a teacher, employer, or clergyman)		
☒ 2. Feeling that interruptions in your speech (e.g., pauses, hesitations, or repetitions) will lead to stuttering		
☐ 3. Making the pitch of your voice higher or lower when you expect to get "stuck" on words		
☐ 4. Having extra and unnecessary facial movements (e.g., flaring your nostrils during speech attempts)		
☐ 5. Using gestures as a substitute for speaking (e.g., nodding your head instead of saying "yes" or smiling to acknowledge a greeting).		
☒ 6. Avoiding asking for information (e.g., asking for directions or inquiring about a train schedule)		
☐ 7. Whispering words to yourself before saying them or practicing what you are planning to say long before you speak.		
☒ 8. Choosing a job or hobby because little speaking would be required.		
☒ 9. Adding an extra and unnecessary sound, word, or phrase to your speech (e.g., "uh," "well," or "let me see") to help yourself get started		
☒ 10. Replying briefly using the fewest words possible		
Prev Page	Done	Next Page

Appendix A(iii): List of all items included in Perceptions of Stuttering Inventory.

Avoiding talking to people in authority (e.g., a teacher, employer, or clergyman).

Feeling that interruptions in your speech (e.g., pauses, hesitations, or repetitions) will lead to stuttering.

Making the pitch of your voice higher or lower when you expect to get "stuck" on words.

Having extra and unnecessary facial movements (e.g., flaring your nostrils during speech attempts).

Using gestures as a substitute for speaking (e.g., nodding your head instead of saying "yes" or smiling to acknowledge a greeting).

Avoiding asking for information (e.g., asking for directions or inquiring about a train schedule).

Whispering words to yourself before saying them or practicing what you are planning to say long before you speak.

Choosing a job or hobby because little speaking would be required.

Adding an extra and unnecessary sound, word, or phrase to your speech (e.g., "uh," "well," or "let me see") to help yourself get started.

Replying briefly using the fewest words possible.

Making Sudden Jerky or forceful movements with your head, arms, or body during speech attempts (e.g., clenching your fist, jerking your head to one side).

Repeating a sound or word with effort.

Acting in a manner intended to keep you out of a conversation or discussion (e.g., being a good listener, pretending not to hear what was said, acting bored, or pretending to be in deep thought).

Avoiding making a purchase (e.g., going into a store or buying stamps in the post office).

Breathing noisily or with great effort while trying to speak.

Making your voice louder or softer when stuttering is expected.

Prolonging a sound or word (e.g., m-m-m-m-my) while trying to push it out.

Helping yourself to get started talking by laughing, coughing, clearing your throat, gesturing, or some other body activity or movement.

Having general body tension during speech attempts (e.g., shaking, trembling, or feeling "knotted up" inside).

Paying particular attention to what you are going to say (e.g., the length of a word, or the position of a word in a sentence).

Feeling your face getting warm and red (as if you are blushing), as you are struggling to speak.

Saying words or phrases with force or effort.

Repeating a word or phrase preceding the word on which stuttering is expected.

Speaking so that no word or sound stands out (e.g., speaking in a singsong voice or in a monotone).

Avoiding making new acquaintances (e.g., not visiting with friends, not dating, or not joining social, civic, or church groups).

Making unusual noises with your teeth during speech attempts (e.g., grinding or clicking your teeth).

Avoiding introducing yourself, giving your name, or making introductions.

Expecting that certain sounds, letters, or words are going to be particularly "hard" to say (e.g., words beginning with the letter "s").

Giving excuses to avoid talking (e.g., pretending to be tired or pretending lack of interest in a topic).

"Running out of breath" while speaking.

Forcing out sounds.

Feeling that your fluent periods are unusual, that they cannot last, and that sooner or later you will stutter.

Concentrating on relaxing or not being tense before speaking.

Substituting a different word or phrase for the one you had intended to say.

Prolonging or emphasizing the sound preceding the one on which stuttering is expected.

Avoiding speaking before an audience.

Straining to talk without being able to make a sound.

Coordinating or timing your speech with a rhythmic movement (e.g., tapping your foot or swinging your arm).

Rearranging what you had planned to say to avoid a "hard" sound or word.

"Putting on an act" when speaking (e.g., adopting an attitude of confidence or pretending to be angry).

