Acquired Aphasia

Edited by MARTHA TAYLOR SARNO

Third Edition

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THIRD EDITION

Edited by

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New York, New York

ACADEMIC PRESS
San Diego London Boston
New York Sydney Tokyo Toronto

This book is printed on acid-free paper. ♾

Academic Press
a division of Harcourt Brace & Company
525 B Street, Suite 1900, San Diego, California 92101-4495, USA
http://www.apnet.com

Academic Press Limited
24-28 Oval Road, London NW1 7DX, UK
http://www.hbuk.co.uk/ap/

Library of Congress Catalog Card Number: 98-84822

International Standard Book Number: 0-12-619322-3

PRINTED IN THE UNITED STATES OF AMERICA
98 99 00 01 02 03 BB 9 8 7 6 5 4 3 2 1

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Preface to the Third Edition

Since the publication of the second edition, the knowledge base and perspective of many of the disciplines represented in *Acquired Aphasia* have experienced important changes. New research findings and clinical experience have led to conceptual revisions and made it necessary for some of the contributing authors to update and rewrite significant portions of their chapters.

A number of new authors have been added to the roster of contributors: Steven W. Anderson, Sandra Bond Chapman, Lisa Tabor Connor, Guido Gainotti, Roberta E. Gallagher, Guila Glosser, Marjorie Nicholas, and Catya von Karolyi. In addition, two new chapters have been included, one by Michel Paradis, addressing aphasia in the bilingual person, and one by Joan Payne, discussing other multicultural issues. These two chapters reflect some of the contemporary cultural, demographic changes in the United States population and their potential impact on the study of aphasia, its assessment, and its management.

Once again, the need to bring together specialists from many fields as contributors to a comprehensive discussion of aphasia is apparent. Unfortunately, the overall size of the text was restricted by publishing constraints, which limited the amount of detail authors were able to include in their contributions. The cumulative aphasia literature published since the first edition of *Acquired Aphasia* would require additional volumes to address the topic in depth. The volume of research and clinical activity coupled with new technology in the years to come will no doubt continue to add to the breadth of understanding of the underlying mechanisms that shape the manifestations of aphasia in all stages of its evolution.

NEW YORK
1998

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Preface to the Second Edition

In preparation for the second edition of *Acquired Aphasia* each author was challenged to keep the original historical context while updating and adding new concepts and research results developed in the past decade. In some instances this was not possible. A case in point are the chapters by Rita Sloan Berndt and Alfonso Caramazza, who coauthored a chapter on lexical-semantic aspects of aphasia in the first edition but who have written separate chapters in this edition on sentence processing and lexical deficits respectively, reflecting the extraordinary amount of new work that has emerged in these areas.

Several authors have been added. Rhoda Au, along with Loraine Obler and Martin Albert, revised the chapter on language in the elderly aphasic. Alexandra Rehak joined Howard Gardner and Ellen Winner on artistry in aphasia, and Brenda Rapp collaborated with Alfonso Caramazza on the lexical deficits chapter. An entirely new chapter on acquired aphasia in children was contributed by Dorothy Aram. Regrettably, Harold Goodglass did not participate in the revision of his original chapter with Edith Kaplan. The chapter on auditory comprehension is not included in this edition since it was not possible for the author, Karen Riedel, to do a complete revision at this time.

I am indebted to Antonia Buonaguro and John Sarno for their assistance and support in reviewing the revised volume and to Laureen VanOudenrode for her secretarial help.

NEW YORK
1991

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Preface to the First Edition

During the past two decades, aphasia has been a subject of increasing interest to a variety of disciplines beyond those of speech pathology, medicine, and psychology. Once the exclusive province of the neurologist, to whom we owe a great debt for pioneer work, aphasia is now studied as well by speech and language pathologists, linguists, cognitive psychologists, neuropsychologists, and others. The burgeoning of clinical and academic activity is the primary impetus for this volume, whose purpose it is to provide an authoritative text and reference book for graduate students, clinicians, and research workers in the many fields now concerned with aphasia.

The contents and roster of contributors reflect the view that no single discipline or author can be expert in all of the areas that contribute to our knowledge of aphasia. It is the editor's intent, therefore, to provide to the student of aphasia a comprehensive, almost exhaustive text by bringing together the writing of some of the most prolific and knowledgeable workers in the field of aphasia. Many of the contributors are pioneers in the areas of their special interest. The result is that all aspects of aphasia are treated in depth; indeed, each chapter is a review of the subject under discussion. The fact that the authors come from a diversity of disciplines adds to the richness and authority of the text.

The breadth of the book may also be judged by the fact that there are chapters on artistry and aphasia, aphasia in children, in closed-head injury, and in the elderly, as well as the emotional aspects of aphasia. Topics relevant to a comprehensive understanding of the disorder are included. "Trendy" subjects have been avoided. A textbook cannot be all things to all people, but the experience and sophistication of the authors of this volume have resulted in a work of broad applicability. Issues and problems, diversity and controversy are liberally represented.

Though the physiology of communication in health and disease remains an enigma at the most basic level, there is a large body of information on the subject reflecting a century of hard work and creative energy. It is another purpose of this volume to provide the student a distillation of that great effort and a thorough review of our knowledge to date.

The volume is logically organized for courses in aphasia. Chapters on the history, anatomy, and nature of aphasia set the stage for reviews of other dimensions of the disorder (e.g., phonology, auditory comprehension, etc.). The "special" chapters alluded to above add to the completeness and uniqueness of the work and reflect the fact that certain populations require different insights and management.

It should be noted that the scope of this volume does not permit a review of the techniques and practice of aphasia therapy. Broad principles have been outlined, but to cover that subject in detail would require another textbook equal to this in length. Aphasia therapy cannot be conducted by formula; it is a process that grows out of the specific needs of the patient and the knowledge and imagination of the therapist.

I am deeply grateful to the authors whose chapters comprise this book. My special thanks to Hanna Damasio for the anatomical illustrations which she created for her chapter, to the patients and staff of the Speech Pathology Service of the Institute of Rehabilitation and Medicine, to Karen Riedel, Eric Levita, and Margaret Naeser for their invaluable help, and to Rae Dorin and Esther Toledo for typing assistance. I am indebted to my father, Abril Lamarque, for his guidance in the overall graphic design, and to Antonia Buonaguro for her dedicated help with all stages of the project. I am especially grateful to my husband, John, for pointing up the need for such a text and for his support, encouragement, and incisive criticism.

NEW YORK
1981

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Aphasia: Historical Perspectives

ARTHUR BENTON and STEVEN W. ANDERSON

The origins of aphasic disorder no doubt go back to the distant past. The association between disturbed speech and traumatic head injury must have been quite familiar to all primitive people who enjoyed the gift of speech. In any case, references to speechlessness as a sign or form of disease can be found in the earliest medical writings, for example, in the Edwin Smith Surgical Papyrus, an Egyptian manuscript that dates back to 1700 B.C. and that is believed to be a copy of a still older manuscript (Breasted, 1930; Finger, 1994).

Early Contributions

Aphasia in Greek Medicine

The Hippocratic writings (ca. 400 B.C.) include many descriptions of speech disturbances, usually within the setting of protracted and often fatal illness. However, it is not clear exactly what was meant by such terms as *aphōnos* and *anaudos;* translators have given them various meanings depending on their context. Thus, whether distinctions were made between aphasia, dysarthria, muteness, and aphonia is not known. A passage in the Coan Prognosis (No. 353 in the translation of Chadwick & Mann, 1950) associates speechlessness following convulsions "with paralysis of the tongue, or of the arm and right side of the body" (p. 248). Another passage in the Coan Prognosis (No. 488 in the Chadwick–Mann translation) states that "an incised wound in one temple produces a spasm in the opposite side of the body" (p. 263). Taken in combination, the two observations pro-

vide a basis for relating speech disorder to injury of the left hemisphere. There is no evidence that the correlation was made.

Aphasia in Roman Medicine

Some developments during the Roman period deserve mention. The Latin author and commentator, Valerius Maximus (ca. A.D. 30), described the first case of traumatic alexia (Benton & Joynt, 1960). Soranus of Ephesus and other medical writers of the period differentiated loss of speech due to paralysis of the tongue from that resulting from other causes (Creutz, 1934). However, the other causes were not specified, and whether these physicians had in mind a distinction between an articulatory and an amnesic type of aphasia is uncertain. Also during this period physicians and philosophers localized specific cognitive functions in different regions of the brain, more often in the cerebral ventricles than in the substance of the brain itself.

Aphasia in the Renaissance

One development during the Renaissance was the application of this schema of ventricular localization to specific problems of diagnosis and treatment. Antonio Guainerio, a fifteenth-century physician, mentioned two aphasic patients; one could say only a few words and the other showed paraphasic misnaming. Reasoning deductively, he ascribed their condition to an excessive accumulation of phlegm in the fourth ventricle, with consequent impairment of "the organ of memory" (Benton & Joynt, 1960). Direct surgical intervention to alleviate traumatic aphasia was another feature of Renaissance medicine. Reports describing cases of depressed skull fracture in which removal of bone fragments in the brain led to restoration of speech in the patient were published by Nicol Massa and Francisco Arceo (Benton & Joynt, 1960; Soury, 1899).

The following statement by Johann Schenck von Grafenberg (1530–1598) indicates that at least some physicians of the time understood that brain disease could cause a nonparalytic type of speech disorder:

> I have observed in many cases of apoplexy, lethargy and similar major diseases of the brain that, although the tongue was not paralyzed, the patient could not speak because, the faculty of memory being abolished, the words were not produced. (Benton & Joynt, 1960, p. 209)

Later writers made the same point. For example, in 1742, Gerard Van Swieten wrote that he had seen "many patients whose cerebral functions were quite sound after recovery from apoplexy, except for this one deficit—in designating objects, they could not find the correct names for them"

(Benton & Joynt, 1960, p. 211). These observations formed the basis for the classification of motoric and amnesic types of aphasic disorder that was made in the early nineteenth century.

Credit for the first explicit, albeit brief, description of the syndrome of alexia without agraphia goes to Gerolamo Mercuriale (1530–1606). In the course of discussing cerebral localization of function, he cited the case of a printer who had lost the ability to read after sustaining an epileptic seizure. He could still write but could not read what he had written. Mercuriale regarded the deficit as symptomatic of a partial loss of memory (Meunier, 1924).

Seventeenth-Century Contributions

Relatively detailed descriptions of cases that leave no doubt that the patient was truly aphasic are first encountered in the seventeenth century. Two of these case reports are of particular interest. One, published in 1676 by Johann Schmidt, described a patient who suffered from a paraphasic expressive speech disorder after a stroke. Eventually he recovered oral speech but was still completely alexic. He could write to dictation but "could not read what he had written even though it was in his own hand. . . . No teaching or guidance was successful in inculcating recognition of letters in him" (cited in Benton & Joynt, 1960, p. 209).

The second case report, entitled "On a Rare Aphonia," described a patient with a nonfluent expressive speech disorder and an equally severe incapacity for repetition. She was unable to repeat even short phrases, such as "God will help." Within this context of grossly defective conversational and repetitive speech, however, she showed remarkably preserved capacity for serial speech. Once she was started off, she could recite the Lord's Prayer, Biblical verses, and the like. It was this dissociation that led Peter Rommel, the author of the report which was published in 1683, to designate his case as an instance of a "rare aphonia" (see Benton & Joynt, 1960, for translations of the Latin texts of Schmidt and Rommel).

Eighteenth-Century Descriptions

Many allusions to different forms of aphasic disorder appeared during the eighteenth century. Among them was the first description, in 1745 by Olaf Dalin, of preservation of the capacity to sing in a patient with a severe expressive speech disorder (see Benton & Joynt, 1960, for a translation of the Swedish text).

The 1770 monograph of Johann Gesner entitled *Speech Amnesia* was the first major study of the disorder (see Benton, 1965). It was a landmark con-

tribution on a number of counts. From a clinical standpoint, the six case reports in it provided a wealth of information about such diverse features of aphasia as jargon aphasia and jargon agraphia, inability to read aloud with preserved ability to read silently for understanding, greater impairment in reading one language than another, and preservation of the ability to recite familiar prayers within the setting of defective conversational speech. Moreover, in contrast to most earlier authors, Gesner emphasized that word-finding difficulties and paraphasic speech reflect not a loss of memory in general but a specific type of memory loss, namely, speech amnesia. Finally, and perhaps most importantly, Gesner was the first to advance a theory of the nature of aphasic disorders in terms of "speech amnesia." In discussing jargon aphasia, he insisted that it did not signify a dementia but only a specific type of forgetting. He pointed out that ideation and the memory for words must be distinguished from each other. Ideation is evoked by the perception of physical objects and the action of the sensory nerves. The evocation of words follows ideation, and hence additional neural energy or action is required for it to take place. Therefore, it is understandable that brain disease could impair the memory for words but leave ideation intact, so that a patient might be able to recognize an object and know its significance, yet misname it or not be able to name it at all. The physical basis for such a disturbance in verbal memory was a sluggishness (Trägheit) in the relationships among the different parts of the brain.

Thus, in a rather vague way, Gesner advanced an associationist theory of aphasia, which stated that the disorder consisted of a failure to connect a perception or idea with its appropriate linguistic sign. He called the disorder SPEECH AMNESIA, as had his predecessors, but he went a step further by ascribing to what today we would call ASSOCIATIVE PROCESSES. Some 25 years later, Alexander Crichton (1798) expressed the idea with greater clarity. Writing about paraphasic speech, he suggested that this

> very singular defect of memory . . . ought rather to be considered a defect of that principle, by which ideas, and their expressions, are associated, than of memory; for it consists in this, that the person, although he has a distinct notion of what he means to say, cannot pronounce the words which ought to characterize his thoughts. (p. 371)

Table 1.1 presents a summary of the knowledge of aphasia accumulated by 1800. It is evident that a substantial amount of information about the disorder was available to the well-informed physician or layman. For the most part, this knowledge was of a clinical nature. Little had been written about the basic nature of the aphasic disorders, although brief statements of an associationist theory were made by Gesner and Crichton. The neu-

TABLE 1.1
Knowledge of Aphasia in 1800

Clinical Descriptions
Nonfluent aphasia: speechlessness
Fluent aphasia: anomia, paraphasia, jargonaphasia (Van Swieten, 1742; Gesner, 1770)
Agraphia (Gesner, 1770; Linné, 1745)
Alexia without agraphia (Mercuriale, ca. 1580; Schmidt, 1676)
Preserved capacity for serial speech (Rommel, 1683)
Preserved capacity for singing (Dalin, 1745)
Dissociation in reading different languages (Gesner, 1770)
Unawareness of defect (Van Goens, 1789; Crichton, 1798)
Law of emphasis on comprehension defects (Morgagni, 1762)
Theory
Defective "organ of memory" (Guainerio, 1481; Schenck von Grafenberg, 1583)
Defective association processes (Gesner, 1770; Crichton, 1798)
Neuropathologic Concepts
Ventricular localization: fourth ventricle (Guainerio, 1481)
Disease of the brain

rological basis of aphasia remained quite obscure as the primitive state of eighteenth century neuroanatomy and neurophysiology precluded the possibility of establishing meaningful correlations.

Aphasia: 1800–1860

During the first decades of the nineteenth century, advances were made along all lines: clinical knowledge, theoretical formulation, and neuropathology. A number of clinical studies contributed to the phenomenology of aphasia. Osborne (1833) described a highly educated patient with severe jargon aphasia who nevertheless was able to understand oral speech and to read. He could even read foreign languages, and, in contrast to his grossly defective speech, his writing was only mildly affected. Lordat (1843) reported what appears to be the first case of dissociation of language loss in a polyglot. The patient could hardly say a word in French but could speak fluently in his native Languedoc. Bouillaud (1825a) described involuntary echolalia in aphasic patients and called attention to the extreme verbosity of some patients. Marcé (1856) wrote a paper on agraphia in which he showed that the severity of the impairment in writing could vary independently of that in oral speech. He therefore postulated the existence of a cerebral center for writing that was distinct from the center for

oral speech. A decade later, Ogle (1867) confirmed the independence of the two forms of expressive language disability and used the term AGRAPHIA to designate impairment in writing.

More sophisticated theoretical formulations of the nature of aphasia were also advanced. The most important was that made by Jean-Baptiste Bouillaud (1796–1881), who classified aphasic disorders into two basic types, articulatory and amnesic. Bouillaud (1825a) insisted that it was necessary to "distinguish two different phenomena in the act of speech, namely the power of creating words as signs of our ideas and that of articulating these same words. There is, so to speak, an internal speech and an external speech." He then pointed out that "it is not uncommon to observe suspension of speech sometimes solely because the tongue and its congenerous organs refuse the pronunciation of words and sometimes because the memory of these words escapes us" (p. 43). Thus, one must distinguish between "two causes which can lead to loss of speech, each in its own way; one by destroying the organ of memory of words, the other by an impairment in the nervous principle which directs the movements of speech" (Bouillaud, 1825b, pp. 285–286). The validity of Bouillaud's division of aphasic disorders into an articulatory, apraxic, aphemic category and an amnesic category is still generally accepted under the rubric of "nonfluent" and "fluent" types of aphasia.

The problem of the neuropathological basis of the aphasic disorders was first brought into prominence by anatomist and phrenologist Franz Joseph Gall (1758–1828). His theory held that the human brain was an assemblage of organs, each of which formed the material substrate of a specific cognitive ability or character trait. Among the approximately 30 traits localized in his system were two cerebral "organs" of language, one for speech articulation and the other for word memory, which he placed in the orbital region of the frontal lobes.

Gall's hypothesis that the brain is not a unitary equipotential organ, but instead consists of an aggregate of functionally specialized areas, attracted both loyal supporters and vigorous opponents. No issue was more hotly debated than his localization of speech and language in the frontal lobes, and there was no more ardent champion of the concept than Bouillaud, who marshaled clinical as well as pathological evidence to support the contention.

However, Bouillaud's evidence was not altogether convincing, and, in any case, empirical testing of the hypothesis by others did not support it. For example, clinical pathologist Gabriel Andral (1797–1876) reported on the clinical status during life of 37 patients in whom he had found lesions of the frontal lobes on autopsy. Speech disturbances had been present in 21 patients, whereas 16 had shown no signs of speech disorder. Moreover, An-

dral had seen 14 cases of aphasia in which lesions were confined to postrolandic areas and did not involve the frontal lobes. He therefore concluded that "loss of speech is not a necessary result of lesions in the anterior lobes and furthermore it can occur in cases in which anatomical investigation shows no changes in these lobes" (Andral, 1840, p. 368; translation by the authors). There were 11 cases of clearly identifiable unilateral disease in Andral's sample, 5 with left hemisphere lesions and 6 with right hemisphere lesions. Of the five left hemisphere cases, three were aphasic; of the six right hemisphere cases, none was aphasic. Apparently this trend toward a hemispheric difference in the occurrence of aphasic disorder failed to capture Andral's attention (cf. Benton, 1984).

Aphasia: 1861–1900

Paul Broca

The protracted controversy over the validity of Gall's placement of language centers in the frontal lobes was no nearer resolution in 1860 than in 1830, but it did serve a most important function. It provided the impetus for surgeon and physical anthropologist Paul Broca (1824–1880) to examine the brains of two aphasic patients who had been under his care during the last months of their lives. The autopsy findings showed that the lesion that was ostensibly responsible for the nonfluent aphasic disorder shown by these patients during life was situated in both cases in the posterior part of the frontal lobe. At the time, Broca interpreted his findings as supporting the Gall–Bouillaud thesis that the seat of language was in the frontal lobes, and he made no particular reference to the fact that the lesions were left-sided. As he collected additional cases, however, his attention was drawn to the unilateral nature of the lesion causing the nonfluent impairment of speech, to which he gave the name APHEMIA. Reporting in 1863 on the autopsy findings in eight aphasic patients (Broca, 1863), Broca noted that all had lesions on the left frontal lobe. He rather cautiously added, "I do not dare to draw a conclusion and I await new findings." The "new findings" were soon forthcoming, and in 1865, Broca enunciated his famous dictum, "We speak with the left hemisphere" (see Wilkins, 1964; Rottenberg & Hochberg, 1977; and Berker, Berker, & Smith, 1986, for English translations of Broca's papers).

The validity of Broca's generalization was readily confirmed, and the doctrine of hemispheric cerebral dominance for language was born. At practically the same time, a number of clinicians added the qualification

that left hemisphere dominance for speech held only for right-handed persons; in left-handers, the right hemisphere appeared to be dominant for language function.

Broca's discovery led to a major revolution in medical and physiological thinking. From a medical standpoint, aphasia was transformed from a minor curiosity to an important symptom of focal brain disease. From a physiological standpoint, the reality of cerebral localization was established, and this led to a period of intense investigation of functional localization in both animal and human subjects (Benton, 1977; Young, 1970).

The place of Marc Dax (1771–1837) in this history deserves mention. In the 1860s, his son, Gustav Dax (1815–1893), asserted that in 1836, Marc Dax had written a paper in which he assembled a mass of evidence to show that aphasia was related to disease of the left hemisphere. This unpublished paper, entitled "Lesions of the Left Hemisphere Coinciding with Forgetfulness of the Signs of Thought," was then published by Gustav Dax in 1865. There followed a minor controversy over whether Dax or Broca should be accorded priority for the discovery of left hemisphere dominance for speech (see Critchley, 1965). Analysis of the question indicates that although Marc Dax did write the remarkable paper that his son published three decades later, there is no evidence that he presented it at a regional medical meeting in Montpellier, as was claimed (Joynt & Benton, 1964). Apparently the paper remained a private document. Thus, it seems that Dax did discover the special relationship between left hemisphere disease and aphasia about 25 years before Broca's first observation (Benton, 1984). However, he did not make his discovery known to the medical world other than through the distribution of copies of his paper to two or three friends.