Avoiding the use of the telephone.

Making forceful and strained movements with your lips, tongue, jaw, or throat (e.g., moving your jaw in an uncoordinated manner).

Omitting a word, part of a word, or a phrase which you had planned to say (e.g., words with certain sounds or letters).

Making "uncontrollable" sounds while struggling to say a word.

Adopting a foreign accent, assuming a regional dialect, or imitating another person's speech.

Perspiring much more than usual while speaking (e.g., feeling the palms of your hands getting clammy).

Postponing speaking for a short time until certain you can be fluent (e.g., pausing before "hard" words).

Having extra and unnecessary eye movements while speaking (e.g., blinking your eyes or shutting your eyes tightly).

Breathing forcefully while struggling to speak.

Avoiding talking to others of your own age group (your own or the opposite sex).

Giving up the speech attempt completely after getting "stuck" or if stuttering is anticipated.

Straining the muscles of your chest or abdomen during speech attempts.

Wondering whether you will stutter or how you will speak if you do stutter.

Holding your lips, tongue, or jaw in a rigid position before speaking or when getting "stuck" on a word.

Avoiding talking to one or both of your parents.

Having another person speak for you in a difficult situation (e.g., having someone make a telephone call for you or order for you in a restaurant).

Holding your breath before speaking.

Saying words slowly or rapidly preceding the word on which stuttering is expected.

Concentrating on how you are going to speak (e.g., thinking about where to put your tongue or how to breathe).

Using your stuttering as the reason to avoid a speaking activity.

Appendix B: Edinburgh Handedness Inventory

Appendix B (I): Screenshots of Response Screen for Edinburgh Handedness Inventory.

Edinburgh Handedness Inventory (modified)

Name Joe Participant Testing Date February 10, 1996

		Left	Right
1	Writing		++
2	Drawing		++
3	Throwing		++
4	Scissors		++
5	Toothbrush		++
6	Comb		+
7	Hammer		++
8	Screwdriver		++
9	Knife (without fork)		++
10	Spoon		++
11	Push Broom (upper hand)	+	
12	Striking Match (match)		++
13	Opening Box (lid)		+
14	Holding a Racquet		++
15	Unscrewing Lid of Jar (lid)		+
1	Which foot do you prefer to kick with?		++
11	Which eye do you use when using only one?	+	

Instructions
Done

Appendix B(ii). Instructions (appear in separate window when "Instructions" button pressed; also given verbally).

PLEASE READ THESE INSTRUCTIONS CAREFULLY BEFORE STARTING

Please indicate your preference in the use of hands in the following activities by putting + in the appropriate column.

Where the preference is so strong that you would never try to use the other hand unless absolutely forced to, put ++.

If in any case you are really indifferent as to which hand you use, put + in both columns.

Some of the activities require both hands. In these cases the part of the task, or object, for which hand preference is wanted is indicated in brackets.

Please try to answer all the questions, and only leave a blank if you have no experience at all with the object or task.

Appendix C: Likert Stuttering Scales.

Each question appeared centered on a single page, and each participant received two versions (adult and child), though the wording of the questions varied depending on the group (stutterer or exstutterer).

Please place an X on this scale to indicate how severe you feel your stuttering to be.

/___/___/___/___/___/___/___/

very mild

very severe

Adulthood version: exstutterers

If you stutter at all now (e.g. when you are tired or under stress) please place an X on this scale to indicate how severe you feel your stuttering can be at its worst.

/___/___/___/___/___/___/___/

not at all

very severe

Childhood version: both groups

Please think back to your childhood and place an X on this scale to indicate how severe you feel your stuttering was when you were a child.

/___/___/___/___/___/___/___/

very mild

very severe

Appendix D: Interview Database.

Screenshot of database used by experimenter to record information from interview.

Database automatically receives scored data from the Edinburgh Handedness Inventory (LQ/O and LQ/SL) and from the PSI (St., Ex. and Av.).

Subject ID: 29 C/b. February 10, 1996

Name Group Excl

Sex: Age Handedness FH:

	Ponton	RC	St	Ch	Ad
1. John Doe				8	6
2. Jane Smith	R/T	8/8	Ex	9	7
3. Joe Participant	LQ/O	100	Av:	9	7
4. Dave F.	LQ/SL	85	T:	26	20

Onset Remission Factors

Sev-A Sev-C

Save Record **New** **Comments**

factors (onset, recovery) entered here, as well as other information from interview (where recruited, duration and kind of therapy, etc)

PSI **Handedness**

Appendix E: Consent Forms

Appendix E(i). Consent Form for Stutterers

Appendix E(ii). Consent Form for Exstutterers

Appendix E(iii). Consent Form for Nonstutterers

Note: Spacing between lines on consent forms presented here has been condensed to allow forms to fit on one page each.

Department of Psychology
Stuttering Research Laboratory

ST

Experiment: Motor and Visual Processes in People Who Stutter

In this study you will be asked to perform 4 different tasks. One of the tasks will involve tapping with your fingers in response to visually presented sequences. One will involve using your two hands to turn two cranks which will manipulate a "pen" on a computer screen in a manner similar to the "Etch-a-Sketch" toy. Two tasks will involve making decisions about visual stimuli flashed very briefly on a computer screen. All of these tasks are intended to be challenging, and it is expected that all participants will make errors on all of the tasks.

In addition to performing those motor tasks, you will be asked to fill out a short questionnaire about your stuttering. You will also be given the opportunity to describe your stuttering in your own words.

Principle Investigator: David Forster

Project Supervisor: Prof. W.G. Webster, Faculty of Social Sciences, Brock University, St. Catharines, ON. Phone (905) 688-5550, ext. 3426

Project Co-Supervisor: Prof. J.F. Campbell, Department of Psychology, Carleton University, Ottawa, ON. Phone (613) 788-2600, ext. 2672.

Name of Participant (print):-----
Participant's Phone Number: -----

Please read the following and sign below to indicate that you understand your rights as a voluntary participant in this study.

I understand that my participation in this experiment is entirely voluntary and that I may withdraw from the study at any time and for any reason without penalty.

I understand that my individual data in this experiment will be strictly anonymous and I will not be identified in any publications of the results.

I understand that I may direct any concerns about the research to Prof. J.F. Campbell (Project Co-Supervisor, 788-2600, ext. 2672), Prof. L. Paquet (Chairman of Ethics Committee, ext. 2692) or Prof. B. Jones (Chairman of Psychology Department, ext. 2648) at the Department of Psychology, Carleton University.

I understand that, in return for my participation in this study, I will receive either appropriate experimental credit towards Psychology 49.100 at Carleton University, or a cash honorarium of \$40.

 (signature of participant)

 (date)

Department of Psychology

ES

Stuttering Research Laboratory**Experiment: Motor and Visual Processes in People Who Stutter**

In this study you will be asked to perform 4 different tasks. One of the tasks will involve tapping with your fingers in response to visually presented sequences. One will involve using your two hands to turn two cranks which will manipulate a "pen" on a computer screen in a manner similar to the "Etch-a-Sketch" toy. Two tasks will involve making decisions about visual stimuli flashed very briefly on a computer screen. All of these tasks are intended to be challenging, and it is expected that all participants will make errors on all of the tasks.

In addition to performing those motor tasks, you will be given a short questionnaire in which you will be asked to recall various aspects of your childhood stuttering. You will also be given the opportunity to describe your stuttering as you remember it in your own words.

Principle Investigator: David Forster

Project Supervisor: Prof. W.G. Webster, Faculty of Social Sciences, Brock University, St. Catharines, ON. Phone (905) 688-5550, ext. 3426

Project Co-Supervisor: Prof. J.F. Campbell, Department of Psychology, Carleton University, Ottawa, ON. Phone (613) 788-2600, ext. 2672.

Name of Participant (print): -----
Participant's Phone Number: -----

Please read the following and sign below to indicate that you understand your rights as a voluntary participant in this study.

I understand that my participation in this experiment is entirely voluntary and that I may withdraw from the study at any time and for any reason without penalty.

I understand that my individual data in this experiment will be strictly anonymous and I will not be identified in any publications of the results.

I understand that I may direct any concerns about the research to Prof. J.F. Campbell (Project Co-Supervisor, 788-2600, ext. 2672), Prof. L. Paquet (Chairman of Ethics Committee, ext. 2692) or Prof. B. Jones (Chairman of Psychology Department, ext. 2648) at the Department of Psychology, Carleton University.