The Contribution of Carl Wernicke

When Broca made his localization, he emphasized that he did not mean to imply that all forms of aphasia were related to left frontal lobe disease but only the motoric type, which he called aphemia and which was essentially the same as the articulatory and asynergic types of the disorder described by Bouillaud and Lordat. It remained for a German neuropsychiatrist, Carl Wernicke (1848–1905), to demonstrate that the occurrence of the other major type of aphasic disorder, that is, the amnesic type, was related to disease of the left temporal lobe. In a monograph published in 1874 (when he was 26 years old), Wernicke described the major features of what he called SENSORY APHASIA, now called WERNICKE'S APHASIA (see Cohen & Wartofsky, 1969, and Eggert, 1977, for English translations of Wernicke's 1874 monograph). These features were fluent but disordered speech, anal-

ogous disturbances in writing, impaired understanding of oral speech, and impairment in both oral and silent reading. The crucial, or at least the most frequently occurring, lesion associated with this aphasic syndrome was situated in the hinder part of the first temporal gyrus of the left hemisphere, the region now known as WERNICKE'S AREA.

Wernicke's contribution was by no means limited to this important discovery (Geschwind, 1967). He also pointed out the danger of mistaking sensory aphasia, characterized by disordered speech and impaired understanding, with a confusional or even a psychotic state. He also emphasized the necessity for distinguishing between an aphasic impairment in naming objects and an agnosic (or asymbolic) failure to recognize objects, a point made by Freud (1891) some 15 years later. Moreover, he not only accounted for known aphasic syndromes by means of the rather simple neural model that he developed, but he also correctly predicted the existence of syndromes that had not yet been described.

Wernicke reasoned what would be the outcome of a lesion interrupting the connection between Wernicke's area and Broca's area (to use current terminology). Because Wernicke's area was intact, the patient's understanding of oral speech as well as his or her silent reading would be preserved. Moreover, the patient's oral speech expression would be fluent, because Broca's area was also intact. Nevertheless, oral speech expression would be disordered, that is, paraphasic, because the break in the connection between Wernicke's area and Broca's area would prevent effective translation of sound-images into spoken sounds. Wernicke called this aphasic syndrome, which had not yet been described clinically and was therefore hypothetical, CONDUCTION APHASIA to distinguish it from syndromes caused by lesions in the cortical centers. Lichtheim (1885) agreed with this formulation and added an important defining feature to the syndrome, namely, the inability to repeat spoken utterances.

The Associationist School

Broca and Wernicke were not only localizationists but also associationists. Like Gesner and Crichton, they thought of aphasic disorders as disturbances in attaching appropriate verbal labels to ideas, objects, or events, with basic intellectual capacity remaining essentially intact. The formulations were in terms of interconnected cortical centers that served as depositories for the auditory and visual memories of words and of the movement patterns of speech and writing. Models of this type were proposed by most of the leading aphasiologists of the late nineteenth century.

The schema of Lichtheim (1885), an elaboration of a simpler model proposed by Wernicke, postulated the existence of five interconnected cortical

centers, four of which serve different aspects of language and a fifth center in which concepts or ideas are formulated.

Reasoning deductively from his models, Lichtheim predicted the existence of seven major aphasic syndromes. Some of them were familiar to clinicians; others were still hypothetical. One syndrome postulated by Lichtheim concerned the consequences of a break in the connection between the concept center (C) and the expressive speech center (M). He predicted that spontaneous speech would be impoverished because ideas could not reach verbal expression. On the other hand, because the basic mechanisms of speech are intact, strictly linguistic performance such as repetitive speech and reading aloud would be preserved. The understanding of speech would also be spared, as both the center for memory of word sounds (Center A) and its connection with Center C remain intact. The real existence of this syndrome, designated by Wernicke (1886) as TRANSCORTICAL MOTOR APHASIA, also was confirmed by subsequent clinical observation.

The approach of Wernicke, Lichtheim, and other "diagram makers" (to use Head's [1926] derisive term) was notably successful in some respects, especially in linking aphasic disorders with brain function. However, there was general agreement that it was only partially successful in accounting for the diverse phenomena of aphasia and the curious combinations of symptoms often encountered in aphasic patients. Moreover, serious objections to this associationist–anatomical approach were advanced by clinicians who viewed aphasia as being more than a linguistic disorder in the narrow sense, believing that it was as much a defect in thinking as in speech (Goodglass, 1988).

The Cognitive School

The influential French clinician Armand Trousseau (1801–1867) was the first major figure to challenge the assumption that thinking per se is not impaired in uncomplicated aphasic disorder. Attacking Broca's concept of aphemia, he cited cases from the literature and from his own practice to show that aphasic patients, whose disability appeared on superficial examination to be of a purely linguistic nature, in fact showed numerous intellectual defects. He concluded categorically that "intelligence is always lamed" in aphasia (Trousseau, 1865).

The theme was then developed in greater depth by English neurologist John Hughlings Jackson (1835–1911), who many consider to be the founder of the "cognitive school" in the field of aphasia. Following an earlier brief formulation by French physician Jules Baillarger (1809–1890), he distinguished between two levels of speech: emotional (or automatic) and intel-

lectual. It is the intellectual level of utterance, involving the statement of "propositions," that is impaired in the aphasic patient, who may show considerable preservation of automatic language in the form of interjections, oaths, cliches, and recurring utterances.

The essential defect in aphasia, according to Jackson (1878), consisted of a loss of this ability to "propositionise," that is, to use words in the service of thought. At the same time, the capacity to use words as a form of emotional expression might well be retained. The capacity for propositional speech is an intellectual, not a narrowly linguistic, ability, and consequently the aphasic patient of necessity "will be lame in his thinking" since "speech is a PART of thought" (Jackson, 1874).

There was resistance to the basic assumption, associated with the localization models of the diagram makers, that a limited cortical region, such as Broca's area or Wernicke's area, was the repository of memory-images of speech movements or word sounds. It seemed incomprehensible to some students of brain function that the nervous elements comprising these cortical centers could be endowed with such extraordinary functional properties (Benton, 1977). It was not that these critics believed in the functional equipotentiality of all regions of the hemisphere or denied the facts of clinical localization of lesions, but they were convinced that the diagram makers had fallen into the error of confusing symptom with function. Jackson (1874) made this point succinctly when he warned that "to locate the lesion which destroys speech and to locate speech are two different things."

Despite opposition, the concept that aphasia was a group of disorders produced by breaks in the connection of objects, events, and ideas with their appropriate verbal signs was the one held by most neurologists as of 1900. However, there was a significant change in thinking during the next 25 years in France and Britain as a result of the contributions of Pierre Marie and Henry Head, both of whom were vigorous proponents of the cognitive position and of a unitary theory of the nature of aphasia.

Early Twentieth-Century Developments

Associationist Models

During the early twentieth century, there were further elaborations of the concept that discrete cortical and subcortical centers and their interconnections provided the neurological basis of language functions. Joseph-Jules Dejerine (1849–1917) was a major figure in this development. Among

his notable achievements was his demonstration of the anatomical substrate of the syndrome of pure alexia without agraphia and his interpretation of it as a "disconnection syndrome" (see Geschwind, 1965; Lecours & Lhermitte, 1979). S. E. Henschen (1847–1930) undertook a monumental analysis of all of the clinicopathological reports in the literature on aphasia and formulated a detailed model that postulated the existence of numerous separate cortical centers for almost every aspect of language function as well as for such allied activities as calculation and musical appreciation and expression (Henschen, 1919–1922). Still another important figure was Karl Kleist (1870–1962), who, on the basis of detailed examination of soldiers with penetrating brain wounds, believed that it was possible to make a precise localization of the cortical regions underlying diverse language performances (Kleist, 1934).

Cognitive Models

The development of explicit cognitive theories of the nature of aphasia was an important feature in the development of thought about the disorder during the first quarter of the twentieth century. In large part this development was due to the contributions of Pierre Marie (1853–1940) and Henry Head (1861–1940). Both of these neurologists viewed aphasia as a single disorder that necessarily incorporated the component of intellectual defect.

The first of Marie's papers on the subject bore the provocative title, "The Left Frontal Convolution Plays No Special Role in Language Function" (Cole & Cole, 1971; Marie, 1906). In it he flatly denied that Broca's area was a center of expressive speech and cited clinical evidence to support this negative conclusion (see Burckhardt, 1891, also Whitaker, Stemmer, & Joanette, 1996, and Dandy, 1922, for descriptions of neurosurgical resections of Broca's area with little or no language impairment). Subsequent papers in the series vigorously attacked the pluralistic concept of discrete types of aphasic disorder and proposed that there was in fact one basic disorder, the syndrome of disturbed fluent speech expression and impaired understanding of speech known as Wernicke's aphasia. In consonance with his unitary conception of the nature of aphasia, Marie proposed a single broad localization of the lesion causing the disorder. Later he modified his opinion to some degree, but at the time when he advanced his famous "Revision of the Question of Aphasia," he implicated a single extensive territory that included the posterior parts of the first and second temporal gyri, the supramarginal gyrus, and the angular gyrus.

The work of Henry Head (1926) in England was equally influential in fostering a significant change in attitude about the role of cognitive factors

in aphasia. Defining the disorder as a loss of capacity for symbolic formulation and expression, Head insisted that it involved an impairment in thinking that was reflected in both the patient's nonverbal performances as well as verbal behavior. Consequently, the extensive test battery that he developed for the assessment of aphasic disorder included nonverbal tasks that are not ordinarily considered to be measures of language functions.

Another figure deserving mention in this context is Kurt Goldstein (1876–1965), who applied cognitive theory with particular force to the category of amnesic aphasia, with its defining features of impairment in object naming and difficulty in finding words in conversation. Goldstein (1924) insisted that these linguistic defects were a direct expression of a loss of the abstract attitude that was also reflected in defective performance on such nonverbal tasks as the sorting of colors and the classification of objects, which also made demands on the capacity for abstract reasoning.

The Modern Period

To divide the history of a topic into periods or stages is necessarily a somewhat arbitrary procedure. We have designated 1935 as the beginning of the "modern period" because this was the year of publication of the comprehensive study by Weisenburg and McBride that generated a substantial amount of new information about aphasia and at the same time provided a methodological model for subsequent investigations.

Weisenburg and McBride (1935) gave an extensive battery of verbal and nonverbal tests to 60 aphasic patients and compared their performances with those of 38 nonaphasic patients with unilateral brain disease and 85 control patients (i.e., who had no evidence of brain disease). The nonverbal component of their test battery included drawing, visuoperceptual, and block-assembling tasks. They found that although a majority of the aphasic patients showed inferior performance on varying numbers of the nonverbal tasks, there was considerable interindividual variability: Some patients performed on a defective level on many tests and others performed adequately on all tests. They also found a positive relationship of moderate degree between the extent of the observed cognitive impairment on the nonverbal tests and the estimated severity of the patient's aphasic disability.

Subsequent studies of cognitive function in aphasia generally followed the pattern established by Weisenburg and McBride of comparing the performances of aphasics with those of nonaphasic patients with unilateral brain disease and control patients or normal subjects. These studies produced a broad spectrum of results that did not permit a simple interpreta-

tion and confirmed the position of Weisenburg and McBride that interindividual variability is an overriding feature of the performances of aphasic patients.

An important development in recent decades has been a concerted effort to describe in precise terms the alterations of speech shown by aphasic patients and to relate these changes to the anatomic substrate of the disorder as well as to the cognitive status of aphasic patients. Dealing with such disturbances as AGRAMMATISM, PARAGRAMMATISM, "word-finding disturbance," and SYNTACTIC APHASIA, this effort required expertise in the field of linguistics. As a consequence, a number of linguists became actively engaged in the study of aphasia, which came to be known as NEUROLINGUISTICS.

Although some neurologists and psychologists (e.g., Alajouanine, Ombredane, & Durand, 1939; Kleist, 1934; Ombredane, 1926, 1933; Pick, 1913) had dealt with neurolinguistic problems in earlier years, the seminal figure in the field was Roman Jakobson. His monograph (Jakobson, 1941) comparing the phonemic disturbances in the utterances of aphasic patients to the speech of children and relating both to more general aspects of phonology had a profoundly stimulating effect on subsequent investigative work.

Efforts also continued on several fronts to define the anatomical basis of aphasia. The extreme localizationist approach to the "anatomy" of the aphasic disorders was carried forth by California neurologist Joannes Nielsen (1890–1969), whose thinking followed the lines laid down by Henschen (1919–1922) and Poetzl (1928). His monograph (Nielsen, 1936) appeared at a time when concern with identifying the neural mechanisms underlying language function and language disorder had diminished somewhat from that of earlier decades. Interest in the area revived after World War II, however, with the expanded opportunities for study afforded by the large number of casualties produced by that conflict.

Contemporary Developments

A pathbreaking and influential paper by Geschwind (1965), in which agnosic, apraxic, and aphasic disorders were interpreted as products of neural disconnection, provided the impetus for a fresh approach to the anatomical study of aphasic syndromes. From the time of Broca's and Bouillaud's descriptions of frontal lobe damage and speech impairment, the study of aphasia had relied heavily on the lesion method, by which correlations were established between circumscribed areas of brain dysfunction and

patterns of behavioral alteration. Geschwind analyzed the lesion studies of the preceding century and proposed a model of brain and language in which the key components were two major language centers located in Broca's and Wernicke's areas, connected by a major pathway (the arcuate fasciculus), which receive contributions from auditory cortices and other peri-Sylvian regions.

Geschwind's paper preceded by only a few years the beginning of a series of technological advances which were to provide unprecedented opportunity for testing models of brain and language. Until this time, the essential means of obtaining anatomical correlates of aphasic signs was to autopsy the brains of aphasic patients, but there were major limitations inherent in relying exclusively on postmortem studies. For example, considerable time often passed between the onset of aphasia and access to the brain during which subsequent neurological events might introduce factors complicating interpretation. In addition, the fact that anatomical study necessarily followed evaluation of patients' language made anatomically driven hypotheses difficult to test in any convincing fashion.

By the mid-twentieth century, pneumoencephalography and angiography were being applied in clinical settings to provide general anatomical pointers to guide management of neurologic patients, including those with acquired language disorders. These procedures had considerable risks associated with the vague anatomical information, but the limited findings reported from retrospective analysis of clinical records were in concordance with known principles of aphasia (cf. Rosenfield & Goree, 1975). Electroencephalography (EEG) lacked the risks of the invasive radiographic procedures, but was even less informative about the neural basis of language disorders (cf. Haaland & Wertz, 1976).

Radioactive isotope brain scans were used by D. Frank Benson in the mid-1960s in the first studies to correlate systematically neuroimaging findings with concurrent aphasic signs (Benson, 1967; Benson & Patten, 1967). These images had poor resolution and could be used only during the time of blood–brain barrier breakdown, typically within the first 6 weeks after stroke onset, during which time aphasic conditions are rarely stable. The scans did not allow the boundaries of lesions to be visualized, but general areas of abnormality could be located on an anterior–posterior axis relative to a rough outline of a lateral view of the brain. This work provided the first radiologic confirmation of Wernicke's distinction between fluent "sensory" aphasia associated with posterior left hemisphere lesions and nonfluent "expressive" aphasia associated with anterior lesions. Radioactive isotope brain scans were applied to a limited extent by other aphasiologists (e.g., Karis & Horenstein, 1976; Kertesz, Lesk, & Mc-

Cabe, 1977; Verhas, Schoutens, & Demol, 1975), but attempts at detailed anatomical interpretations were based more on associated neurological signs than on the blurred radiographic images (e.g., Rubens, 1975).

It was the advent of computerized tomography (CT) in 1973 that set the stage for the next round of advances in aphasiology. By passing narrow, highly restricted beams of radiation through the head from multiple points and summing the radiation intensity measurements for all beams passing through a region, CT allowed detection of small differences in tissue radiodensity. The resolution of early CT images was poor by today's standards, but the level of detail far surpassed that of any prior method of visualizing the brains of living patients. Not only was relatively precise definition of lesion boundaries possible, but CT also allowed for reliable identification of major anatomical landmarks, including the ventricular system, the sylvian fissure, and the central sulcus.

The superiority of CT over angiography and radioisotope scans for the study of aphasia was demonstrated directly by Yarnell, Monroe, and Sobel (1976) in an outcome study of 14 patients. Mohr showed the potential utility of CT for addressing heretofore puzzling anatomical issues in early CT-based reports of aphasia caused by lesions in the left thalamus (Mohr, Watters, & Duncan, 1975), and in Broca's area versus Broca's area plus surrounding regions (Mohr, 1976). In 1977, Hayward, Naeser, and Zatz published the first systematic study of aphasia to apply this powerful new technology (see also Naeser & Haward, 1978). They recognized a number of potential methodological pitfalls which often were ignored as use of CT became widespread. Their study included only patients with single, focal lesions caused by stroke; they used CT scans obtained after the lesions were relatively stable; and they conducted image analyses blind to the patients' behavioral profile. The findings were consistent with the classical anatomical localizations for the major aphasia diagnostic categories, and were interpreted as generally supporting Geschwind's model of aphasia.

A number of CT scan studies of aphasia appeared in the late 1970s and early 1980s, taking advantage of further technological developments, including improved image resolution and the development of template systems for more reliable and precise lesion localization (e.g., H. Damasio, 1983). In most cases, the new findings were confirmatory, in that they bore out classical anatomical–linguistic relationships previously discerned from autopsy studies. In other instances, application of the new imaging technique led to findings that had not been predicted by earlier studies or models of aphasia (e.g., Mazzocchi & Vignolo, 1977).

Castaigne, Lhermitte, Signoret, and Abelane (1980) used CT to study the preserved brain of Broca's patient Leborgne in an effort to resolve the historical disagreement between Dejerine and Marie regarding the anatomi-

cal basis of his aphasia. Other investigators applied CT to distinguish between the relatively mild and transient speech problems that arise from damage limited to the inferior frontal gyrus, and the syndrome of Broca's aphasia as traditionally defined, which requires substantially larger areas of damage to the frontal operculum, underlying white matter, and the insula (e.g., H. Damasio, Damasio, Hamsher, & Varney, 1979; Mohr et al., 1978; Naeser & Hayward, 1978).

Computerized tomography studies also refined views on global aphasia, which historically was viewed as the outcome of a massive lesion in the territory of the left middle cerebral artery, with combined damage to Broca's and Wernicke's areas and subcortical structures. With CT imaging, the syndrome was found to be more anatomically heterogeneous than previously expected. For example, it was possible for global aphasia to occur despite preservation of Wernicke's area (Mazzocchi & Vignolo, 1977). In the infrequent cases of global aphasia without hemiparesis, lesions were limited to prerolandic regions, or preserved regions of motor cortex were found between areas of damage to prefrontal and posterior language cortices (Deleval, Leonard, Mavroudakis, & Rodesch, 1989; Tranel, Biller, Damasio, Adams, & Cornell, 1987; Vignolo, Boccardi, & Caverni, 1986). The refined views of the anatomical underpinnings of global aphasia were of more than academic interest, as the various imaging profiles helped predict long-term outcome.

One of the more important advances that resulted directly from the application of CT was elucidation of the sites of subcortical damage associated with "atypical" aphasias which did not correspond behaviorally to any of the classical diagnostic categories. Circumscribed left hemisphere lesions involving the anterior limb of the internal capsule, the head of the caudate nucleus, the anterior portion of the putamen, and the thalamus were found to be sufficient to cause aphasia, and models of the neuroanatomical basis of language were adjusted accordingly (e.g., A. R. Damasio, Damasio, Rizzo, Varney, & Gersch, 1982; Graff-Radford & Damasio, 1984; Naeser et al., 1982).

The new anatomical data that arose from CT studies of Wernicke's aphasia contributed substantially less to revising views on the neural basis of this condition. In fact, a number of modern-era neuroimaging studies provided considerable support for Wernicke's (1886) original description (e.g., H. Damasio, 1981; Kertesz, Harlock, & Coates, 1979; Naeser & Hayward, 1978). Even in this situation, where the original autopsy-based findings were accurate and noncontroversial, CT studies broadened understanding of the condition, for example, by revealing which specific temporal and parietal lobe regions of damage were most predictive of long-term outcome (Kertesz, Lau, & Polk, 1993; Naeser et al., 1987).

Before the advent of CT, conduction aphasia was recognized as an independent aphasia diagnostic category, but little information was available regarding its anatomical basis (Benson et al., 1973; Geschwind, 1965; Konorski, Kozniewska, & Stepien, 1961). Several investigators applied the new technology to define the various areas of damage that could cause this syndrome, including the supramarginal gyrus and subjacent white matter, the auditory cortices, and the insula (H. Damasio & Damasio, 1980; Kertesz et al., 1979; Naeser & Hayward, 1978). Although, like virtually all aphasias, some white matter damage was typically present in conduction aphasia, there was also damage to cortex in nearly all cases. The traditional account of the repetition defect of conduction aphasia caused by isolated interruption of white matter connections between Wernicke's and Broca's areas was no longer tenable.

Likewise, transcortical motor aphasia (TMA) was a clearly recognized clinical entity but information on its anatomical basis was sparse. On the basis of radionuclide scans and associated neurological signs, Rubens (1975) posited that TMA resulted from lesions in superior and mesial premotor regions. These findings were confirmed and further refined by CT studies (A. Damasio & Kassell, 1978; Freedman, Alexander, & Naeser, 1984; Naesser & Hayward, 1978). Contemporaneous CT scan studies also refined descriptions of the anatomical basis of transcortical sensory aphasia, further ingraining the essential role of CT in lesion studies of aphasia (Kertesz et al., 1979; H. Damasio, 1981).