I understand that, in return for my participation in this study, I will receive either appropriate experimental credit towards Psychology 49.100 at Carleton University, or a cash honorarium of \$40.

 (signature of participant)

 (date)

Department of Psychology
Stuttering Research Laboratory

Informed Consent Form

Experiment: Motor and Visual Processes in People Who Stutter

In this study you will be asked to perform 4 different tasks. One of the tasks will involve tapping with your fingers in response to visually presented sequences. One will involve using your two hands to turn two cranks which will manipulate a "pen" on a computer screen in a manner similar to the "Etch-a-Sketch" toy. Two tasks will involve making decisions about visual stimuli flashed very briefly on a computer screen. All of these tasks are intended to be challenging, and it is expected that all participants will make errors on all of the tasks.

Principle Investigator: David Forster

Project Supervisor: Prof. W.G. Webster, Faculty of Social Sciences, Brock University, St. Catharines, ON. Phone (905) 688-5550, ext. 3426

Project Co-Supervisor: Prof. J.F. Campbell, Department of Psychology, Carleton University, Ottawa, ON. Phone (613) 788-2600, ext. 2672.

Name of Participant (print): -----
Participant's Phone Number: -----

Please read the following and sign below to indicate that you understand your rights as a voluntary participant in this study.

I understand that my participation in this experiment is entirely voluntary and that I may withdraw from the study at any time and for any reason without penalty.

I understand that my individual data in this experiment will be strictly anonymous and I will not be identified in any publications of the results.

I understand that I may direct any concerns about the research to Prof. J.F. Campbell (Project Co-Supervisor, 788-2600, ext. 2672), Prof. L. Paquet (Chairman of Ethics Committee, ext. 2692) or Prof. B. Jones (Chairman of Psychology Department, ext. 2648) at the Department of Psychology, Carleton University.

I understand that, in return for my participation in this study, I will receive either appropriate experimental credit towards Psychology 49.100 at Carleton University, or a cash honorarium of \$40.

(signature of participant)

(date)

Appendix F: Debriefing Form

Carleton University
Department of Psychology

Brain Mechanisms in Recovered and Persistent Stuttering

Thank-you very much for volunteering your time to participate in this experiment. The experiment is part of an on-going program of research in which we are attempting to use simple behavioural techniques to improve our understanding of the neurological mechanisms that underlie stuttering. Specifically, we are interested here in testing some hypotheses we have about stuttering and recovery from stuttering in childhood. Approximately 3/4 of all children who stutter will spontaneously recover from stuttering before reaching adulthood. We are attempting to address the issue of what underlies that recovery by comparing the performance of adults who stutter with adults who stuttered as children but no longer do. This is referred to as a "retrospective" design, and is necessary because we are currently unable to predict which children who stutter will ultimately go on to recover.

Based upon past research in this and other laboratories, we have identified two "neurological" factors that appear to be involved in stuttering. One is a very mild difficulty in the timing or sequencing of complex sequential movements. The other is an unusual pattern relating to the balance of activity between the two cerebral hemispheres; adults who stutter appear to have a less stable pattern of activity with a tendency for one or the other hemisphere to become over-activated relative to the other. We believe that either of these two factors would be insufficient to cause stuttering on its own, and that the individual is likely to stutter only when both occur together.

We have further speculated that one of the two conditions, the mild difficulty in sequencing complex movements, may exist in *all* children as a part of the normal course of development. Thus, any person with the unusual pattern of activity between the two cerebral hemispheres might be expected to stutter as a child, because both conditions would be met. However, as his motor system developed toward maturity, his stuttering would improve. In some of these people, the motor system may never reach the typical adult state. They may continue to have a subtle difficulty in sequencing complex movements. These are the people who would be expected to continue stuttering into adulthood.

Each of the four tasks you performed were designed to test one of the two factors we believe to be associated with stuttering. The tapping task was designed to assess motor sequencing. The crank-turning task is intended to indirectly assess the same brain mechanisms that we believe underlie the difficulty in motor sequencing. The two tasks that involved responding to stimuli flashed on the computer are intended to assess patterns of cerebral activity. By flashing a stimulus quickly to the right or left while you stare at the center, we can send the information directly to one of your cerebral hemispheres. By comparing performance with and without the "precue" (the dot over the words or the frame around the dots) we can assess overall patterns of activity between your two cerebral hemispheres.

We expect to find that adults who stutter will demonstrate both conditions outlined above: a *mild* difficulty in motor sequencing, and an unusual pattern of cerebral activity. Those who stuttered as children but no longer do should have normal motor performance, but may demonstrate the unusual pattern of cerebral activity. Fluent speakers with no past history of stuttering will provide the "normal" baseline on all four tasks.

People who stutter are *not* grossly uncoordinated. The difficulties described here require sensitive tasks to reveal themselves, and are probably not relevant outside of the speech domain in normal daily life.

In summary, if we do not get the pattern of results that we have predicted, it is not necessarily a bad thing. Either our results will support our hypotheses and our model of stuttering, or they will force us to modify them. Either way we will have learned something valuable, and will hopefully have moved closer to a good understanding of the mechanisms that underlie stuttering.

If you have any questions or concerns about the study you may contact us to discuss them or you may direct them to Prof. J.F. Campbell (Project Co-Supervisor, 788-2600, ext. 2672), Prof. L. Paquet (Chairman of Ethics Committee, ext. 2692) or Prof. B. Jones (Chairman of Psychology Department, ext. 2648) at the Department of Psychology, Carleton University.

Thanks again for your participation.

Dave Forster (788-2600, extn. 8211)

William G. Webster ((905) 688-5550, ext. 3426)

Note: There is an excellent resource available in the Ottawa area for people who stutter. If you stutter or know someone who stutters, you (or they) are encouraged to contact:

Carol Bock,
Coordinator, Stuttering Treatment Program
Communication Disorders Department
Royal Ottawa Rehabilitation Centre
505 Smyth Road
Ottawa, ON
K1H 8M2

Phone: 737-7350, extn. 302

Appendix G: Protocol

ORDER OF INTERVIEW

All fields up to severity

PSI First Administration

Adulthood first for NS, ST (printed instructions)

Childhood first for ES

•I want you to think back to your childhood, and particularly to your speech when you were a child. I am going to ask you to go through the following questionnaire, and to answer it with respect to your speech when you were a child.

PSI Second Administration

For NS, ST

•I want you to think back to your childhood, and particularly to your speech when you were a child. I am going to ask you to go through the PSI a second time, but this time answer it with respect to when you were a child.

For ES

•Now I want you to think about your speech as it is now. I am going to ask you to go through the PSI a second time, but this time answer it with respect to your speech as it has been during your adulthood.

Below for exclusionary criteria, general information

Add any positive answers to comments field

Speech Therapy (ST and ES)

Speech therapy

Recency – how recently, if at all, have you received formal speech therapy

Intensity – was the therapy intensive (i.e. all day for at least several weeks, or was it part time (i.e. a weekly appointment)

Neurological/birth

- Do you suffer from any neurological impairments such as epilepsy or migraine headaches?
- Are you aware of there having been anything exceptional about your birth, such as being born premature, jaundiced, having breathing difficulties or being a blue baby?
- Were you hospitalized longer than usual following your birth?

Learning Disabilities

- Do you believe that you have or have ever had a learning disability or attentional disorder?
- Have you ever been diagnosed by a qualified professional as having a learning disability or attentional disorder?

Music

- Do you play any musical instruments?
- how long?
- formal training

Education

- What is your highest level of education obtained?
- What is your current occupation?

Recruitment

- How did you get involved in this research

TURN OFF LIGHTS, AIM DESK LAMP AT WALL

PUSH MONITOR ALL THE WAY BACK

Lexical Decision Task

In this task you will be making decisions about stimuli flashed rapidly to the computer screen. More specifically, three-letter words* will be flashed for 117 ms to either the left of the right of a fixation point at which you will be staring. You will be asked to indicate by tapping with either your right or left index finger whether the stimulus represents a real English word* or a nonsense word (i.e. not a real word*).

Rest your hands on the Plexiglas platform such that your two index fingers are on the two side by side buttons, and your two thumbs are both resting on the long narrow button at the bottom. Pressing the right (left) button will indicate that the stimulus you saw was a real word*. Pressing the left (right) button will indicate that the stimulus was not a real word. The long button at the bottom (we will call it a "spacebar") will be used to initiate each trial. The button centered at the top of the platform will only be used if any trials need to be repeated.