Magnetic resonance imaging (MRI) was developed almost a decade after CT. This process measures differences in the response of the atomic nuclei of various tissue types to a brief pulse of radio waves applied within a strong magnetic field. It was the first neuroimaging technique not based on radiation physics. Thus MRI had advantages for repeated scanning without excess radiation exposure for the patient, and also provided enhanced anatomical detail, such as the ability to distinguish between gray and white matter. The increased detail generated by MRI has provided evermore impressive and definitive images of the structural correlates of aphasia, and technological progress has continued to increase its value. For example, the human brain now can be reconstructed in three dimensions by high resolution MRI without interslice gaps, and computerized techniques for individualized image analysis allow reliable identification of every major gyrus and sulcus (H. Damasio, 1995). Despite this, the impact of MRI on aphasiology has been subtle compared with that of CT, with advances generally taking the form of modest refinement and elaboration of pre-MRI findings.

Magnetic resonance imaging has helped the study of progressive apha-

sia, a relatively infrequent condition marked by the gradual onset and progression of isolated language impairments, including anomia, impaired fluency, and impaired auditory comprehension. Pick's disease or focal spongiform changes are most often involved, and the anatomical changes can be visualized as slowly progressive atrophy in the left anterior temporal lobe (Graff-Radford et al., 1990).

Functional imaging, including single photon emission computed tomography (SPECT), positron emission tomography (PET), and functional MRI (fMRI), has provided yet another window into the brains of aphasic patients. Unlike the structural information provided by CT and MRI, these techniques allow visualization of regions of increased or decreased metabolic activity or blood flow. Beginning with the work of Petersen, Fox, Posner, Mintun, and Raichle (1988), much of the contribution of functional imaging to the understanding of brain function and language has been obtained from activation studies of normal, nonaphasic subjects, and thus is beyond the scope of this chapter. However, this rich source of information clearly complements the study of aphasia, and there have been attempts to link PET language activation profiles in normals directly with language impairment profiles in patients with focal lesions (e.g., H. Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996).

Functional imaging also has proven useful in elucidating aphasic syndromes for which CT and MRI findings have been poorly correlated with clinical conditions. For example, crossed aphasia, in which a right hemisphere lesion in a right-handed patient causes aphasia, is a rare and puzzling condition. Both right hemisphere language dominance and diaschisis (reduction of metabolism in the contralateral hemisphere) have been proposed as explanatory mechanisms. Recent SPECT and PET studies demonstrate that some hypometabolism in the structurally intact left hemisphere does occur in many crossed aphasics in the acute phase following aphasia onset; but persistent right hemisphere hypometabolism appears to be primarily responsible for the chronic condition, favoring the explanation of right hemisphere language dominance (e.g., Baker, Kirshner, & Wertz, 1996; Gomez-Tortosa, Marin, Sychra, & Dujovny, 1994; Perani, Papagno, Cappa, Gerundini, & Fazio, 1988).

As noted earlier, progressive aphasia may be accompanied by focal atrophy in left temporal regions, which can be seen with MRI. However, the MRI may be normal in some cases of progressive aphasia, whereas PET reveals hypometabolism in left hemisphere language-related regions (e.g, Chawluk et al., 1986). Functional imaging studies that combine activation paradigms in normal and aphasic subjects also provide new information on aphasic processes without macroscopic lesion correlates, such as the

naming impairment associated with progressive aphasia (Grabowski, Damasio, & Anderson, 1997), and the neural correlates of recovery from aphasia (e.g., Weiller et al., 1995).

Functional MRI detects increases in blood oxygenation which reflect local changes in blood flow to active brain areas. A major advantage over PET is faster temporal resolution, but the spatial resolution has also advanced to a point beyond the hopes of most cognitive neuroscientists even 5 years ago. The implications of these advances for the study of aphasia have yet to be realized.

Taken as a whole, the evolution of neuroimaging techniques over the past quarter century has changed forever the study of the neural basis of aphasia. These developments rest firmly on the autopsy studies that precede them, with most of the findings from the earlier studies standing the test of time. The theoretical accounts based on the early findings have fared less well, as new data have contributed to the development of more elegant models of the neural basis of language. The lesion method, reinvigorated by progress in imaging, remains a cornerstone of modern aphasiology, but it has been separated from the theoretical accounts traditionally associated with it.

Advances in neuroimaging have had a greater impact on some aspects of aphasia (e.g., subcortical "atypical" aphasias) than on others (e.g., Wernicke's aphasia), with the greatest progress being made in those areas where autopsy findings were inconclusive. Much of the progress has been in a greater appreciation of the complexity and heterogeneity of the anatomical substrates of the basic syndromes. Furthermore, in vivo imaging techniques, together with electrophysiological explorations of language cortices of epileptic patients prior to corticectomies (e.g., Ojemann, 1983), have extended beyond the peri-Sylvian area the boundaries of the neural regions believed to function in language. With the exquisite detail provided by current neuroimaging capabilities, access to neuroanatomical data no longer is the limiting factor it once was for progress in aphasiology.

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2

Signs of Aphasia

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This chapter discusses the clinical presentation of the aphasias, the major types, and the principal signs. A discussion of the neuroanatomy and neurophysiology of language (and, by extension, a discussion of the physiopathology of the aphasias) is outside the scope of this text. However, the comments on the clinical evidence presented here reflect a particular theoretical perspective on neuroanatomy, neurophysiology, and physiopathology, which has been documented elsewhere in detail (A. Damasio, 1989a, 1989b, 1989c; A. R. Damasio & Damasio, 1994; H. Damasio & Damasio, 1989).

It is important to note that when I refer to the LOCALIZATION VALUE or CORRELATE of a given sign, I do not mean to say that the correlated brain locus normally operates to produce whatever function a sign reports as missing. In the perspective outlined in the earlier articles, language, along with other complex cognitive processes, depends on the concerted operation of multicomponent, large-scale neural systems. The anatomical components are often widely dispersed and each acts as a partial contributor to a complicated process rather than as a single purveyor. In this view, Wernicke's area, a part of which is a component of a language system, does *not* accomplish anything as complicated as auditory comprehension, although its impairment leads to auditory comprehension defects (A. Damasio, 1992).

Aphasia

Aphasia is a disturbance of the complex process of comprehending and formulating verbal messages that results from newly acquired disease of the central nervous system (CNS).

The disease processes that cause aphasia are acquired (e.g., cerebral infarction, tumor, contusion) rather than congenital (e.g., genetic or environment-induced perinatal cerebral defect). The former befalls individuals previously capable of using language appropriately. The latter may produce developmental language defects in individuals whose ability to use language will never attain a normal level.

Although it is clear that all mental activity and communication stems from the activity of the CNS, reference to the CNS is important because aphasia is not the result of a peculiar utilization of language related to psychogenic or social deviations.

Throughout the chapter I refer to VERBAL COMMUNICATION and LANGUAGE almost interchangeably. Under the terms verbal and language I include both auditory-based words and the visuomotor-based signs of sign languages. On the other hand, language and speech are not interchangeable. The latter should be reserved for the act of "speaking a verbal message" independently of the process of formulating the message itself. In this definition, I use VERBAL MESSAGES to call attention to the fact that aphasia relates exclusively to a disturbance in the use of words and signs, as opposed, for example, to the use of gestures, facial expressions, or body expressions, which are also important components of social communication but are not language in the sense used here.

Aphasia can affect comprehension of the language the patient either hears spoken or sees written, or both. It can also affect the comprehension of visuomotor signs from a sign language. Aphasia can affect the formulation of oral language production, writing, or both. In users of a sign language it affects the ability to sign in a linguistically correct manner.

Aphasia often disturbs both reception and expression of language, in both visual (written) and auditory (spoken) modes. Yet, each of the several fundamental types of aphasia compromises one of these modes preponderantly. Indeed, in some instances (e.g., in PURE ALEXIA or in PURE WORD DEAFNESS), only one of these abilities suffers while all others remain unaffected. There is more about this particular question later in the chapter.

The emphasis on COMPREHENDING and FORMULATING is especially pertinent. Aphasics have trouble comprehending verbal messages, that is, deciphering their meaning as opposed to hearing or seeing those messages. Neither deafness (peripheral or central) nor blindness is the problem. A deaf or blind person cannot comprehend language in the modality of the perceptual impairment, but will comprehend the same verbal message normally when processed by an intact sensory channel, for example, tactile Braille reading in the blind. Aphasics also have trouble formulating verbal messages, for example, selecting the lexical and syntactical items necessary to convey meaning and deploying them in a relational frame-

work such that meaning is indeed imparted on the receiver of the message. Yet an impediment of phonation that prevents speech production has nothing to do with formulation of verbal messages (people can still write what they cannot say), nor does the loss of one's hands interfere with language formulation (people may still say it if they have formulated it, and they may even write with a pen held between the teeth or the toes).

To characterize the nature of the disturbance, stating what aphasia is not becomes just as important as stating what it is. To begin with, aphasia is not a disturbance of articulation. Many patients suffer from speech disturbances due to acquired disease of the basal ganglia, of the brain stem or cerebellum, or even of the cerebral cortex, and yet few of those patients will have aphasia. Although their speech sounds are poorly formed or are inappropriately repeated, word selection and sentence structure are grammatically correct, appropriate to the intentions of their author, and understandable to the attentive listener. That is to say, such patients have a speech disturbance but it does not follow that they have a verbal language disturbance: Their language formulation is normal, their communication is linguistically correct, and thus they do not have aphasia.

Patients with mutism, who can be entirely silent, are not necessarily aphasic, although on occasion their absence of speech does conceal an aphasia. Often these patients fail to indicate any desire to communicate by gesture, mimicry, or writing. Consequently, little is known about what they do or do not comprehend, or about what they may or may not want to say (or think, for that matter). However, when most mutism patients awake from these peculiar states of apparent indifference, they resume language communication and show no evidence of aphasia. When probed about their abnormal behavior, these patients clearly relate a strange experience of avolition and diminished richness of thought content, but not of any problem with the actual composition of verbal communication. Most such patients have disease in areas of the brain that are different from those that produce the aphasias, for example, in the supplementary motor area or in the cingulate gyrus as opposed to the region surrounding the Sylvian fissure. A few have acute psychotic states and no macroscopically detectable brain disease, although they may suffer from profound changes in neurotransmitter systems innervating certain regions of the brain.

Also not aphasic are patients with aphonia that may result from diseases of the larynx and pharynx. They are mute, in the narrow sense of the word, and are suffering from an impediment in their phonatory apparatus that prevents them from speaking. They should be able to comprehend language (and indicate so by nodding or pointing responses), and they should be able to turn their thoughts into language by writing, in addition to being able to mouth words. The exception, other than for malingering, is a

conversion reaction, the currently infrequent psychiatric diagnosis of HYSTERICAL APHONIA.

Finally, it should be noted that the language disorder experienced in altered states of awareness is not an aphasia. Any patient with a confusional state will produce disturbed language and fail to comprehend verbal communication. But such patients have a concomitant disorder of their thought processes that parallels the language disturbance. Unlike the patient with aphasia who struggles to turn properly organized meanings into language and fails (or tries, without success, to turn the message heard into internal meaning), patients with confusional states communicate their disordered thought processes verbally, with remarkable success. Confusional states are most commonly produced by metabolic disturbances or by substance intoxication, but they can also be the result of cerebral tumors (directly or indirectly affecting brain structures that sustain vigilance) or of trauma.

A picture of a patient with aphasia should begin to emerge:

1. An aphasic produces some speech, or even abundant speech, which does not conform to the grammatical rules of the language being used. The errors include omission of words, such as conjunctions or prepositions (functor words), erroneous choice of words (substitution of the intended word for another that may or may not be related in sound or meaning), and disturbances of the relationship among words in a sentence (e.g., as expressed in word order). The rule that aphasic patients always produce some speech may not be true during the first hours or days of onset of disease. But even during a phase of speechlessness, most aphasic patients attempt to communicate by gesture or facial expression.

2. An aphasic often has difficulty in comprehending a purely verbal command (i.e., a verbal message given through auditory or visual means, without accompanying gestures, facial expressions, or meaningful emotional intonation). The errors of comprehension may range from an almost complete inability to understand any but the most elementary questions, to mild defects that surface when complex sentences are presented (e.g., sentences with double negatives or dependent clauses).

3. An aphasic is aware of self and environment, and when medically examined has an appropriate level of awareness. She or he is intent on communicating thoughts regarding her or his own condition and surroundings. Again, there may be exceptions, particularly in the first few hours after an acute brain lesion, when aphasic patients may appear inattentive and uninterested in communication. There may be patients, well into their chronic stages, in whom a depression will not permit an appropriate relation with examiners and surroundings. In general, however, as far as can

be determined given that the view of the world available to an aphasic is impoverished by lack of intact verbal processing, it is *not* the view of a confused, or demented, or psychotic patient. Accordingly, the appearance of the aphasic patient is not that of an alienated individual. More often than not, beyond the barrier of handicapped communication, the examiner of an aphasic patient can empathize with the subject. The student of aphasia should develop a keen sensitivity to this important aspect of aphasia.

Signs of Aphasia

Naming Disturbances and the Production of Paraphasias

At the core of language formulation lies the ability to select from the verbal lexicon a word that conveys the meaning of a given thought. The selection process is often automatic although we may deliberately search for the precise lexical item, a process designated as WORD FINDING. When word selection fails, the result is either the omission of the intended item or a substitution of an incorrect and unintended word. The latter is termed PARAPHASIA and probably is the central sign of aphasia. If an entire word is substituted, the paraphasia is called VERBAL or GLOBAL. If the incorrectly selected item belongs to the same semantic field (e.g., *chair* for *table*), it is termed a SEMANTIC paraphasia. Too many verbal paraphasias appearing in sentence after sentence give rise to JARGON SPEECH.

Paraphasias can be entirely novel words which do not exist in the lexicon of a given language (NEOLOGISTIC paraphasias). The mechanism for the formulation of the new word may be a succession of phoneme substitutions. When a single phoneme is substituted or added (e.g., *table* becomes *trable* or *fable*), the sign is known as a PHONEMIC or LITERAL paraphasia. Too many phonemic paraphasias produce an unintelligible neologism (and too many neologistic paraphasias become NEOLOGISTIC JARGON).

Paraphasias can appear in spontaneous speech or in a dialogue, on repetition of spoken sentences or on reading aloud, in naming tasks, and in writing; but they are generally absent in automatic speech (emotional exclamations, series of numbers, calendar sequences).

One of the most exciting new developments in aphasia research comes from the identification of patterns of word-finding defects related to varied domains of conceptual knowledge. Patients with naming defects are not impaired for all words that decode all categories of knowledge. On the contrary, the defects are more pronounced in some categories than in oth-

ers, and their seeming selectivity reveals some of the underpinnings of lexical organization (see A. Damasio, 1990; A. Damasio & Damasio, 1992; A. R. Damasio, Damasio, Tranel, & Brandt, 1990; A. R. Damasio & Tranel, 1993; H. Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996).

Disturbance of Fluency

Although the general characteristics of speech in aphasic patients are not always easily classifiable, they often fall into one of two categories: FLUENT or NONFLUENT. The designations can have slightly different meanings for different authors, but for most aphasiologists, fluent speech approximates normal speech in terms of the rate of word production, the length of each sentence, the melodic contour of the sentences, and the overall ease of the speaking act. In practical terms, it is usually measured by the longest continuous string of words that the patient produces in conversation. Fluent aphasic speech may be actually more abundant than normal speech. Nonfluent speech is the opposite: The rate is low, sentence length is short, the melodic contour is lost, the production is effortful, and there may be more pauses than actual words in a given time unit.

Judging the quality of articulation is a separate matter from judging fluency. Most patients with fluent speech have normal articulation, although some may have minor difficulties. Many patients with nonfluent speech also have perfect articulation, although some do not. As noted, the ability to articulate speech and the ability to formulate language are different. Even patients with severe nonfluent speech are able to produce perfectly articulated automatized verbal sequences (as in counting or in emotional exclamations).

A measure of fluency may help with clinical classification and provide a rough indication for the localization of lesion. Most patients with fluent aphasias have lesions located in the posterior aspect of the perisylvian region. Most patients with nonfluent aphasias have lesions located in the anterior aspect of the perisylvian region (Benson, 1967).

Disturbances of Repetition

A failure to repeat words or sentences is another hallmark of aphasia. The ability to repeat may be entirely lost, or may be marred by phonemic paraphasias or omissions of sounds and words. Repetition is impaired in most aphasias, and actually dominates the clinical presentation of conduction aphasia largely because other pronounced defects are lacking.

The impairment of repetition has major localization value. Its presence

places the lesion firmly in the perisylvian region of the dominant hemisphere. Repetition defects are notably absent in the transcortical aphasias and in the so-called anomic aphasias, whose correlated lesion is located outside the perisylvian ring. Patients with transcortical aphasias may actually repeat only too well, echoing the examiner's words immediately after they are pronounced, often with little or no comprehension of what they are parroting. Such a defect is called ECHOLALIA.

Disturbances of Auditory Comprehension

Auditory comprehension can be impaired to variable degrees. Some patients are able to participate in a colloquial conversation, giving appropriate verbal replies or indicating that they understand the content of the messages by nodding, pointing responses, facial expression, or gestures. Yet, confronted with laboratory tests, they may fail many items, especially when the question aims at specifics rather than generalities, and when the linguistic structure is complex rather than transparent. Other patients may be quite impaired even in a simple conversation, let alone in the laboratory tests.

Disturbances of Grammatical Processing

AGRAMMATISM is another important sign of aphasia. It refers to difficulty with generating the syntactical frames into which lexical selections must be placed, and to a defective utilization of grammatical morphemes. Grammatical morphemes include functor words (free grammatical morphemes) or the inflectional affixes that mark tense, aspect, or person when placed at a verb ending (bound grammatical morphemes). It used to be thought that only patients of the Broca type were agrammatic, but it is now clear that patients with other aphasia types, namely the commonly encountered Wernicke's aphasia, can be agrammatic too. Agrammatism is discussed at length elsewhere in this volume.

Disturbances of Reading and Writing

Reading comprehension can be disturbed in much the same way as auditory comprehension, although the two defects do not necessarily go together. For instance, patients with auditory comprehension defects usually have some reading impairment, but the proportion of those with both defects is small. On the other hand, impairment of reading can appear in pure form without impairment of auditory comprehension or writing. In

most cases of aphasia, however, reading, writing, and auditory comprehension are impaired together, although rarely to the same degree.

Apraxia

Many aphasic patients also present with apraxia, forms of which, from a practical and clinical standpoint, can be considered yet another sign of aphasia. APRAXIA may be defined as a disorder of the execution of learned movement that cannot be accounted for by weakness, incoordination, sensory loss, or impaired comprehension or attention to commands. From a theoretical point of view, however, it should be clear that apraxia can appear in isolation, without aphasia, and that its many varieties and mechanisms justify a separate entity status. The presence of apraxia should be investigated in all aphasic patients, as it may interfere with the performance of acts requested through verbal command. Students of aphasia should be aware of the fact that patients do not "complain" of apraxia and that, except for the extreme forms of ideational apraxia, the phenomenon is neither immediately disruptive to the patient's life nor evident to the examiner. The reader is referred to Geschwind (1975; A. Damasio & Geschwind, 1984; Geschwind & Galaburda, 1985) for a comprehensive view of the phenomenon.

Classifications of Aphasia

Classifications are a necessary evil. Attempting to review the classification systems of aphasia is probably foolhardy. The variety of criteria used over the past 100 years may disorient the reader at first. The diversity of the nomenclature is exasperating. The seeming conflict between systems that include as many as eight different varieties of aphasia and those that limit themselves to two or three is a source of puzzlement. Yet, the student of aphasia should realize that the diversity reflects the evolution of the science of the aphasias and the conflicts are more apparent than real—the systems derive from different points of view about the phenomena of aphasia. For instance, Weisenburg and McBride's (1935) classic designations of EXPRESSIVE, RECEPTIVE, and MIXED aphasia reflect a clinical vantage point. The terms in Luria's (1966) nomenclature—for example, EFFERENT and AFFERENT MOTOR, or DYNAMIC—reflect a physiological approach. On the other hand, Jakobson's (1964) description of CONTIGUITY (or combination) and SIMILARITY (or selection) defects draws on a psycholinguistic point of view. The systems do not conflict but rather complement each other. Be that as

it may, a modern researcher or clinician should have a working knowledge of the different classification systems, from Wernicke's (1874) to Geschwind's (1965). This should be complemented with a conversance in one of the modern classification systems: the proper definition of each of the categories, their anatomical and physiological significance, and their prognostic implications.

The system generally associated with the Boston school of aphasia is perhaps the most useful. It can be used in conjunction with most forms of laboratory and bedside assessment and does not necessarily require the use of the Boston Diagnostic Aphasia Examination (BDAE). The Boston classification comprises all of the frequently encountered aphasias for which there is an established and accepted anatomical correlation. The nomenclature utilizes a combination of eponyms, clinically descriptive terms, and physiologically based terms and is not especially mysterious (see Goodglass & Kaplan, 1972).

The following paragraphs contain descriptions of the aphasias most frequently encountered in clinical practice. I refer to them as "types" because each description corresponds to the averaged mental representation that an experienced observer forms, out of many comparable exemplars, bringing together signs that are salient over different epochs after the onset of aphasia. The reasons why signs cluster themselves in fairly distinct patterns are largely biological. Damage to certain neural units tends to produce, fairly consistently, a given sign of dysfunction. By the same token, the reason why the precise cluster of signs varies from individual to individual—a sign may be missing from the usual combination or some sign may be more or less pronounced than usual—is largely neuroanatomical. The precise lesion placement varies from individual to individual because of individual variations of normal neural and vascular anatomy, combined with variations of neuropathologic dynamics. Furthermore, there are individual variations in the assignment of components of normal psychological function to separate anatomical components of neural networks.

In spite of all of these sources of variance, the astonishing fact remains the consistency with which certain signs do cluster, in case after case. Consequently, the aphasia typology discussed here will continue to be useful to clinicians in sorting diagnostic and management issues and in communicating effectively among themselves. On the other hand, it is clear that aphasia types, or even finer-etched aphasia syndromes, are generally not helpful in establishing groups of patients for research, and are of little use as a basis for neurophysiological reasoning. Research purposes are best served by making individual signs the variables with which neuroanatomical information or the result of cognitive experiments are connected.