Each trial will begin with a dialog box appearing in the middle of the screen telling you to "Press Spacebar to Continue." You will then press the spacebar with both of your thumbs simultaneously. The dialog box will disappear and a small "-" will appear in the center of the screen. After a brief but variable period of time (between 1 and 2s), the "-" will change into an "=". You must respond to this change by pressing the spacebar a second time, once again with both thumbs simultaneously. You only have 500 ms to make this response or the trial will be repeated. The purpose of this sequence is to ensure that your eyes are fixated on the center of the screen.

Immediately after you press the spacebar the second time (in response to the "="), the word will be flashed either to the right or the left of the location of the "=". The word will also be accompanied by a dot* which will be located just above the word. The purpose of the dot* is simply to help attract your attention to the word.

When the stimulus is flashed, you must decide as quickly as possible without sacrificing accuracy, whether it was a word or non-word. As mentioned, you indicate your response either by pressing the button under your right (left) index finger to indicate a word, or by pressing the button under your left index finger to indicate a non-word. Press relatively hard on the response button - don't be shy, or it might not register your response.

You will know when it has registered your response by a tone that will sound when you press the button.

It is important to note that your responses are being timed: if you are unsure on a given trial, you should just go with your first instinct rather than contemplating what you saw. Most of your responses will take between half a second and 1.5 s to make, so if you spend several seconds contemplating a given trial, your overall data may be substantially influenced by that trial. Just use your instinct – it is usually right. If you have no idea (i.e. you couldn't read the word), then just guess.

With the above in mind, you should still give accuracy more importance than speed. Do not unduly rush your responses in order to have short response times.

We will begin by having you perform 16 practice trials. Begin by aligning your chair so that the middle of your body is in line with the middle of the response apparatus and the screen. You should sit up straight as well, and try not to move your chair during the session.

(PRACTISE TRIALS)

You will now perform 160 real trials. This should take between 20 and 25 minutes. If you get tired during this period, just stop and rest for a moment (i.e. don't press the spacebar to initiate the next trial. You may then simply continue from where you left off.

Any questions?

Sequence Reproduction Task

PUSH BOX AND MONITOR ALL THE WAY BACK

In this task, you will be tapping sequences with your fingers on the set of four telegraph keys you see in front of you. The sequence you tap will be different on each trial, and will be presented to you visually via a dot that will jump between four fingers that will be graphically illustrated on the computer screen. The sequences will vary in length from three to five elements. You will be asked to both initiate the sequence as quickly after presentation as you can, and to continue tapping the sequence over and over as quickly and as accurately as you can for five seconds.

Rest the four fingers (excluding your thumb) of your right hand on the four telegraph keys inside the box. I will close the lid so you can't see your hand. Move the box and your chair so that the box is directly in front of the screen and your hand is resting as comfortably as possible on the keys. Hold this switch in your left hand.

Each trial will begin with a dialog box telling you to press the hand switch to continue. After you press the switch, another box will briefly display the trial number, and this will be followed by a line drawing of a hand. A black dot, appearing over the fingertip, will jump back in forth between the fingers indicating the sequence you will be required to tap. The sequence will involve from three to five elements, meaning that one finger may be repeated in the sequence. Immediately following the last element in the sequence, a tone will sound. When you hear this tone, begin tapping the sequence as illustrated. Begin as soon as you can after hearing the tone, and continue to tap the sequence over and over as quickly and as accurately as you can until you hear a second tone, five seconds later.

Once again, it is important to note that your performance is being assessed in terms of both speed and accuracy. Neither is more important here - try to tap both as quickly and as accurately as you can.

If you do not see the sequence for some reason, do not tap anything for that trial. Five seconds after the tone, you will be given the opportunity to repeat the trial. If you need to repeat a trial that you did begin tapping (for example if your fingers were on the wrong keys) then do not initiate the next trial. Tell me, and I will reset the trial.

We will begin with four practice trials. Start whenever you are ready.

(PRACTISE TRIALS)

You will now perform 36 trials in two blocks of 18. This should take around 15 minutes. Once again, if you get fatigued, you may stop and rest your fingers and eyes between trials.