Major Aphasia Types

Wernicke's Aphasia

Wernicke's aphasia is perhaps the least controversial of the aphasia types. Speech is fluent and well articulated, with frequent paraphasias (both verbal and literal). Syntactic structure appears less disturbed than in Broca's aphasia, but it is reasonable to say that *both* Wernicke's and Broca's aphasics exhibit some form of agrammatism. Aural comprehension is defective. Repetition of words and sentences is also defective. In general, both reading and writing are disturbed.

Most patients may have no other evidence of neurological disease, as right hemiparesis is infrequent or transient; right visual field defects are not the rule. Thus, the diagnosis rests almost solely on the language signs and accuracy of the diagnosis is mandatory: For the unskilled examiner, a patient with acute Wernicke's aphasia may sound "confused," with the consequence that a psychiatric rather than neurological diagnostic approach may be taken. Even assuming that the mistake is eventually corrected, the delay can be disastrous.

Patients with Wernicke's aphasia are less easily frustrated than those with Broca's aphasia. Yet, a tendency for paranoid ideation is more evident in Wernicke patients than in Broca patients. It should be recalled that these are among the few neurological patients who can develop a major paranoid syndrome and become homicidal.

This complex syndrome, which combines both output and input disturbances, is also known as RECEPTIVE aphasia, from Weisenburg and McBride's classification (1935), and as SENSORY aphasia, as Wernicke himself called it (1874), with appreciable modesty but little physiological sense. Kleist (1934) aptly called it WORD DEAFNESS, but the term is rarely used; whereas Brain (1961) named it PURE WORD DEAFNESS, an inaccurate designation, as patients with Wernicke's aphasia are indeed word deaf but clearly not in pure form. (Patients with pure word deafness do exist, however; they are unable to understand speech and to repeat words but speak fluently and *without* paraphasias). Head (1926) called this type of aphasia SYNTACTIC aphasia, a rather useless designation.

Broca's Aphasia

The existence of Broca's aphasia is currently well established. Yet some of the major controversies in the history of aphasia have revolved around its nature and pathological correlation. The first patient described by Bro-

ca in 1861 did not have what came to be known as Broca's aphasia, and it is clear that the degree of involvement of Broca's area and of the surrounding frontal operculum produce considerably different degrees of aphasia (Mohr et al., 1978). What currently is called Broca's aphasia can be defined as the opposite of Wernicke's aphasia. The speech is nonfluent. There are few words, short sentences, and many intervening pauses. What words emerge are produced with labor and often with distorted sounds. The melodic contour is flat. Syntactical structure is more disturbed than in Wernicke's. The general appearance of speech is telegraphic, due both to the selective deletion of many functor words and to disturbances of canonical word order. On the other hand, aural comprehension is relatively intact in colloquial conversation, although formal testing often discloses a defective performance. Repetition of words and sentences is impaired. Broca's aphasia should be distinguished from APHEMIA, an articulatory disorder caused by generally small lesions underneath the motor cortices or in the vicinity of the basal ganglia (Schiff, Alexander, Naeser, & Galaburda, 1983).

Unlike patients with Wernicke's aphasia, the patient with Broca's aphasia invariably presents with a right-sided motor defect (often a complete hemiparesis more marked in the upper extremity and face). As a consequence, patients with Broca's aphasia are less vulnerable to misdiagnosis. Their presentation is clearly neurological. On the other hand, they are often depressed and may respond to testing failures with "catastrophic" reactions (sudden weeping and refusal to proceed with the examination) more frequently than do Wernicke's aphasics.

Broca's aphasia has also been known as EXPRESSIVE (Weisenburg & McBride, 1935) and MOTOR (Goldstein, 1948; Wernicke, 1874) aphasia. For a time it was refused the status of aphasia and instead was called ANARTHRIA (Marie, 1906) and, later, DYSARTHRIA (Bay, 1964). Head (1926) called it VERBAL aphasia.

Conduction Aphasia

The speech of conduction aphasics is fluent although usually less abundant than that of Wernicke's. Commonly there are minor defects in aural comprehension, although understanding of colloquial conversation is intact. The impairment in repetition of words and sentences dominates the picture. The defect takes many forms. Most commonly, patients repeat words with phonemic paraphasias, but often they will omit or substitute words, and they may fail to repeat anything at all if function words rather than nouns are requested. Comprehension of the defectively repeated sen-

tences is good. Similarly, patients comprehend the sentences that they read aloud with numerous paraphasias.

Conduction aphasics often have some accompanying motor signs (paresis of the right side of the face and of the right upper extremity), but recovery is good. The syndrome has been known as CENTRAL aphasia, Goldstein's (1948) curious designation, and as AFFERENT MOTOR APHASIA, Luria's term. Luria attempted to break down the condition, giving it a motor component (AFFERENT MOTOR) and an auditory one (ACOUSTIC AMNESIC). Kertesz (1979) proposed a comparable distinction (EFFERENT CONDUCTION and AFFERENT CONDUCTION).

Transcortical Sensory Aphasia

Patients with transcortical sensory aphasia (TSA) have fluent and paraphasic speech (global paraphasias predominate over phonemic) and a severe impairment in aural comprehension. Yet their repetition is intact (occasionally echolalic), setting them clearly apart from Wernicke's aphasics. The distinction is important because the localization of the lesion is different (see Chapter 3 on localization). This underscores the need to test repetition in every aphasic patient.

TRANSCORTICAL was the original designation of Goldstein, and it has held up well through the years, both for TSA and for transcortical motor aphasia, some cases of which Luria preferred to call DYNAMIC aphasia (Luria & Tsevtkova, 1968).

Transcortical Motor Aphasia

Patients with transcortical motor aphasia (TMA) have intact repetition, as do patients with TSA, and can have echolalia as well. But the speech is nonfluent and troubled by phonemic and global paraphasias, perseveration, and loss of connective words. Auditory comprehension is also impaired when tested formally, although patients can often carry on a simple conversation at bedside.

Patients with TMA should be distinguished from those with mutism on several counts. First, patients with TMA are inclined to communicate and do so within their verbal imitations. Patients with mutism do not and are as impoverished in nonverbal communication as in verbal communication. Secondly, the speech of TMA is clearly aphasic: there are unquestionable phonetic, lexical, and syntactical errors, whereas patients with mutism either produce no speech at all or utter a few short but linguistically correct sentences. Again, the distinction is important because the localization of the lesion is different.

Global Aphasia

As the name implies, global aphasics present with an almost complete loss of ability to comprehend or formulate verbal communication. Propositional speech may be reduced to a few words, and the remainder of verbal communication consists of emotional exclamations and serial utterances. Auditory comprehension is often reduced to a variable number of nouns and verbs, and the comprehension of functor words or of syntactically organized sentences is virtually negligible.

Hemiplegia accompanies most global aphasias but not all. In global aphasia without hemiplegia the defects are less pronounced and the recovery is better (Tranel, Biller, Damasio, Adams, & Cornell, 1987).

Anomic Aphasia

It is important to distinguish pure anomic aphasia from anomia as a sign of aphasia, as the latter is present in practically all aphasias. Anomic aphasia is characterized by a pervasive impairment of word finding, which contrasts with intact repetition and speech that is fluent, well articulated, and grammatically correct. The neuroanatomical basis of anomic aphasia is being elucidated. These patients have damage to language areas outside the perisylvian circle, largely within anterior and inferior left temporal regions (A. R. Damasio & Damasio, 1992; H. Damasio & Damasio, 1989; H. Damasio et al., 1996; Graff-Radford et al., 1990; Tranel, Damasio, & Damasio, 1988). Different entities and conceptual–lexical categories are impaired or spared in a dissociated manner (e.g., patients are better at naming entities that are manufactured than natural, and subgroupings within those broad classes reveal further dissociations). Such disparities, along with the theoretical formulations necessary to account for them, provide an important source of evidence for studies of lexical representation. The terms AMNESIC (amnestic) aphasia, NOMINAL aphasia and VERBAL AMNESIA are synonymous.

The signs of anomic aphasia can also be found in the so-called progressive aphasias. As the name indicates, progressive aphasias are conditions in which the language impairment appears gradually rather than acutely. Rather than being caused by a stroke or a head injury, which are the most frequent causes of standard acute aphasia, the progressive aphasias are caused by degenerative diseases. Examples are Pick's disease, spongiform encephalopathies, and even Alzheimer's disease.

In the most typical presentation, patients develop gradual difficulties with word retrieval. The grammatical class most frequently involved is nouns, and that frequent defect correlates well with a pronounced neu-

ropathological involvement of the left temporal region. In some instances the predominant defect may involve verbs, and the presentation may even begin with an impairment in the retrieval of word-forms for verbs rather than for nouns. In those cases the neuropathological compromise tends to be more pronounced in the left premotor and premotor regions than in the temporal lobe. In general, as the condition progresses, both grammatical classes come are affected.

It should be noted that patients with progressive aphasia may develop cognitive impairments outside the language realm. In those circumstances the aphasia becomes part of a dementia syndrome.

Alexia with Agraphia

Alexia with agraphia is a true rarity. More often than not, patients with both alexia and agraphia have signs of Wernicke's aphasia or transcortical sensory aphasia. In the absence of aphasia, they generally have signs of parietal lobe dysfunction. The diagnosis of alexia with agraphia only applies when the disturbances of reading and writing predominate over the aphasic or parietal symptomatology. The fact that this syndrome can be associated with impaired as well as intact repetition, and with a greater or smaller extent of accompanying signs, suggests that a large segment of parietal and temporal lobe structures, cortical and subcortical, is engaged in the complex processes of reading and writing. The anatomical significance of this is considerably smaller than that of alexia *without* agraphia (pure alexia).

Alexia without Agraphia (Pure Alexia)

As the designation implies, patients presenting alexia with agraphia become unable to read while they continue to be able to write, spontaneously or to dictation. Many such patients can also copy writing, although they do so with difficulty. Speech, auditory comprehension, and repetition are intact. Oral spelling of words (or its converse, the construction of words spelled orally) is normal. Reading in the tactile mode is also normal. Whatever visual reading the patient can do is of single letters. This often allows the patient to read aloud the letters of a word, one by one, and then reconstitute the word from the spelled out components.

Although neither writing nor oral language impairments are present, most patients have some form of accompanying impairment of visual function (see A. R. Damasio & Damasio, 1983, for review). It can be a right homonymous hemianopia (the field of vision to the right of the vertical median is blind) or a right hemiachromatopsia (loss of color perception with-

out true blindness in the right hemifield). Most patients also have color anomia, a disturbance of naming colors with otherwise normal color perception. Some present with optic ataxia, a disturbance in the visual guidance of hand movements.

First described by Dejerine (1892), the syndrome was long forgotten and even denied, but it was revived by Geschwind (1965), who used it as a cornerstone for his theory of disconnection syndromes.

Pure Word Deafness

Patients with pure word deafness have a profound loss of auditory comprehension and a complete impairment of repetition. Yet they produce normal fluent speech, mostly without paraphasias. It could be argued that pure word deafness, like pure alexia, is not a true aphasia, because language formulation itself is not affected. From a physiopathological standpoint, both conditions reflect the inability of verbal information to reach structures capable of processing it into meaning. Inner language operations as well as exteriorization of well-formulated language remain intact. There are reasons, however, why the two conditions should be discussed along with the aphasias. First, they resemble aphasias from the standpoint of the communication impairment they produce. Second, the anatomical and physiological knowledge derived from studying these two "input" disorders has contributed importantly to the understanding of the aphasias.

"Atypical" Aphasias

A considerable number of cases of aphasia fail to conform to any of the types described here. This happens for a variety of neurobiological, neuropsychological, and cultural reasons. Perhaps the most frequently encountered atypical exemplars are ascribed to a so-called nonstandard cerebral dominance disposition (right cerebral dominance for language in a left-hander or right-hander, or "ambidominance" in a left-hander). However, another important source of "atypical" aphasias is damage in a noncortical sector of language networks. The lesion can be located in the deep nuclear gray masses (basal ganglia or thalamus) and may involve white matter in the vicinity (e.g., the anterior limb of the internal capsule). As an example, aphasia can arise after nonhemorrhagic infarction, which damages the left head of the caudate and the anterior limb of the internal capsule (A. R. Damasio, Damasio, Rizzo, Varney, & Gersh, 1982; H. Damasio, Eslinger, & Adams, 1984; Naeser et al., 1982). Damage to the left thalamus from either hemorrhagic (Alexander & Lo Verme, 1980; Hier & Mohr, 1975)

or nonhemorrhagic infarction (Graff-Radford, Damasio, Yamada, Eslinger, & Damasio, 1985) also causes aphasia.

Acknowledgment

This work was supported by NINCDS Grant PO1 NS19632.

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3

Neuroanatomical Correlates of the Aphasias

HANNA DAMASIO

This chapter provides an overview of the neuroanatomical correlates of the major aphasia types. Discussions on the physiopathology of aphasia signs and types and the neural basis of language processes are outside the scope of this chapter, although information on the neuroanatomical correlates of the aphasias is critical to the experimental work on the neural underpinnings of normal and abnormal language. This text complements that of Chapter 2 regarding types and signs of aphasia and makes use of the same theoretical and practical framework (see A. R. Damasio, 1989a, 1989b, 1989c; H. Damasio & Damasio, 1989). All of the cautions expressed in Chapter 2 about syndromes, types, and signs of aphasia apply here as well. Most importantly, the description of consistent neural correlates and of reliable lesion localization provided by certain signs should not be confused with an attribution of specific functions to the areas damaged by a lesion.

The history of cerebral localization of the aphasias begins with Broca's discovery of a relation between a disturbance of language and damage to the lower posterolateral aspect of the left frontal lobe (Broca, 1861a, 1861b). Broca's work called attention to the asymmetry of the brain in relation to language, in what became the first modern study in cerebral dominance, and prepared the groundwork for further correlations between acquired aphasia and cerebral lesions. The next historical step came with Wernicke's report (1874) of the association between the symptom complex of Wernicke's aphasia and damage to the posterior aspect of the first left temporal gyrus. This finding strengthened the notion of left cerebral dominance

for language, and helped establish the concept that varied pathological behaviors could be related to different brain lesions. Wernicke proceeded to predict the anatomical lesion responsible for a third aphasia type, CONDUCTION APHASIA. The lesion, he thought, would fall between those found in Wernicke's and Broca's aphasias, most probably in the insular region. It is currently apparent that, in essence, his prediction was correct.

The next important step came with Déjérine's descriptions of ALEXIA and AGRAPHIA (1891) and ALEXIA WITHOUT AGRAPHIA (1892). His studies established a connection between written language and specific brain regions, much as Broca's and Wernicke's had established similar links for aural languages. Alexia with agraphia was associated with damage to the left parietal lobe in structures interposed between Wernicke's area and the visual association cortices. On the other hand, alexia without agraphia was found to follow a lesion in the left occipital lobe which damaged visual association cortices exclusively. Déjérine proposed that such a lesion prevented access of visual information to the very structures of the parietal and temporal lobes with which impairments of reading, writing, and aural comprehension had previously been associated. He believed the lesion was strategically located at a crossroads of visual information traffic. The lesion interrupted the flow of information from the right to the left visual cortices by means of damaging either the corpus callosum proper or its outflow (the forceps major). It also severed crucial connections between the left visual cortex itself and the left parietal and temporal language cortex.

Another important step in the anatomical mapping of the aphasias should be noted. Wernicke, and later Goldstein (cf. Goldstein, 1948), reported on the appearance of peculiar forms of aphasia associated with lesions different from the ones described before then and distinguished by a lack of impediment in verbal repetition. These were designated as TRANSCORTICAL MOTOR aphasia and TRANSCORTICAL SENSORY aphasia. The anatomical loci of the associated lesions were, respectively, anterior and posterior to the loci associated with the other varieties of aphasia. For transcortical motor aphasia, the lesion was in the frontal lobe structures rostral (anterior) to Broca's area. For transcortical sensory aphasia, damage was found in parietal and occipitotemporal structures caudal (posterior) to Wernicke's area. Figure 3.1 illustrates the localization of these fundamental syndromes of aphasia.

During the decades that followed, knowledge of aphasia localization was consolidated. Numerous case reports of the principal types of aphasia, accompanied by more or less expert descriptions of clinical symptomatology, confirmed the valuable discoveries of the founding fathers of aphasiology. Occasionally such case reports gave rise to conflicting evidence, but rarely if ever did such conflicts shatter the anatomical foundations provided by Broca, Wernicke, Déjérine, and Goldstein. On the

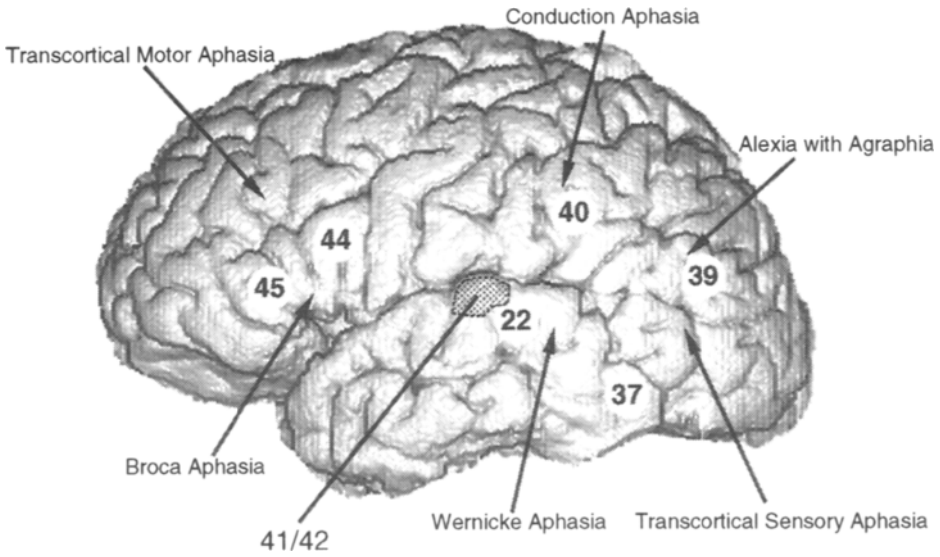

FIGURE 3.1. *Lateral view of the left hemisphere of a normal adult brain using thin continguous MR slices and Brainvox. Brodmann's areas 44 and 45 correspond to the classic Broca's area, and area 22 to Wernicke's area. Areas 41 and 42 correspond to the primary auditory cortex; these are located in the depth of the sylvian fissure and cannot be seen in a lateral view of the brain. Area 40 is the supramarginal gyrus; area 39 is the angular gyrus. Area 37, principally located in the posterior sector of the second and third temporal gyrus, does not have correspondence in gyral nomenclature.*

contrary, the mapping of the earlier days has been refined, rendering knowledge on the anatomical correlates of the aphasias ever more complex but along the same fundamental lines.

In 1965, Geschwind drew on this remarkable body of morphological evidence to interpret the aphasias and associated disorders in modern anatomical and physiological terms. In the years that followed, stimulated by Geschwind's seminal monograph, researchers reassessed the anatomical correlation of the various aphasic syndromes. New tools were also added: The radionuclide brain scan made its appearance, permitting new anatomical insights, and the Boston Aphasia Research Center's classification categories became widely accepted, making comparison of cases from different centers somewhat easier. The notion of fluency as a useful variable for correlation was also introduced. Benson (1967) used radionuclide brain scans to demonstrate a consistent association between nonfluent speech and prerolandic lesions. The nonfluent aphasias were those of the Broca and transcortical motor types. Within the limits of brain scan resolution, they correlated as expected with left frontal lobe lesions. The fluent aphasias included Wernicke's, conduction, and transcortical sensory,

which tended to cluster in the posterior left quadrant. For a variety of reasons, some exceptions to this pattern were found; however, the adherence to this pattern was more impressive than the departure from it. Only one partially conflicting study came to light (Karis & Horenstein, 1976), whereas a more extensive study corroborated Benson's results (Kertesz, Lesk, & McCabe, 1977).

The advent of computerized tomography (CT) in 1973 changed the panorama of the anatomical study of higher behavior and cognition in humans and was especially beneficial for the field of aphasia. It provided the possibility of studying with considerable anatomical detail not only a large variety of cerebral lesions but also the surrounding intact cerebral tissue. The first study of CT correlations with aphasia came from Naeser and Hayward (1978). Their localization of Broca's, Wernicke's, conduction, and global aphasias conformed to the classical localizations. Kertesz, Harlock, and Coates (1979) replicated Naeser and Hayward's findings, adding data on the differences produced by acute and chronic stages of aphasia. Later, by establishing a correspondence between gross brain anatomy and Brodmann's cytoarchitectonic map, other authors attempted to gain further anatomical information from CT studies with specifically developed templates of CT cuts (Basso, Lecours, Moraschini, & Vanier, 1985; H. Damasio, 1983, 1989; H. Damasio & Damasio, 1989; Gado, Hanaway, & Frank, 1979; Hayward, Naeser, & Zatz, 1977; Kertesz et al., 1979; Luzzatti, Scotti, & Gattoni, 1979; Matsui & Hirano, 1978; Mazzocchi & Vignolo, 1979; Poeck, De Bleser, & von Keyerlingk, 1984). By using bony structures and the ventricular system as landmarks, researchers plotted the lesions seen in the CT scan into the best-fitting templates and then read them as a three-dimensional reconstruction.

In the 1980s the advent of magnetic resonance (MR) improved the detection of structural abnormalities in the living human brain and permitted an unprecedented detail of anatomical characterization in vivo (H. Damasio, 1989; H. Damasio & Damasio, 1989). More recently, improved techniques for visualizing the information obtained with MR imaging—the three-dimensional reconstruction of thin-cut MR images (e.g., H. Damasio & Frank, 1992; Frank, Damasio, & Grabowski, 1997)—has allowed even greater detail in the descriptions of lesion sites. The new techniques make possible the exploration of the living brain with much the same detail permitted by autopsy, but with several added advantages. First, the brain can be "sliced" and "resliced" in whatever incidence is deemed more appropriate to the study of a particular lesion. Often this means comparing different sets of slices at the same time on the computer screen (e.g., see H. Damasio, 1995). Furthermore, given that the MR images are obtained at the time of the insult or, preferably, in the chronic stage that

follows, for instance, an infarct, the subjects are available for additional testing, which can be directed to specific questions related to the site of the lesion or to the disruption of a particular network that is presumed to function in a specific neuropsychological task.

These techniques, along with modern functional imaging techniques such as positron emission tomography (PET) and functional magnetic resonance (fMR), have strengthened beyond expectations the potential to investigate issues related to language processing (H. Damasio, Rezai, Eslinger, Kirchner, & Van Gilder, 1986; Habib, Démonet, & Frackowiack, 1996; Martin, Wiggs, Ungerleider, & Haxby, 1996; Petersen, Fox, Posner, Minton, & Raichle, 1988; Wise et al., 1991). Moreover, the enhanced lesion method and the functional imaging methods can be combined to investigate the same hypotheses, offering an especially powerful approach (see, e.g., H. Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996).

Fluent Aphasias

Conduction Aphasia

The anatomical correlates of conduction aphasia have been documented with postmortem studies (Benson et al., 1973) and CT and MR scan studies (H. Damasio, 1989; H. Damasio & Damasio, 1980, 1989; Kertesz et al., 1979; Naeser & Hayward, 1978; Rubens & Selnes, 1986).

Conduction aphasia is associated with left perisylvian lesions involving the primary auditory cortex (areas 41 and 42), a portion of the surrounding association cortex (area 22), and to a variable degree the insula and its subcortical white matter as well as the supramarginal gyrus (area 40). Not all of these regions need to be damaged in order to produce this type of aphasia. In some cases without involvement of auditory and insular regions, the compromise of area 40 is extensive (H. Damasio, 1989). In others, the supramarginal gyrus may be completely spared and the damage limited to insula and auditory cortices (A. R. Damasio & Damasio, 1980) or even to the insula alone (Rubens & Selnes, 1986). It is important to note that the area of damage does not involve the posterior sector of the superior temporal gyrus or Wernicke's area (See Figures 3.2 and 3.3). The compromise of the insula found in conduction aphasia (A. R. Damasio & Damasio, 1980) is an intriguing pointer to the possible involvement of the insula in language processing. The region may cooperate with inferior parietal structures in the process of phonological assembly, and Dronkers (1996) connects it to speech articulation.

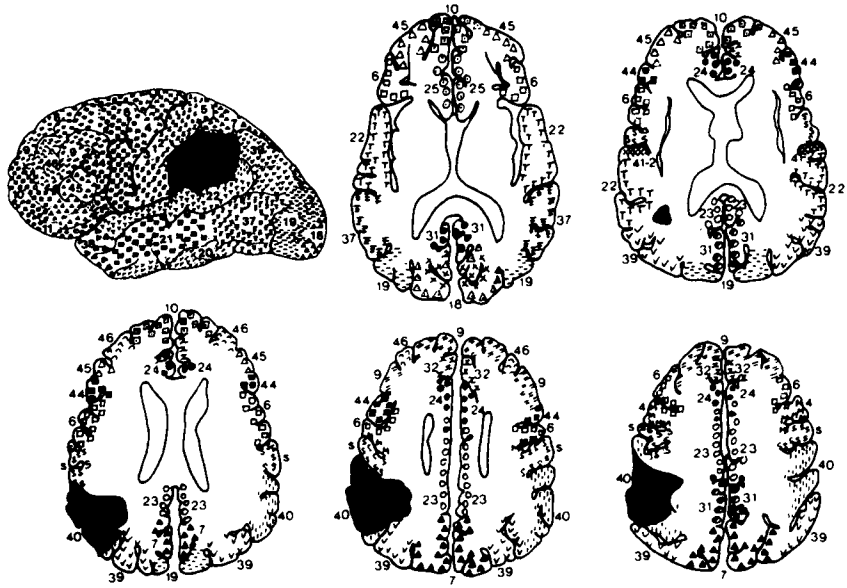

FIGURE 3.2. *Computerized tomographic template from a patient with conduction aphasia (LR0194). The lesion was limited to the left supramarginal gyrus.*

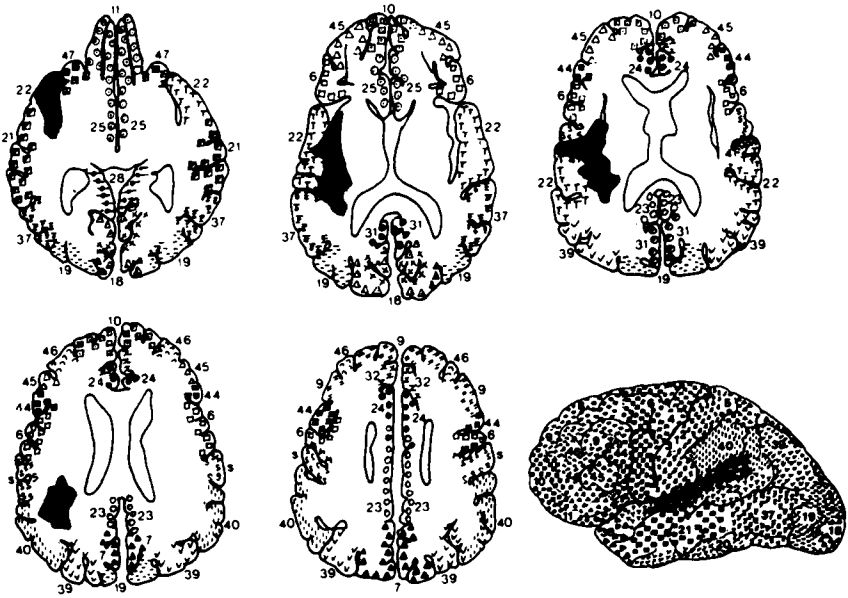

FIGURE 3.3. *Computerized tomographic template from a patient with conduction aphasia (RF089) in whom the lesion involved the left auditory cortex* and *the insula. The cortex of the supramarginal gyrus remained intact. The crosshatched area represents the area of damage that cannot be seen in the lateral surface of the brain.*

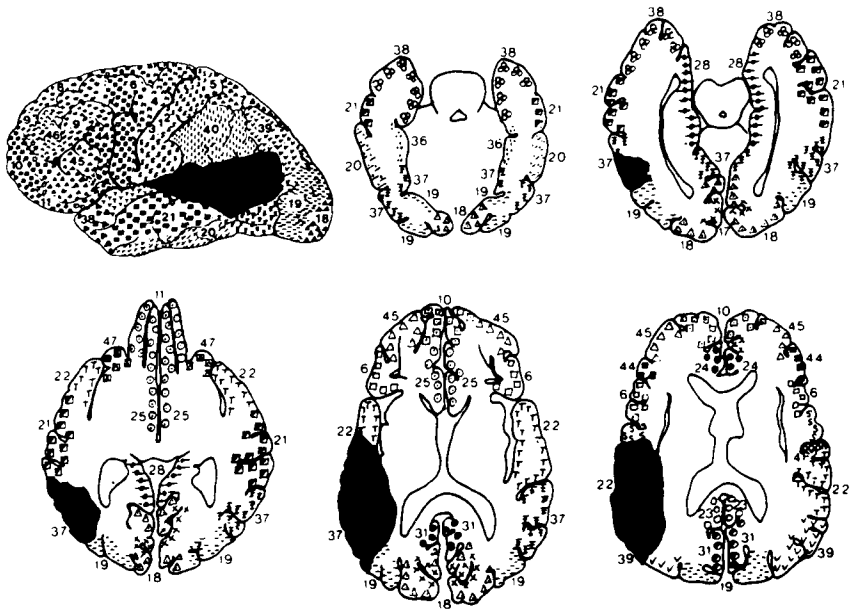

FIGURE 3.4. *Magnetic resonance template from a patient with Wernicke's aphasia (WG0988). The lesion involved the posterior sector of the left superior and middle temporal gyri but did not extend into the parietal lobe.*

Wernicke's Aphasia

The core of the lesions in Wernicke's aphasia maps to the posterior region of the left superior temporal gyrus. A typical example is shown in Figure 3.4. This has been the case since Wernicke's original description in 1886 and no significant changes have been introduced by more recent investigations (H. Damasio, 1989; Kertesz et al., 1979; Knopman, Selnes, Niccum, & Rubens, 1984; Mazzochi & Vignolo, 1979; Naeser & Hayward, 1978; Selnes, Knopman, Niccum, & Rubens, 1985; Selnes, Knopman, Niccum, Rubens, & Larson, 1983; Selnes, Niccum, Knopman, & Rubens, 1984). However, it also became clear that the lesions often extend into the second temporal gyrus and into the nearby parietal region (the lower segment of the supramarginal and angular gyri). Figure 3.5 shows an example of such a case. It is important to note that, as with conduction aphasia, the extent of damage in the different brain areas may vary and this may be related to the profile of linguistic disability. For instance, the overall severity of auditory comprehension deficit may be related to the extent of damage in the primary auditory cortices (areas 41 and 42), and the severity of visual naming deficit may well depend on the extent of damage to the middle and in-

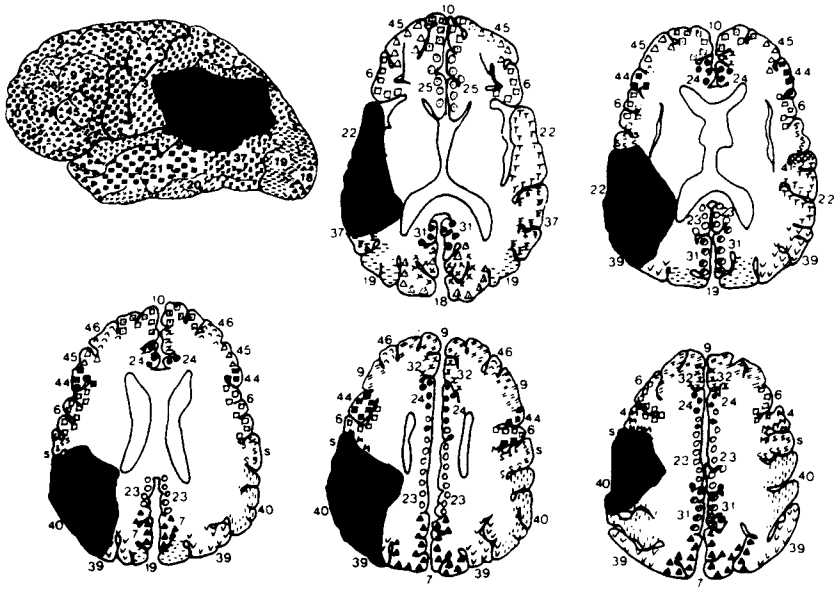

FIGURE 3.5. *Magnetic resonance template from a patient with Wernicke's aphasia (MS 0319). The lesion involved the left superior temporal gyrus and most of the inferior parietal lobule (both supramarginal and angular gyri).*

ferior temporal gyrus (areas 20, 21, and 37). Furthermore, paraphasic errors may be more or less prominent in certain categories of names (e.g., animals vs. manipulable manufactured tools), depending on whether the damage extends mostly into lower and anterior temporal regions or into the parietal region (for further examples, see H. Damasio, 1989; H. Damasio & Damasio, 1989).

Anomic Aphasia

As indicated in Chapter 2, the previously enigmatic anatomical correlates of anomic aphasia have been elucidated. Studies of numerous instances of anomic aphasia of both progressive (Graff-Radford et al., 1990; see also Silveri et al., 1997) and nonprogressive (A. R. Damasio, Damasio, Tranel, & Anderson, 1990; H. Damasio et al., 1996; see also Goodglass & Wingfield, 1997; Henaff Gonon Bruckert, & Michel, 1989) varieties have led to the conclusion that anomic aphasia requires damage to left temporal cortices located outside the traditional aphasia-producing territories (see Figure 3.6). When patients have difficulty retrieving words for concrete enti-

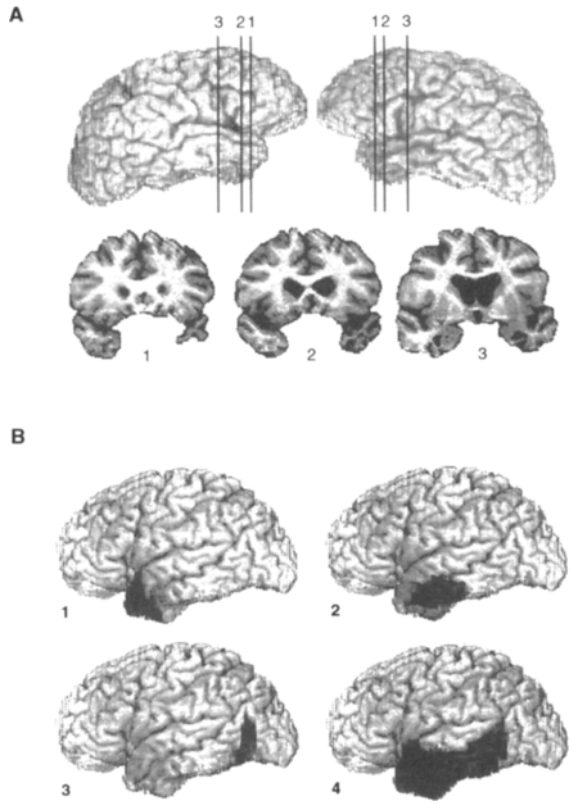

FIGURE 3.6. *(A) Thin-cut MR (using Brainvox) of a 73-year-old woman presenting with a progressive deficit in the retrieval of words for concrete entities with preservation of retrieval of words for actions. Note the marked atrophy of the left anterior temporal cortices. The position of the three coronal cuts is indicated by the vertical lines superimposed on the lateral views of the hemispheres. (B) Normal template brain onto which the lesions of four different subjects (MR, 1, 3, 4; CT, 2 were transferred with MAP-3 technique (Frank et al., 1997). Subject 1 had a left temporal lobectomy causing an inability to retrieve words for unique entities; subject 2 had a hemorrhagic infarct in the anterior inferotemporal region from an arteriovenous malformation causing a deficit in retrieval of words denoting animals; subject 3 had an infarct in the posterior inferotemporal region causing a deficit of retrieval of words for man-made manipulable tools; and subject 4, with extensive damage to all of the left inferotemporal region plus the temporal pole from herpes simplex encephalitis, had a severe deficit in word retrieval for all three categories of concrete entities. All subjects demonstrated normal concept retrieval for the concrete entities they could not name (see H. Damasio, Grabowski, Tranel, Hichwa, & Damasio, 1996; and Tranel, Damasio, & Damasio,1997).*

ties, in the absence of other linguistic deficits, they usually have lesions in the left temporal pole or in the middle or inferior temporal gyri. These lesions spare both the perisylvian language-related cortices and the struc-

tures in the mesial temporal region, namely, the parahippocampal gyrus and the hippocampus. Depending on the exact location of the damage in this large sector of the temporal lobe, the anomia may involve (a) words for unique entities when the lesion favors the temporal pole (area 38); (b) words for living entities (e.g., animals) when the lesion occupies the anterior and middle sector of the middle or inferior temporal gyri (areas 21 and 20); or (c) words for inanimate objects (e.g., manufactured manipulable objects such as tools and utensils) when the lesion involves the most posterior sector of the inferior temporal gyrus. A combination of deficits can be seen when the lesion compromises more than one of these subsectors. If, on the other hand, a progressive language deficit begins with the inability to conjure up words denoting actions, unaccompanied by defective retrieval of words for concrete entities, the degeneration process is more likely to have begun in the left frontal operculum rather than in the left temporal lobe.

Transcortical Sensory Aphasia

The neuroanatomical correlates of this type of aphasia only became clear after the advent of CT (H. Damasio, 1989; Kertesz et al., 1979). The posterior segment of area 22 in the superior temporal gyrus (or Wernicke's area) is never entirely damaged in this type of aphasia. The same applies to the primary auditory cortices (areas 41 and 42). The lesions can be seen in the posterior sector of the middle temporal gyrus (area 37) and in the angular gyrus (area 39), or in the white matter underlying these cortices (Figure 3.7).

In comparing the three main types of fluent aphasia, it is evident that there are some areas of anatomical overlap. Nevertheless, the core of each anatomical pattern is distinctive. Along a rostro–caudal axis, the anatomical core of Wernicke's aphasia seems to occupy the midsector, with involvement of the planum temporale (the posterior portion of area 22, contained in the sylvian fissure) plus some anterior and posterior extension. The anatomical core of conduction aphasia is more anterior and superior, often extending into the insula. Overlap with Wernicke's loci takes place in the anterior portions of area 22. The core for transcortical sensory aphasia occupies the more posterior sector, encompassing area 37 and extending into the visual association cortex and angular gyrus. The core of anomic aphasia occupies the middle and inferior temporal gyri and the temporal pole. Overlap takes place in the more posterior portion of area 22. Just as important, none of these three anatomical loci overlap with those for the nonfluent aphasias discussed in the next section.

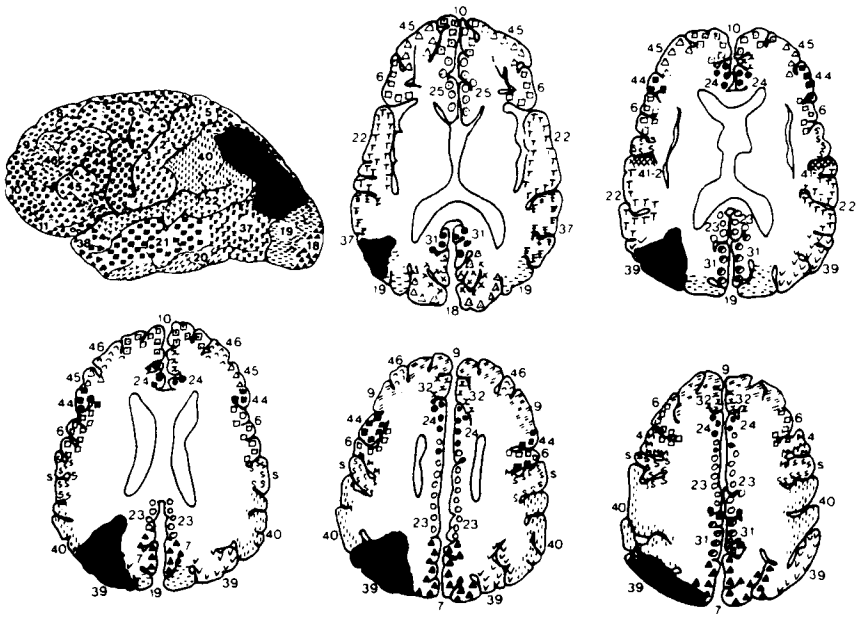

FIGURE 3.7. *Magnetic resonance template from a patient with transcortical sensory aphasia (LH1356). The lesion involved the left angular gyrus and the posterior sector of the supramarginal gyrus. Note that the temporal lobe is spared.*

Nonfluent Aphasias

Broca's Aphasia

The designation of Broca's aphasia, associated with lesions in the left frontal operculum, began with Paul Broca's description in 1861 (1861b) and has not survived without controversy (the most notorious debates occurred at the beginning of the century between Déjérine and Pierre Marie at the Société de Médicine in Paris [1908]). The advent of modern imaging methods has clarified several of the questions raised then, and even settled the 1908 debates (see Castaigne, Lhermitte, Signoret, & Abelanet, 1980). Parceling of both the clinical findings and the underlying deficits has also been attempted (H. Damasio, 1989; H. Damasio & Damasio, 1989; Kertesz et al., 1979; Mohr et al., 1978; Naeser & Hayward, 1978; Schiff, Alexander, Naeser, & Galaburda, 1983; Tonkonogy & Goodglass, 1981). In general, it is fair to say that lesions in Broca's aphasia may encompass not only the

frontal operculum (fields 44 and 45) but also premotor and motor regions immediately behind and above, in addition to extending into underlying white matter and basal ganglia as well as the insula. As might be expected, the extension of damage into these many different regions correlates with diverse accompanying deficits and also with the extent of recovery. Two cases illustrate this point.

Case 1 is that of a 40-year-old, right-handed woman with a thrombotic infarction. Initially she had a complete inability to utter single words or sentences but had remarkably intact aural comprehension and gestural communication. A right central facial paresis completed the neurological picture. Speech improved rapidly to a nonfluent halting discourse in which connectives were missing. Repetition of sentences was impaired and so was writing. The CT scan showed a small area of gray matter enhancement suggestive of a lesion in the left frontal lobe that would involve (a) areas 45 and 44 (Broca's area), (b) the portion of area 6 immediately above (the so-called Exner's area), and (c) the nearby facial motor region. Subcortical extension was minimal (see Figure 3.8). This patient continued to recover so well that 1 year later she had only minimal signs of impairment. Her speech is now slow but grammatically correct. An MRI obtained 7 years poststroke showed a stable area of encephalomalacia in precisely the same location, and emission tomography studies performed with a language-activating task demonstrated increased signal intensity both in the intact lower left frontal operculum and in the opposite hemisphere (see H. Damasio, 1989).

Case 2 represents the other extreme of Broca's aphasia. A 76-year-old, right-handed man suffered a thrombotic infarction. In the acute phase, he had minimal speech output, an inability to repeat words or sentences, a defect in aural comprehension, impaired reading and writing, and right-sided neglect. The CT scan of this man also showed a lesion in the left frontal lobe, but it included far more than Broca's area. Areas 45 and 44 were involved, but the lesion extended into area 6, as well as into the motor cortex and the anterior segment of the insula. Furthermore, it extended deep into the white matter, reaching close to the anterior horn of the left lateral ventricle and damaging part of the head of the caudate nucleus as well as part of the lenticular nucleus (see Figure 3.9). It should be noted, however, that regardless of being an extensive lesion, it did not reach posteriorly into the temporal lobe region; that is, it never overlapped areas related to the fluent aphasias. The evolution of this case was quite different from the previous one. Speech, as it emerged, was nonfluent, with the usual characteristics of Broca's aphasia. Recovery was modest compared with the first case.