Any questions?

Bimanual Crank-Turning

This task will involve turning the two cranks at the same time to manipulate a dot on the computer screen. At the beginning of a trial, you will see two parallel lines on the screen that will represent a track going up and to the right. There will be a dot at the bottom of the track, representing a pen that you will manipulate with the cranks. The cranks move the pen in the same way an "Etch-a-Sketch" toy works. The left crank moves the pen up and down, with clockwise motion causing upward displacement. The right crank moves the pen left and right, with clockwise movement causing rightward displacement.

Your task will be to move the pen through centre of the track as quickly as possible without going outside of the track. Try to keep both cranks turning at the same time, as opposed to alternating between them in a stair-like fashion. Try to go in as straight a line as possible through the centre of the track, and try to generate as smooth a trace as possible.

The track will appear in one of two different angles on any given trial. The two different angles were chosen such that you will have to turn one crank twice as fast as the other in order to maintain optimal performance. Also, in all trials, because the track always goes up and to the right, you will be turning both cranks in a clockwise fashion.

At the beginning of each trial, I will say "Ready?" and if you say yes, the track will appear on the screen. The appearance of the tracks will signal the beginning of the trial, but your performance won't be monitored until you begin turning the cranks. In fact, your performance won't be monitored until you move the pen into the track. The trial ends when the pen goes out the other end of the track.

If you go outside of the track, the trial will be repeated. You will perform sixteen trials in total, eight with each track orientation.

Dot Enumeration Task

This task will be very similar to the first task you performed. However, this time the stimuli will be different and the decision you make will be different. The stimulus that will be flashed will be a random pattern of dots, ranging in number from 3 to 6 dots, no more and no less. The dots will be enclosed inside a 2 cm square frame. Your task will be to count the dots and decide if the number of dots was even, 4 or 6 dots, or odd, 3 or 5 dots. Otherwise, the task is identical to the first one. The minus sign and equal sign will appear and you will have to react in the same way by tapping the spacebar.

Once again, if you get tired, you may stop and rest your eyes at any point.

We'll start with 16 practice trials. Any questions?

----- POST TESTING -----

- Verbal Debriefing

- Honarium Receipt

Thank you very much for your time and assistance.

Appendix H: Tapping Sequences for Sequence Reproduction Task

<u>Length = 3</u>	<u>Length = 4</u>	<u>Length = 5</u>
1-2-3	1-2-3-4	1-2-3-4-3
1-2-4	1-2-4-3	1-2-4-3-4
1-3-2	1-3-2-4	1-3-2-4-3
1-3-4	1-3-4-2	1-3-4-2-4
1-4-2	1-4-2-3	1-4-2-3-3
1-4-3	1-4-3-2	1-4-3-2-4
2-1-3	2-1-3-4	2-1-3-4-3
2-1-4	2-1-4-3	2-1-4-3-4
2-3-1	2-3-1-4	2-3-1-4-3
2-3-4	2-3-4-1	2-3-4-1-4
2-4-1	2-4-1-3	2-4-1-3-3
2-4-3	2-4-3-1	2-4-3-1-4

1 = index finger
 2 = middle finger
 3 = ring finger
 4 = little finger

Appendix I: Stimulus Set for Lexical Decision Task**Words****Low Frequency, Low Imagery**

YE, FIR, GIG, ION, MAR, RAP, RYE, SEW, SOP, VOW

Low Frequency, High Imagery

APE, EEL, GEM, HOP, LAD, NUN, PEG, RAM, RUM, TOY

High Frequency, Low Imagery

AGE, BAD, BUY, CUT, JOB, LED, LOW, SIN, TAX, WIN

High Frequency, High Imagery

ARM, BAY, BOY, EAT, FAT, GUN, KEY, LEG, RED, SEA

Non-Words

ACK, DAK, DAS, DEA, DOI, EDO, EGA, EPE, EWL, GED

GIR, GOX, ILA, JAS, LEL, LUN, MYM, NAD, NAS, NID

NUG, OIN, ORN, OYD, POB, REE, RIN, RYL, SRY, TAS

TEP, TUL, VUX, WAW, WEP, WIB, WOX, YEB, YIB, YOD

END

1 | 9 - 1 1 - 9 6

FIN