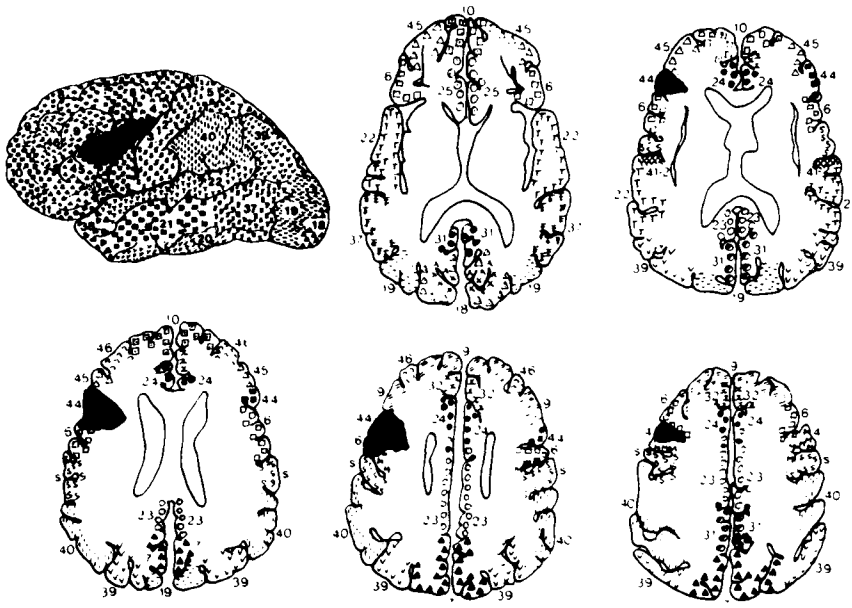

FIGURE 3.8. *Computerized tomographic template from a patient with Broca's aphasia (MW0018). The lesion involved only the superior sector of Broca's area (area 44) and the premotor region (area 6) immediately above it.*

Transcortical Motor Aphasia

The principal difference between transcortical motor aphasia and Broca's aphasia is in verbal repetition, which is possible in the former and impaired in the latter. Patients with transcortical motor aphasia often have echolalia in the setting of an otherwise nonfluent speech. Lesions are almost invariably located outside Broca's area, either anteriorly or superiorly, either deep in the left frontal substance or in the cortex. Figure 3.10 shows the MR template of such a case. The lesion is small and located just above the frontal operculum. It barely touches area 44 and extends superiorly into premotor and motor cortex. In other cases, it may be small and located deep in the white matter of the left frontal lobe, lying close to the anterior horn of the left lateral ventricle. In those cases, the lesion does not involve cortical areas but it most certainly disrupts connections between mesial structures of the frontal lobe, namely, the supplementary motor area, and structures of Broca's area and the motor area (see H. Damasio, 1989).

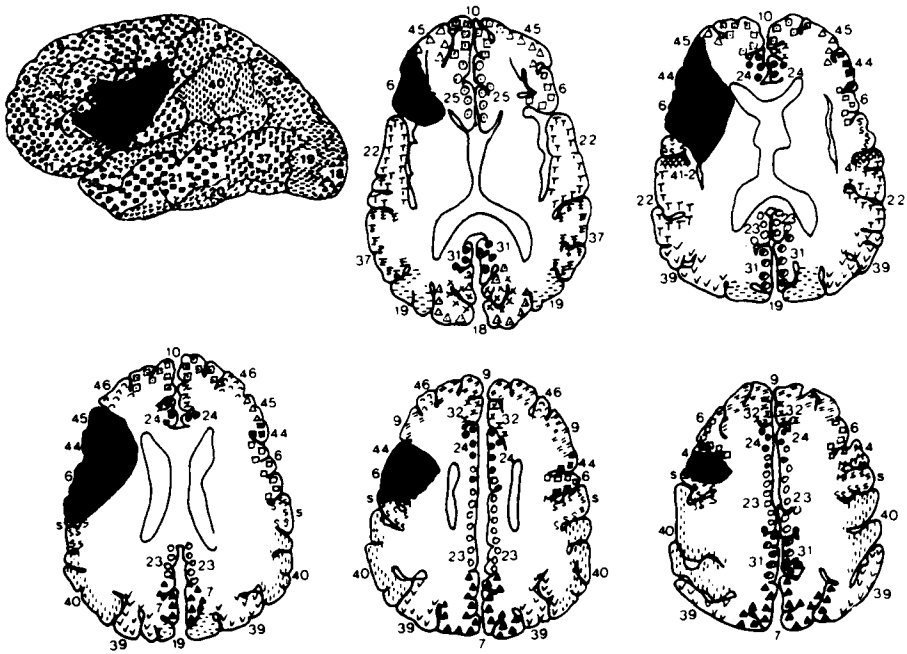

FIGURE 3.9. *Magnetic resonance template from a patient with Broca's aphasia (JP 1172). The lesion involved most of Broca's area (areas 44 and 45), and motor and premotor regions (areas 6 and 4), above and behind the frontal operculum, as well as the underlying white matter, the insula, and part of the basal ganglia.*

Mutism

Mutism is not, in the strict sense, a type of aphasia. In mutism, speech output is minimal or absent at all times, unlike the aphasias, in which it is only absent during the acute phase. However, the place of mutism in a discussion of localization of the aphasias is justified because mutism is often mistaken for transcortical motor aphasia and even for Broca's aphasia or aphemia. However, just as the clinical differences are clear, so are the anatomical correlates. Patients with mutism are aspontaneous both in relation to their nonexistent speech as well as in relation to other motor behaviors, for example, gestural communication and motor drive toward new stimuli. If stimulated enough, however, they can repeat words and sentences normally, and their comprehension of aural and written language is intact. The recovery of these patients is also different from that of patients with Broca's aphasia or transcortical motor aphasias: improvement is usually faster (in fact it can be sudden) and proceeds into gram-

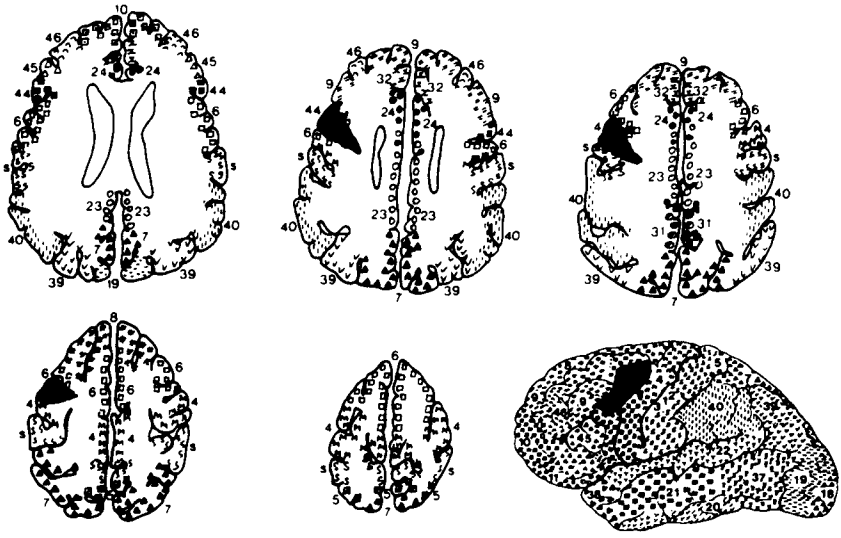

FIGURE 3.10. *Magnetic resonance template from a patient with transcortical motor aphasia (RW0680). The lesion involved left premotor and motor cortices, just above Broca's area.*

matically correct speech with normal fluency without an intervening agrammatical stage. True mutism is associated with lesions in the mesial aspect of the frontal lobe, which involves the supplementary motor area (the mesial portion of area 6), its connections, and the nearby anterior cingulate (area 24).

Figure 3.11 shows an example of a patient with mutism. The lesion appears in the very high portion of the left frontal lobe, above the level of the lateral ventricles. The core of damage involves the cortex and white matter immediately underneath in the mesial portion of area 6. The nearby anterior cingulate cortex and its connections are involved. In other cases, the damage can extend to involve motor areas mainly in their mesial aspect (corresponding to the cerebral representation of the lower limb), but also in their lateral portion (related to upper limb representation). This generally correlates with the presence or absence of paralysis of the foot and leg or of the arm. However, the lesions do not extend into the frontal regions, which were found to be affected in the aphasias.

Global Aphasia

The typical lesion in a patient with standard global aphasia involves the whole left perisylvian region, affecting all areas whose damage correlates

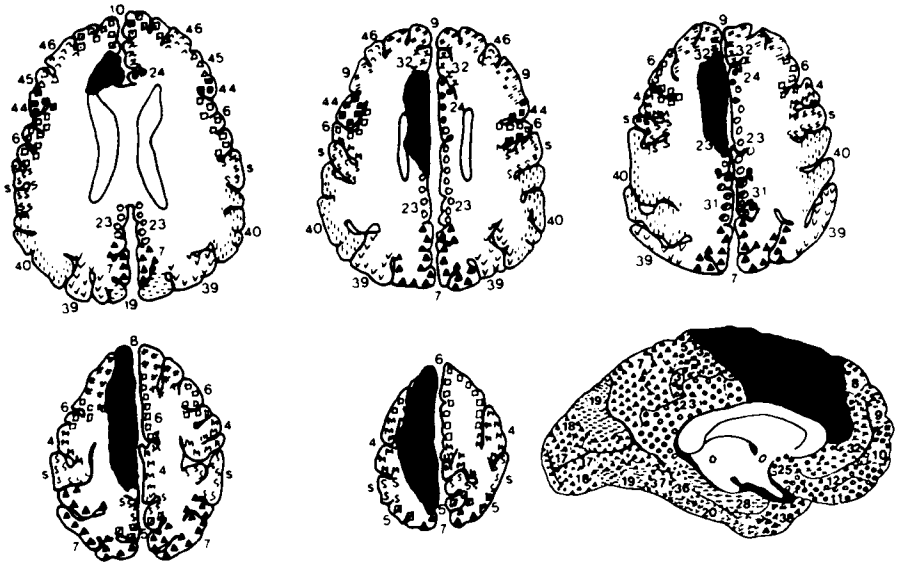

FIGURE 3.11 *Magnetic resonance template from a patient with akinetic mutism (DM0414). The lesion involved the left anterior cingulate gyrus, the supplementary motor area, and the mesial motor region.*

with the aphasias. Figure 3.12 shows the lesions of such a case. The damage is the result of an infarction in the territory of the middle cerebral artery. All of the perisylvian language areas are involved. The damage extends from 45 and 44 anteriorly to prefrontal cortices, as well as posteriorly to the insula, to auditory areas 41, 42, and 22, to area 40, and in part to areas 39 and 37. The motor and somatosensory areas 4, 3, 1, and 2 are also involved. The damage, however, is not limited to the cortex: the underlying white matter is involved as well as part of the lenticular and the caudate nuclei.

A similar clinical picture appears with the combination of two lesions in the left hemisphere, one anterior and one posterior (Tranel, Biller, Damasio, Adams, & Cornell, 1987). This is illustrated in Figure 3.13. In this case the anterior lesion involves the superior sector of area 44 as well as the underlying white matter, and extends into area 6 immediately above. The posterior lesion involves the angular gyrus together with the caudal sector of the supramarginal gyrus and the superior sector of area 37 and the white matter underlying these cortices. However, the insula, basal ganglia, and the temporal lobe are spared, as are the motor cortices. This patient had a global aphasia with severe impairment in all linguistic abilities, but did *not* have hemiplegia.

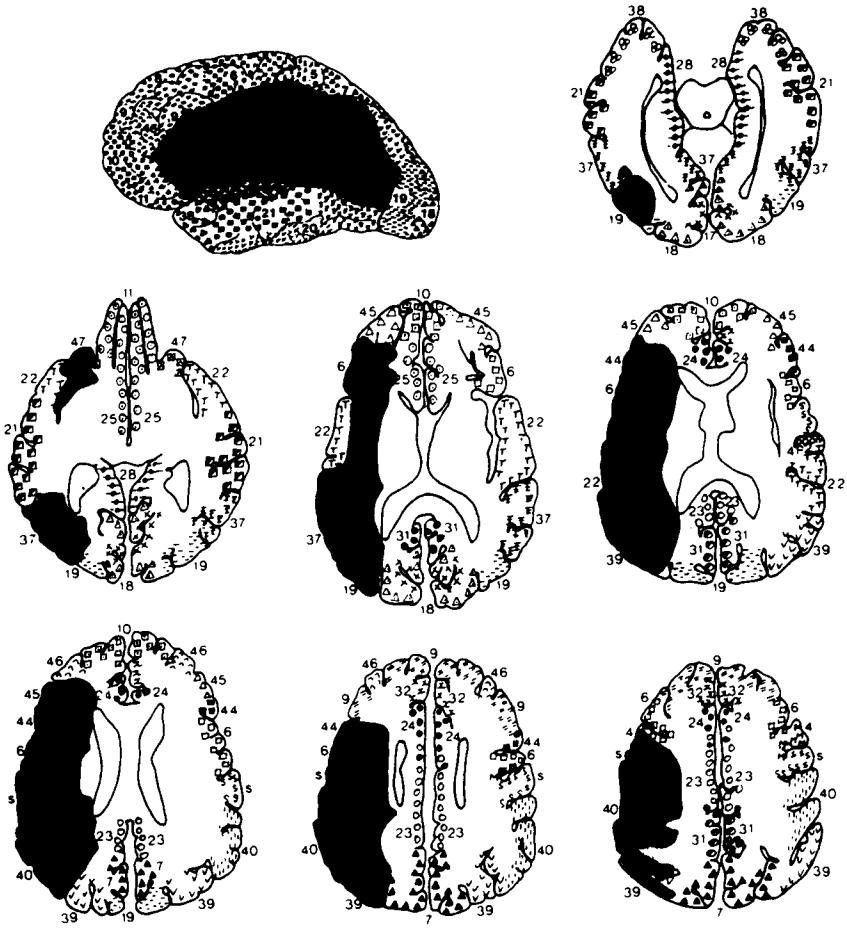

FIGURE 3.12. *Magnetic resonance template from a patient with global aphasia and hemiparesis (VD1266). The lesion involved most of the cortices and subcortical white matter supplied by the left middle cerebral artery.*

Another difference relates to recovery. In the case shown in Figure 3.13, the language-related cortices are not damaged to the same extent as in the case shown in Figure 3.12, and recovery is far superior. These patients may not recover to normal speech and language, but neither do they remain severe global aphasics.

One other anatomical pattern in global aphasia is that of a patient with a lesion in the left frontal operculum, underlying white matter, basal ganglia, insula, and even part of the parietal operculum, but it spares the tem-

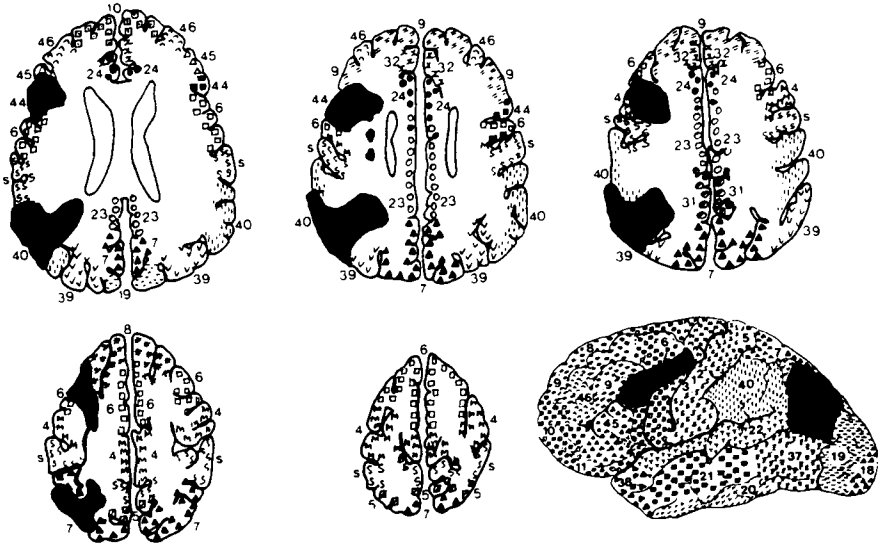

FIGURE 3.13. *Magnetic resonance template of a patient with global aphasia but without hemiparesis (JMcC0656). In this patient there were two lesions in the left hemisphere, one in the superior sector of the frontal operculum and premotor cortex immediately above, and another in the angular gyrus.*

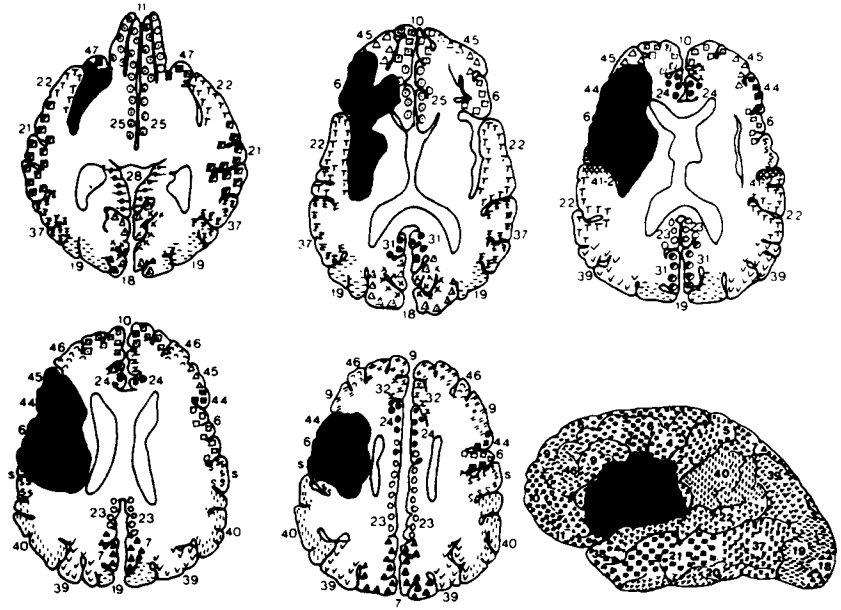

FIGURE 3.14. *Magnetic resonance template of a patient with global aphasia and hemiparesis (BD0638) in the acute stage, who later became a severe Broca's aphasic. Note that the lesion involved the left frontal operculum (areas 44 and 45), the premotor and motor cortices immediately behind and above Broca's area, as well as the insula and basal ganglia, but spared completely the temporal and parietal lesions.*

poral lobe. Such patients also tend to recover and, in the chronic stable state, come to resemble a Broca' aphasic (Figure 3.14).

Alexia without Agraphia (Pure Alexia)

Alexia without agraphia is not an aphasic disorder as such, inasmuch as speech output and aural comprehension are intact. The condition is associated with a remarkably consistent anatomical localization (A. Damasio & Damasio, 1983). Figure 3.15 shows a case with alexia without agraphia. The lesion extends from the occipital cortex deep into the white matter, reaching the left lateral ventricle at the level of the trigone and occipital horn (the paraventricular area). It involves the primary visual cortex (area 17) and part of the visual association cortices (areas 18 and 19), and extends into the mesial occipitotemporal junction involving mesial area 37 and the posterior sector of the parahippocampal gyrus. The corpus callosum is intact but the lesion disrupts interhemispheric connectional systems that course through the splenium of the corpus callosum, in the forceps major,

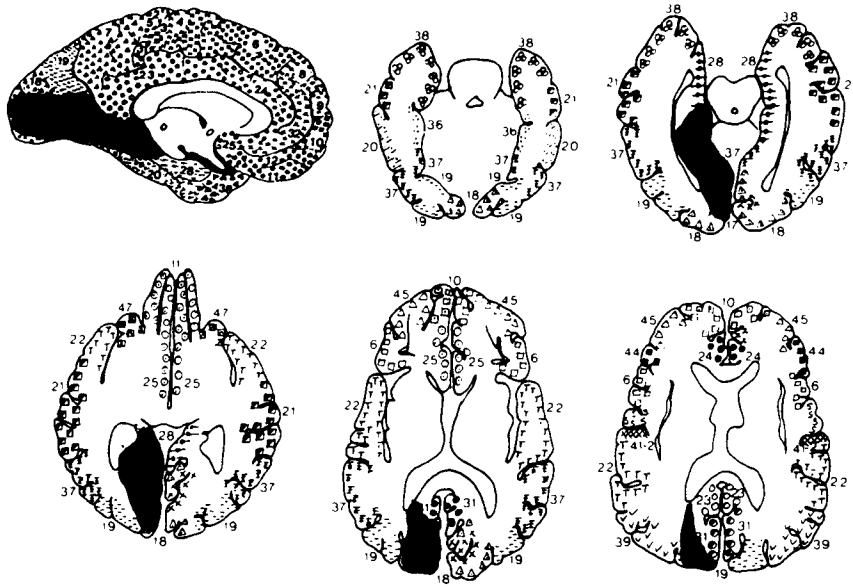

FIGURE 3.15. *Computerized tomographic template of a patient with alexia without agraphia (PA0321). The lesion involved the mesial sector of the left occipital lobe, the mesial occipitotemporal junction, and the white matter in the paraventricular region (where the forceps major courses), but spared the corpus callosum proper.*

and interlock the visual cortices. Thus, even if the corpus callosum proper does not show any damage, its outflow is compromised.

Atypical Aphasias

The advent of modern neuroimaging techniques led to the identification of the left basal ganglia as the lesion correlate for a group of aphasias known as "atypical," for lack of a better term. These aphasias are generally of the fluent type, in some way resembling Wernicke's aphasia. Yet, unlike typical fluent aphasias, there are also disturbances of articulation and, even more deviantly, a right hemiparesis is present (A. R. Damasio, Damasio, Rizzo, Varney, & Gersh, 1982; H. Damasio & Damasio, 1989; H. Damasio, Eslinger, & Adams, 1984; Naeser et al., 1982).

The lesions are located deep in the left hemisphere and invariably include portions of the caudate nucleus and putamen and the anterior limb of the internal capsule. They often occur in younger patients and are caused by embolic events, where an embolus becomes lodged in the proximal segment of the middle cerebral artery at the level of the lenticulostriate arteries, which supply the head of the caudate, the anterior limb of the internal capsule, and the lenticular nucleus. Another atypical aphasic syndrome, with a strong resemblance to transcortical sensory aphasia, can occur with infarcts in the left thalamus when the anterior nuclei are involved (Graff-Radford, Damasio, Yamada, Eslinger, & Damasio, 1985). In neither of these cases is there cortical damage in acute or chronic stages.

Conclusion

A large variety of acquired aphasic syndromes and of closely associated disturbances (mutism and pure alexia) can be correlated to relatively specific brain lesions located at varied sites in the left cerebral hemisphere. More than 100 years of study of anatomoclinical correlations, with autopsy material as well as CT and MR scans, has proven that in spite of the inevitable individual variability, the correlation between aphasia types and locus of cerebral damage is surprisingly consistent. Not surprisingly, there are exceptions, which can be found, in particular, in left-handed subjects, whose cerebral dominance for language is nonstandard and variable from subject to subject, and even in a minority of right-handed subjects who have right cerebral dominance for language (lesions within right hemispheres in these subjects produce "crossed" aphasias).

The value of these consistent correlations for clinical management is unquestionable. It should be clear, however, that the correlations per se pro-

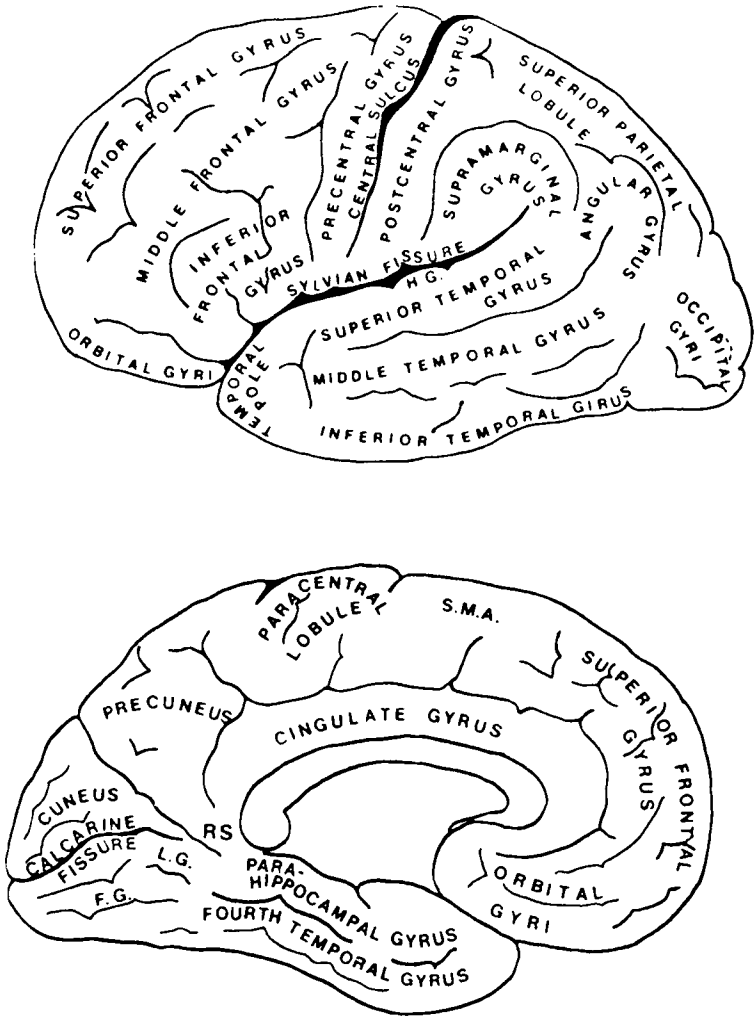

FIGURE 3.16. *Left hemisphere, lateral and mesial aspects, with identification of major fissures and gyri. Note that the insula cannot be seen on a lateral view because it is buried in the depth of the sylvian fissure, in its anterior portion, and is covered by the frontal operculum (the more posterior and inferior portion of the inferior frontal gyrus and the inferior portion of the precentral gyrus) and that Heschl gyri also cannot be seen because they occupy the superior surface of the superior temporal gyrus, buried inside the sylvian fissure. H.G., Heschl gyri (seen only in the superior surface of the superior temporal gyrus); L.G., lingual gyrus; F.G., fusiform gyrus; R.S., retrosplenial area; S.M.A., supplementary motor area.*

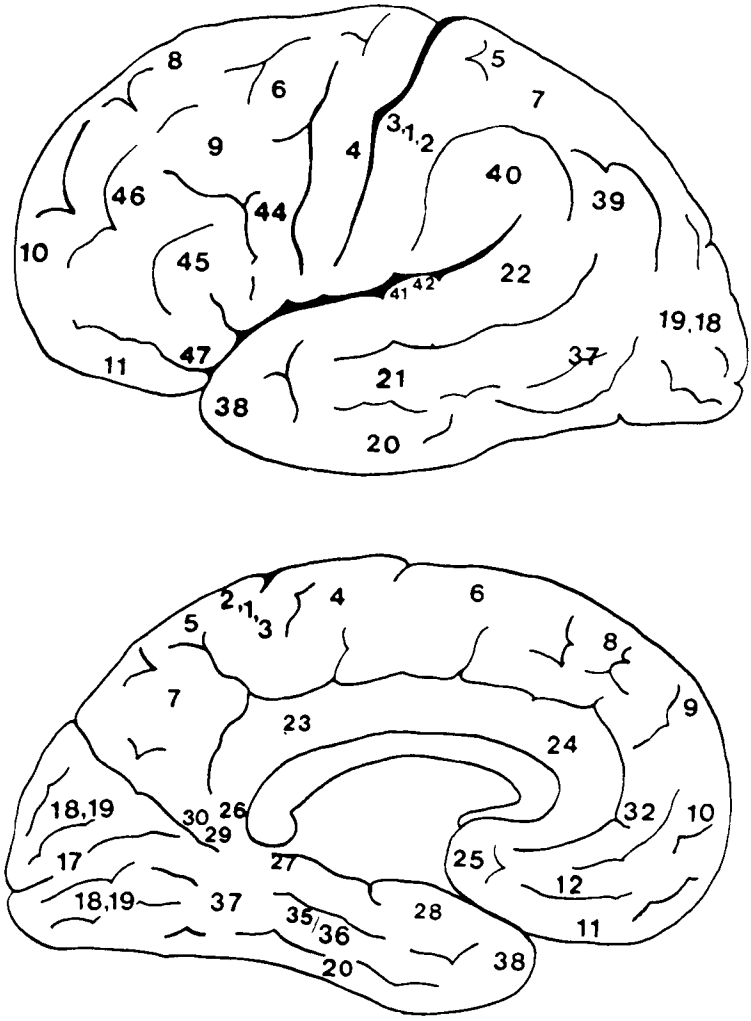

FIGURE 3.17. *Left hemisphere, lateral and mesial view with Brodmann's cytoarchitectonic nomenclatures. Note that areas 41 and 42, corresponding to Heschl gyri, are not seen in this view because they occupy the superior surface of the superior temporal gyrus, inside the sylvian fissure.*

vide only limited information about the neurobiological mechanisms of language, in health and in disease.

Figures 3.16 through 3.21 present different views of the brain with its areas, fissures, and gyri delineated.

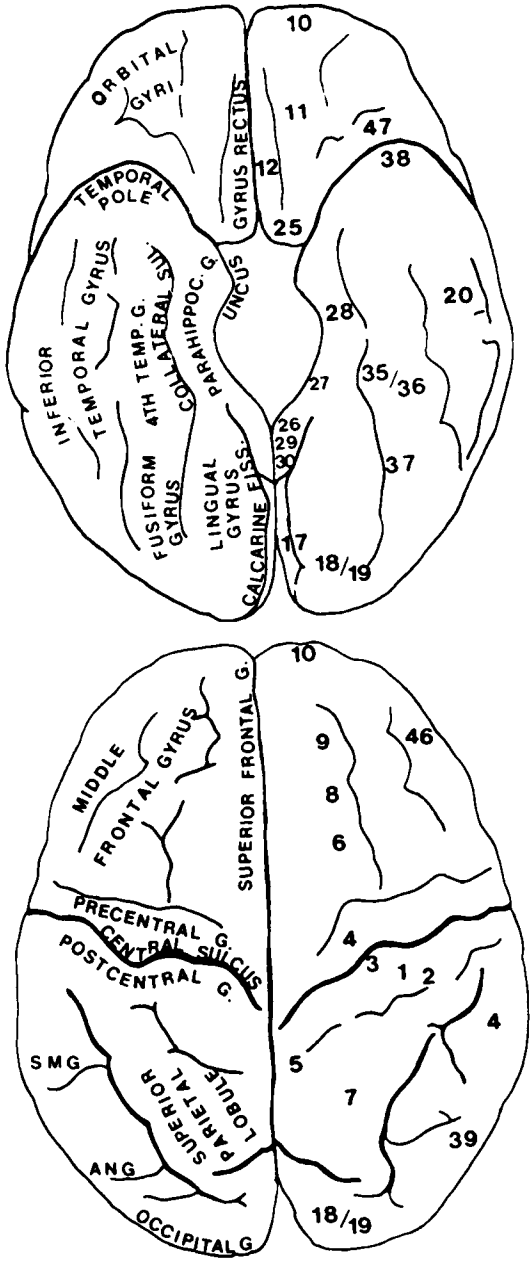

FIGURE 3.18. *Inferior and superior views of both hemispheres. On the left-hand side of each view, the major gyri and fissures are marked, and on the right-hand side Brodmann's cytoarchitectonic fields are shown.*

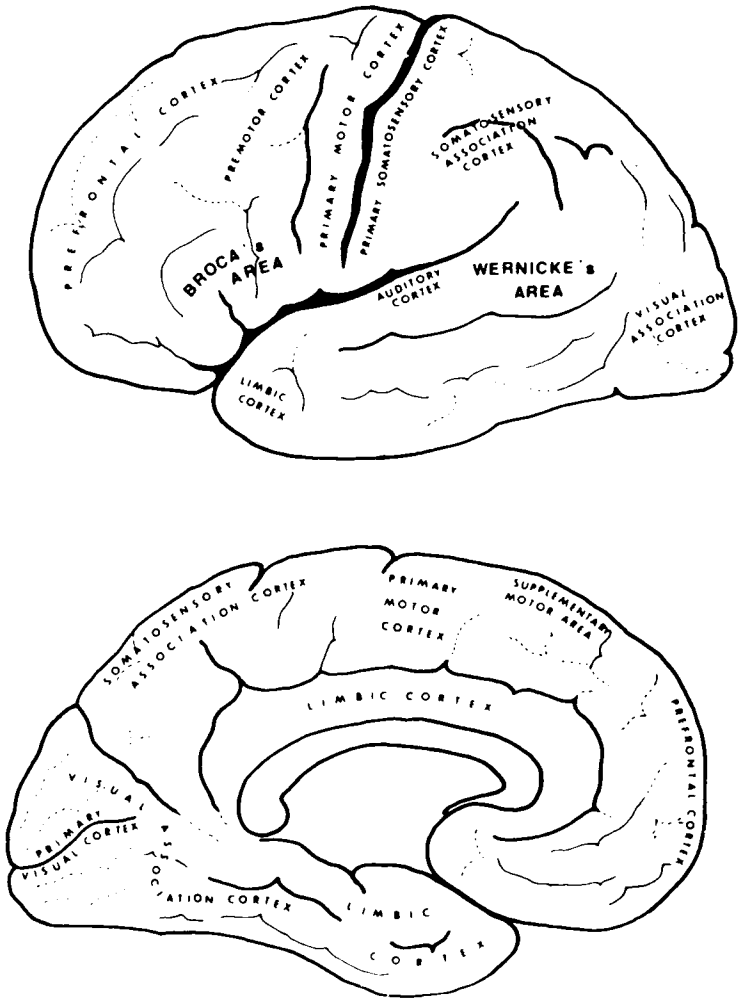

FIGURE 3.19. *Left hemisphere, lateral and mesial aspects, with identification of the major functional areas. Note that the auditory cortex occupies both the lateral aspect of the superior temporal gyrus and the superior aspect, inside the sylvian fissure (not seen in this lateral view) where the transverse temporal gyri (the primary auditory areas) and the planum temporale are located.*

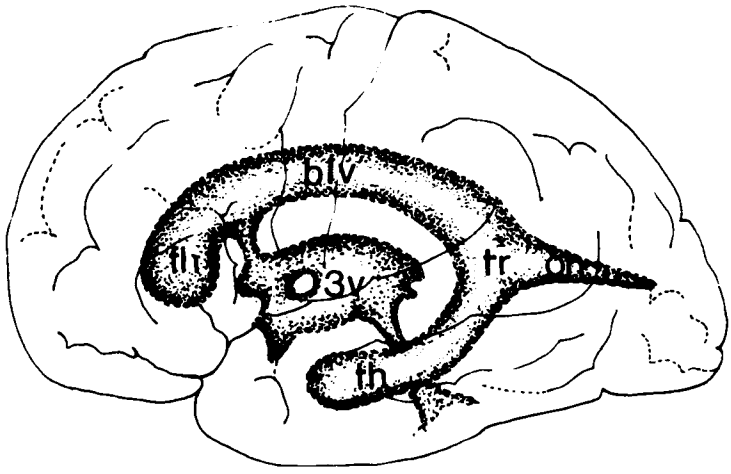

FIGURE 3.20. *Left hemisphere with the ventricular system. fh + blv + tr + oh + th = left lateral ventricle; fh, frontal horn; blv, body; tr, trigone; oh, occipital horn; th, temporal horn; 3v, third ventricle which connects with both lateral ventricles through the foramina of Monro (the left one is marked with an arrow) and continues caudally into the aqueduct (double arrow).*

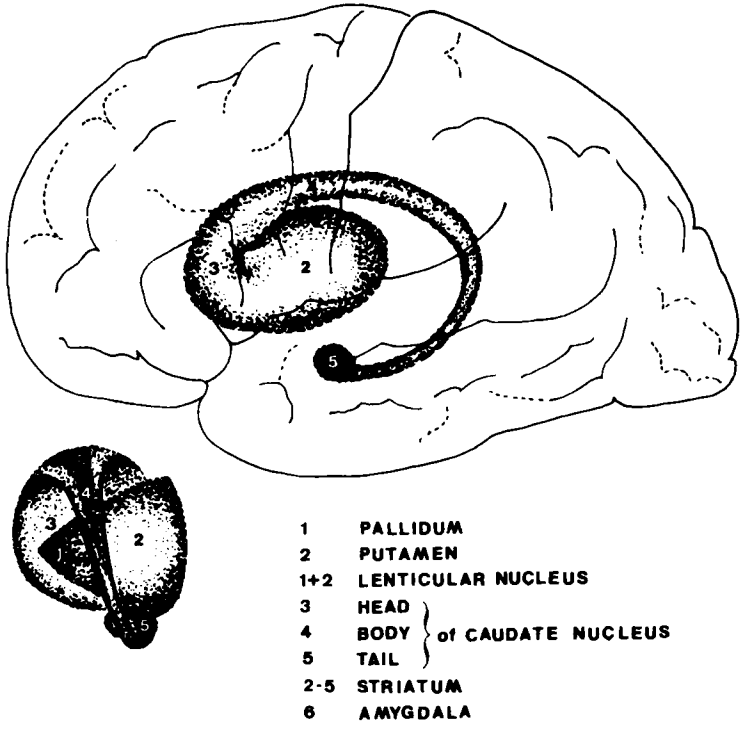

FIGURE 3.21. *Left hemisphere with the basal ganglia seen in lateral view. The insert shows the basal ganglia seen from the occipital pole.*

Acknowledgment

This work was supported by NINCDS Grant PO1 NS19632.

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4

Assessment of Aphasia

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Even the earliest records of medical knowledge refer to language disorders after brain damage (Benton, 1964). Accounts of simple clinical examinations were often included in such reports, but it was not until the second half of the nineteenth century, specifically since the publications of Broca (Joynt, 1964), that aphasia was explored more systematically. Case reports by Wernicke (1874/1908) and contemporaries contained detailed descriptions of examination procedures for individual patients. Whereas some of these examinations were probably standard procedure in certain hospitals, others were invented on the spot to explore individual features of a specific syndrome of aphasia. Understandably, these reports focused on the patient's specific disorder rather than on the examination procedure.

The clinical examination as developed in the late nineteenth century has been modified and augmented, but it has remained the essential tool of the clinical neurologist. Such examinations are exemplified in the writings of Jackson (1915) and Pick (1913). The standard repertoire of many clinical examinations includes such routine procedures as the Paper Test of Pierre Marie (1883), the Hand–Eye–Ear Test of Henry Head (1926), and Geschwind's (1971) "no ifs, ands, or buts" repetition as a simple task with high multiple demands on the patient's understanding, processing, and repetition ability.

The clinical examination has a number of disadvantages, which gradually led to the development of more generally applicable and standardized assessment instruments. Clinical examinations tend to vary from one place to another, both in content and in the way in which they are administered. What is considered abnormal remains up to the subjective judgment of the

clinician. Early attempts to produce a more standardized examination were published by Head (1926), who insisted on a detailed "clinical protocol." Another examination procedure was published by Froeschels, Dittrich, and Wilhelm (1932).

The first comprehensive battery of tests for aphasic patients was used by Weisenburg and McBride (1935) in a 5-year study of 60 aphasic patients. Schuell (Schuell, Jenkins, & Jiminez-Pabon, 1964) called this study a landmark because it was the first to use control subjects, to compare aphasic with nonaphasic brain-damaged subjects, and to use standardized methodology. Several other batteries were developed in the 1950s by Wepman (1951), Eisenson (1954, 1993), and Schuell (1955), partly as a result of intensive treatment efforts with World War II veterans. Benton (1964) reviewed the development of assessment procedures and noted the work done in various centers, criticizing that none of these procedures had been published in "usable form." The descriptions of procedures were insufficient, no standardization information was presented, and neither exact criteria for scoring nor detailed guides for interpretation were included. He compared the state of the art of aphasia testing with the "pre-Binet stage" in intelligence testing: "We are today where intelligence testing was in 1900" (p. 263).

The situation has changed in the 35 years since Benton's review. Instruments have been published that present detailed administration and scoring criteria and, at least in part, provide information on standardization and interpretation procedures. Benton (1994), although accepting limitations of the status quo, concluded that standardized tests have proven to be effective in monitoring the status of aphasia and in guiding rehabilitation strategies. Our review deals primarily with contemporary assessment techniques. Other reviews have been presented by Davis (1993), Kertesz (1979), Lezak (1995), and Spreen and Strauss (1998).

Purposes of Assessment and Testing

Assessment procedures vary greatly, depending on the examiner's goal. It is important to consider the purpose in evaluating and choosing specific instruments. Matching the assessment procedure to the patient requires a flexible and knowledgeable approach to assessment and testing. Four general types of evaluation purposes may be distinguished: (a) screening, (b) diagnostic assessment, (c) descriptive testing for rehabilitation and counseling, and (d) progress evaluation. A balanced approach using any of the four types when appropriate is empirically and clinically sound, and requires thoughtful decision-making by the practitioner.

Screening

Screening refers to a brief and cursory examination to detect the presence of a disorder. Three types of aphasia screening can be identified: (a) the bedside clinical examination; (b) screening tests per se; and (c) tests of specific aspects of language functioning that are sensitive to the presence of aphasia.

The BEDSIDE CLINICAL EXAMINATION is a clinical evaluation in the tradition of classical neurology (see Benson, 1979b; Strub & Black, 1985) and historically has been the primary method of assessing aphasia. It permits a brief and practical evaluation of language disorders, and remains a standard tool for many attending physicians, neurologists, and speech clinicians. The skilled clinician makes maximal use of his or her communicative interactions with the patient to rule out aphasia, establish a diagnosis, or reach the decision whether or not a more comprehensive assessment is warranted.

SCREENING TESTS reflect the once-popular application arising out of the clinical psychology of the 1950s of screening for the presence or absence of "organicity" (a loose term referring to any form of damage to the nervous system affecting psychological functions), particularly in high-risk groups and in conjunction with psychiatric evaluations. In relation to aphasia, some relatively brief and highly sensitive screening tests are available, but testing for the purpose of screening has lost its attractiveness and usefulness since the 1950s. One reason is that the accuracy of screening devices is limited, usually around 80% (Spreen & Benton, 1965). Another reason is that in clinical practice such cursory "detective work" is rarely necessary, as most patients are referred with an established clinical impression of aphasia, a known organic etiology, and neuroradiological localization. Finally, the information obtained from such instruments offers poor specificity (i.e., there are many reasons why a nonaphasic patient may fail a language screening test) and reveals little to indicate the severity of the detected aphasia. Thus, screening tests have been all but abandoned.

SPECIFIC-FUNCTION TESTS that explore a highly sensitive aspect of language functioning are easily incorporated into comprehensive evaluations, such as the neuropsychological assessment, to "screen" (explore) the nature of language functioning and to determine the need for additional and/or ancillary testing to better describe an observed deficit. The Token Test (TT) has proven itself to be the most durable and broadly used of such specific-function tests. The Boston Naming Test also has developed appeal as a stand-alone "screen" of language. Subtests of established aphasia batteries are also used, such as the Neurosensory Center Comprehensive Examination for Aphasia (NCCEA) Word Fluency ("F-A-S") task.

Diagnostic Assessment

Diagnostic assessment refers to an overall examination of a patient's language performance to arrive at both a diagnostic impression and a detailed description of areas of associated strengths and weaknesses. Because of the comprehensive nature of this examination, it is suitable for patients who are medically stable in the acute or postacute period of their recovery and for initial and/or follow-up evaluations of patients with subjective complaints of language problems. Diagnostic assessments tend to elicit brief samplings of performance in many different areas and may not necessarily be of use to the speech clinician interested in a detailed exploration of a particular problem. When the evaluation is confined to performances on language and aphasia-related tasks, the diagnostic impression may either refer to the type of aphasia present (i.e., classification) or go beyond the description of the functional deficit and arrive at speculative conclusions about the nature and location of the underlying brain disorder. Impressions from a broader cognitive (neuropsychological) evaluation include the type of aphasia as only one of a number of differentially diagnosable neurobehavioral syndromes, such as dementia, confusional states, amnestic syndrome, attentional disorder, and so forth, in order to determine the full spectrum of the patient's deficits (i.e., differential diagnosis).

Descriptive Assessment

For the purpose of rehabilitation and counseling, a descriptive assessment would seem to be the most sensible approach to take, choosing, when warranted, a variety of assessment procedures—a diagnostic battery, a functional communication scale, and a number of ancillary tests. Rehabilitation and counseling pose different questions than can be answered from a strictly diagnostic assessment. In particular, it is important to gain as much information as possible about areas of functional strength, as well as about the presence of deficits, as this allows better-reasoned advice on what treatment activities to pursue, what vocational options remain open to the patient, and the actual communicative level the patient can attain.

The assessment shifts from a strict testing situation to the observation of communicative behavior. A bridging of test contexts is necessary to compare the relation between specific language behaviors and deficits with the general ability to communicate. Descriptive assessment in the rehabilitation setting also involves (a) making predictions about recovery and response to treatment, and (b) measuring the ability of the patient to process, learn, and remember new material and, hence, to participate actively in and benefit from individualized treatment programs.

Progress Evaluation

Closely related to the descriptive assessment, progress evaluation permits an examination of spontaneous recovery when initial tests are repeated or when daily progress is charted in treatment settings. The clinician caring for the patient would like to be able to chart changes accurately over time rather than rely on subjective judgments or enthusiastic endorsements of the usefulness of therapy made by the patient or relatives. No formal tests have been developed specifically for this purpose, mainly because such progress assessments have to be tailor-made for each individual and her or his current level and range of deficit. Additionally, criteria need to be established for what will qualify as significant progress, as this likely would vary on a case-by-case basis depending on the patient's premorbid characteristics. Ad hoc assessments may be formulated for entire domains of language functioning or for the modification of specific language behaviors.

The ability of the patient to relearn or compensate for what he or she has lost should be part of the progress evaluation. This is a neglected aspect of aphasia assessment. Most tests merely measure the status quo but deliberately exclude any practice or learning during the testing procedure. Providing cues to the patient during diagnostic evaluations usually is seen as contributing to measurement error, and hence should be avoided. If a test were designed to provide information on the relearning capacity of the patient, the approach would systematically include a variety of short learning trials with different kinds of cues to determine whether the patient's language performance improves, at least within the immediate testing situation. It should be obvious that the inclusion of such procedures in the assessment of aphasia would dramatically change the usual form of testing, affect test reliability, and presumably add to the length of the test. Yet it is our impression that the benefit of such tests will outweigh the additional problems of test construction and validation, and that such tests will be a major focus of test development in the future.

Psycholinguistic Evaluation of Aphasic Language

Some studies have attempted to record and analyze conversational speech of aphasic patients in a setting that makes no specific demands on the patient. The main goal of such studies is not, however, an assessment of communicative abilities but a more detailed study from a psycholinguistic point of view. Studies of use and abuse of syntax, grammar, word

choice, frequency of word usage, pauses, and hesitations, speed of utterance, and so forth, can be conducted with such "free-speech" samples. The alternative approach is to focus on each aspect of psycholinguistic analysis individually and construct an experimental setting that allows an analysis of the types of errors produced by an aphasic patient.

Both the open-ended free speech and the experimental approach have been used extensively in aphasia research (Goodglass & Blumstein, 1973; Spreen, 1968). Insofar as these studies represent experiments rather than attempts to assess the aphasic patient's deficit, such studies are not reviewed here. However, some of these studies have led to conclusions about the nature of the deficit in specific types of aphasia and can be translated into suitable methods of assessment.

The first comprehensive psycholinguistic studies were conducted by Wepman and collaborators (Wepman & Jones, 1964). They calculated various linguistic parameters from stories told in response to looking at pictures, including grammatical form class usage, grammatical correctness, intelligibility, and word-finding problems. The studies involved complex calculations as well as judgments by linguistically trained researchers; they cannot be readily translated into more directly accessible forms of assessment.

The second major project was conducted by Howes and collaborators (Howes, 1964, 1966, 1967; Howes & Geschwind, 1964) and involved a detailed analysis of conversational speech of 5000 words each of more than 80 aphasic and nonaphasic speakers. The analyses concentrated on lexical diversity (i.e., the frequency of word usage in aphasic vs. nonaphasic speakers) and also distinguished between "fluent" and "nonfluent" speakers, who were viewed as similar to Wernicke's and anomic aphasics and to Broca's aphasics, respectively. Benson (1967) developed a simplified and clinically useful rating scale system based on the information from Howe's study with some additional rating dimensions.

A third comprehensive psycholinguistic analysis of conversational speech by Spreen and Wachal (1973) used a computer-scored interaction analysis of aspects of spoken language. Crockett (1972, 1976) designed 5-point rating scales for 17 characteristics of speech—including rate of speech, prosody, pronunciation, hesitation, and so on—to translate psycholinguistic speech characteristics into basic rating scale dimensions. Interrater agreement among five judges was satisfactory after some training, and after a carefully worded description of each characteristic was given.

Although Benson's (1967) and Crockett's (1972, 1976) ratings of psycholinguistic aspects of aphasic speech appeared to be quite successful within the limited scope of the research problems under investigation, further use of this approach has been limited. Several of the ratings have been

incorporated into the Boston Diagnostic Aphasia Examination (BDAE). Shewan (1988) developed a system to describe and quantify aphasic subjects' connected language in describing pictures, using phonologic, semantic, and syntactic components, as well as in general parameters of output (e.g., number of utterances, paraphasias). The Shewan Spontaneous Language Analysis (SSLA) also received some psychometric support, indicating adequate intrajudge, interjudge, and test–retest reliability, and validity based on clinical judgment of severity of connected language impairment. More recently, L. E. Nicholas and Brookshire (1995) have developed what they termed a Correct Information Unit (CIU) analysis of connected language. The CIU is a standardized, rule-based scoring system for which psychometric properties have been documented. Another detailed analysis of free conversational speech samples (Crockford & Lesser, 1994) stressed the analysis of editing elements (Schlenk, Huber, & Willmes, 1987) produced by the patient, amount and type of collaborative "repair" (Barnsley, 1987), and the proportion of conversational "load" (P. Hawkins, 1989; Miller, 1989) carried by the patient and the conversational partner, as well as unfilled and filled pauses, unsolicited repetitions, phonemic approximations, circumlocutions, and neologisms. In addition, Gerber and Gurland (1989) developed an Assessment Protocol of Pragmatic Linguistic Skills (APPLS), and P. M. Lesser and Coltheart (1992) published the Psycholinguistic Assessment of Language Performance in Aphasia (PALPA).

These studies demonstrate clearly that the somewhat elusive aspects of speaking style can be translated into scales that are readily understood and usable. Perhaps one reason for the infrequent use of such ratings has been that most have been developed in the context of relatively complex research rather than as part of the development of assessment tools. Another reason may be that psycholinguistic aspects of aphasic speech are rather complex in themselves and are not readily understood without prior linguistic training; hence, the clinically oriented examiner tends to shy away from psycholinguistic evaluations and to use relatively more concrete standard testing methods.

Construction Principles of Aphasia Tests

Both clinical examinations and tests may investigate the same areas of difficulty; the distinction lies in the quantification of the test examination and in the opportunity to compare quantitative scores with reference norms. Hence, a test could be defined as a clinical examination that meets a number of psychometric requirements.

The following section describes the psychometric requirements for a

well-constructed test to establish the information that should be critically evaluated before a test is put to use in daily practice. Few tests in the area of aphasia assessment fully meet the stringent psychometric requirements often demanded by the psychometric specialist and by groups concerned with the standards of testing (American Psychological Association, 1985). The reason for this is that most tests in the field of aphasia have been developed in individual laboratories, in the context of clinical work, and are not generally adopted by a large number of services and institutions. The collection of norms and the validity and reliability studies proceed slowly and are almost entirely dependent on the resources of test authors and their collaborators. The demand for such tests remains small compared, for example, with tests of general intelligence. In other words, test development is demanding in terms of both time and money. Aphasia tests are not best-sellers; as a result, development has been less than optimal.

General Requirements for Tests

The most frequently stated requirements for tests of any kind are demonstrated reliability, validity, and standardization (Anastasi, 1988; Cronbach, 1990).

STANDARDIZATION

STANDARDIZATION refers first to the test administration itself, which should be constant from patient to patient and from one examiner to another. If test administration and the conditions under which the test is administered are kept as controlled as possible, measurement error can be kept to a minimum. Any deviation from a standard administration procedure (e.g., prompting if the patient cannot respond readily, extending the time limits for answering) will inevitably produce more variability in test scores, and hence undesirable variance when the scores of patients or groups of patients are compared. The clinician may be tempted to use the test material to explore how much a patient may improve as a result of simple aids given during the testing. However justified, it should be understood that test results achieved under such modified conditions are no longer comparable to the published norms; that is, they contain an undesirable degree of measurement error. Exploration of the impact of aids and cues in a nonstandard, ad hoc basis has often been referred to as "testing the limits," and is frequently used for descriptive rather than diagnostic purposes.

Standardization also refers to the establishment of norms against which the performance of an individual patient can be compared. Norms are essentially a set of scores obtained from a reference group. For example, a

score at the 90th percentile indicates a performance better than that of 90% of the reference population. If the test is constructed for a variety of populations, separate norms have to be established (e.g., if scores tend to vary greatly with age, sex, or educational level). Often, additional norms can be avoided by using correction scores for these factors, but this may be impractical if more than two of these factors interact with each other. Norms are usually produced for a group of healthy men and women without neurological impairment or aphasia, which allows the examiner to see where a given person's performance lies in the distribution of scores. Clinically, the examiner can determine whether a patient's performance is within normal limits, is a borderline performance, or is defective (e.g., a performance lower than the 1st or 5th percentile).

RELIABILITY

RELIABILITY is the demonstration that on repeat administration after a reasonable time interval and under the same conditions similar results will be obtained for the same subject. Reliability is often demonstrated by giving an alternate form of the test during the same or at a subsequent session, by comparing alternate items of the test, or by subdividing the test. Generally, reliability is best demonstrated with normal, healthy subjects, as the measurement error in patient populations and the likelihood of change in performance due to changes in the patient's condition are both high. In practice, interscorer differences can be reduced to a minimum if the test manual contains sufficient scoring instructions and samples of how a given response can be scored. One form of expressing scoring reliability is to give the test records to two or more independent scorers and compute a correlation between scorers. Such interscorer reliability is highly desirable, as poor reliability of this type will obviously not only affect the general reliability of the test, but also introduce measurement error into studies of validity and other psychometric properties.

VALIDITY

Validity is probably the most crucial requirement of any test. It refers to the demonstration that a test measures what it claims. Validity can be measured in a variety of ways; usually, a distinction is made between PREDICTIVE (or criterion-related), CONTENT, and CONSTRUCT VALIDITY. Of the three forms, the demonstration that a test is a valid "predictor" of whether the patient is aphasic is the most popular, but of limited value in several ways. The demonstration of validity relies entirely on the fact that the aphasic patient's performance can be discriminated from that of normal subjects' on the basis of test results; in other words, the demonstration of validity comes close to the screening problem described earlier. Such a demonstration re-

lies on the clinical judgment made for the aphasic group but neglects the fact that the discrimination between aphasics and normals could result from entirely irrelevant (for aphasia) or trivial test items. In the ideal case, other contrast groups in addition to healthy, normal subjects (i.e., brain-damaged patients without aphasia) should be used.

Construct validity is often demonstrated by investigating the correlation of a new test with another test of known validity. Since few tests in the field of aphasia have known validity, another form of validity examination—the demonstration of factorial construct validity—is frequently used. In this case, factor analytic statistical techniques are used to show whether the tests in a given battery all contribute to a major factor of common variance that represents language functions.

Content validity refers to the adequacy of sampling from the domain of behaviors to be measured. In the case of testing for aphasia, for example, measuring verbal fluency alone would not be sufficient, because it does not appear to sample adequately the whole range of language behavior. In other words, test items should be based on sound reasoning, and should not be trivial or selectively biased. The content should also agree with the content areas as defined by other researchers. The range and diversity of the content of a test can also be explored by factor analysis.

Specific Requirements for Tests with Brain-Damaged and Aphasic Patients

In addition to the general requirements, several specific problems frequently occur in tests that are designed for use with brain-damaged patients and specifically with aphasics. These problems include the range of item difficulty, the need to clarify the nature of specific deficits revealed by the tests, the overlap of examinations for aphasia with measures of intelligence, the usefulness of a test in conjunction with recovery and therapy, and the overall conceptualization of the nature of aphasia.

RANGE OF ITEM DIFFICULTY

Range of item difficulty is usually determined by selecting from a range of "very easy" to "very difficult" items. In a well-constructed test, items should be homogeneously distributed; that is, the difficulty range (expressed in percentage of subjects passing each item) should rise in linear fashion from the first to the last item. The principle of homogeneity of item distributions is relatively easy to follow if we are dealing with a test for a normal population. However, if aphasics were given such a test, most items would be far too difficult for a majority of the patients. As a result, most aphasics would have scores in the bottom range of the distribution.

Consequently, aphasia tests must shift the difficulty of item distribution toward the lower or "easy" end to make it possible to discriminate mild, moderate, and severe levels of aphasia and to determine aphasic subtypes. This shift inevitably produces a "ceiling" effect if the test is then applied to normal subjects, as they would likely score at or near the 100% correct range. It is, of course, possible to include items that are easy enough to discriminate different degrees of aphasia as well as items difficult enough for a normal population. Such a test, however, could be lengthy and impractical.

OVERLAP WITH INTELLIGENCE TESTS

The overlap of assessment of aphasia with measures of intelligence, such as the Wechsler Adult Intelligence Scale (WAIS and its WAIS-R and WAIS-III revisions), has often gone unnoticed. It deserves special consideration in the context of item selection and in the context of other defects found in association with aphasia. It should be stressed that in the examination of aphasia the demands on the general intellectual abilities of the patient should be kept to a minimum. In addition, previously acquired knowledge of specific concepts and terms should influence the assessment of aphasia as little as possible.

The problem does not usually arise with the "easy" items used in aphasia tests. But when items for the "difficult" level are constructed, the separation of what is strictly language and what is intelligence becomes blurred. For example, naming tasks can be advanced to any level of difficulty by adding rare words and concepts that are likely to be found only in the vocabulary of the college-educated person of above-average intelligence. Tasks requiring definitions invariably tend to place higher value on abstract, elegant wording and penalize the uneducated, less intelligent subject. Tasks requiring oral arithmetic reasoning or the finding of superordinate concepts or similarities are, in fact, part of standard intelligence tests presently in use. For this reason, tests must be carefully scrutinized for content that exceeds the basic examination of language abilities. If such content cannot be avoided because of the range of item difficulty, the test must contain separate norms for patients of different ranges of intellectual and educational background or must apply adequate corrections for such factors.

USE IN MEASURING RECOVERY

The use of tests in the context of recovery and therapy poses two problems. The first is essentially an additional validity problem, that is, whether the test is suitable for the measurement of recovery. Tests adequate for the

measurement of recovery may be slightly different in content from tests that indicate the presence or type of aphasia, and may require more items in certain difficulty ranges to allow measurement of small steps in recovery. Related questions are the ability of the test to predict recovery or predict response to therapy, which must be established independently or in addition to other validation procedures. Interpretation may be complicated if changes over time merely reflect the patient's learning of the test ("practice effect") or remembrance of it, rather than actually indicating any underlying recovery.

CONCEPTUALIZATION OF THE NATURE OF APHASIA

The conceptualization of the nature of aphasia underlies many of the considerations outlined in this section. Test construction is directly influenced by whether we see aphasia as a specific disorder of selected abilities or as a pervasive disturbance of communication, and by whether we conceive of aphasia as unitary in nature or as consisting of many "subtypes." Benton (1967) pointed out that the choice of a model of language functioning determines what kind of test we construct or use. He indicated that the problem is similar to the one posed by the conceptualization of intelligence; it is similar also in the sense that no common agreement exists. Two approaches to test construction should be recognized as equally reasonable at this time:

1. To construct tests on the basis of one of the currently accepted conceptions of aphasia. This "taxonomic" or diagnostic approach ensures that the test measures all aspects viewed as important in a specific theoretical approach, but makes it probable that it will not be widely used as long as different conceptualizations of aphasia are held by other workers in the field.
2. To approach the problem pragmatically, avoid specific conceptualizations, and construct a test that contains a variety of probes of all abilities described by researchers of widely differing theoretical viewpoints. This pragmatic approach, quite commonly used in the field of intelligence testing, will not be fully satisfactory to any of the prevailing schools but may gain wider acceptance if the test instrument is otherwise well constructed and of demonstrated use in clinical practice.

Both approaches have been applied in the construction of currently used tests. Whether evaluation proceeds from an objective psychometric or from a theoretically based framework is a topic that has also been discussed by Kertesz (1994). In the following description of individual tests, we make specific reference to the conceptual framework used in each for the information of the reader unfamiliar with a given instrument.

Current Methods for the Assessment of Aphasia

The following selective review provides introductory information to readers unfamiliar with some of the assessment procedures to help them choose those methods most likely to meet their needs. Table 4.1 lists, in alphabetical order, the rating scales, and Table 4.2 the tests, that will be reviewed along with each test's primary reference and the acronym used in this chapter. Information is also given on the test procedure itself, its psychometric properties, the theoretical position of the test authors, and the most likely areas of use.

Clinical Examination

The advantage of the clinical examination lies in its flexibility, brevity, and suitability for even severely physically impaired patients. The examiner can conduct a cursory examination at the bedside during acute recovery and follow up on any errors made by the patient by administering additional tasks and quickly skipping across areas of strength where there is no obvious impairment.

Numerous versions of the clinical examination have been recorded, in formal descriptions within the contexts of the mental status examination in neurology (Strub & Black, 1985), a general neurological examination (Poeck, 1974), and specifically designed clinical examinations (Benson, 1979b), as well as in individual case descriptions (e.g., Geschwind & Kaplan, 1962). Luria (1966) provided a detailed description of a clinical examination. The clinical examination usually includes examining spontaneous and conversational speech, repetition, comprehension of spoken language, word-finding (naming common objects and object parts), reading (headlines or text from newspapers or magazines), and writing (including dictation).

TABLE 4.1
Rating Scales for Assessment of Aphasia

Scale	Abbreviation used in text	Source
Communicative Abilities of Daily Living	CADL	Holland (1980)
Communicative Effectiveness Index	CETI	Lomas et al. (1989)
Functional Abilities of Communication Profile	FACS	Frattali et al. (1995)
Functional Communication Profile	FCP	M. T. Sarno (1969)
Pediatric Evaluation of Disability Inventory	PEDI	Haley et al. (1992)

TABLE 4.2
Aphasia Assessment Instruments

Full title	Abbreviation used in text	Source
Aphasia Screening Test	AST	Reitan (1991)
Appraisal of Language Disturbances	ALD	Emerick (1971)
Arizona Battery for Communication Disorders of Dementia	ABCD	Bayles and Tomoeda (1990)
Auditory Comprehension Test for Sentences	ACTS	Shewan (1988)
Boston Assessment of Severe Aphasia	BASA	Helm-Estabrooks et al. (1989)
Boston Diagnostic Aphasia Examination	BDAE	Goodglass and Kaplan (1983)
Boston Naming Test	BNT	Kaplan and Goodglass (1983)
Controlled Oral Word Association	COWA	Spreen and Benton (1977)
Discourse Comprehension Test	DCT	Brookshire and Nicholas (1997)
Minnesota Test for Differential Diagnosis of Aphasia	MTDDA	Schuell (1965, 1973)
Multilingual Aphasia Examination	MAE	Benton et al. (1994)
Neurosensory Center Comprehensive Examination for Aphasia	NCCEA	Spreen and Benton (1977)
Pantomime Recognition Test	—	Benton et al. (1983)
Phoneme Discrimination Test	—	Benton et al. (1983)
Porch Index of Communicative Ability	PICA	Porch (1981)
Reporter's Test	—	De Renzi (1980)
Sklar Aphasia Scale	SAS	Sklar (1973)
Sound Recognition Test	SRT	Spreen and Benton (1974)
Token Test	TT	many versions, see text
Western Aphasia Battery	WAB	Kertesz (1982)

Because the clinical examination varies greatly in form as well as detail from one setting to another, we do not attempt a comprehensive evaluation of different examination methods. Clinical examination skills must be acquired under close supervision in a clinical setting.

In her reevaluation of the Short Examination for Aphasia, Schuell (1966) carefully debated the merits of the clinical examination in comparison to the comprehensive test. She stressed that only a comprehensive test can assess all aspects of "aphasia, [which] deals with one of the most complex and perhaps the only unique function of the human brain" (p. 138).

Screening Tests

Screening tests are designed to screen quickly for the presence or absence of aphasia. They are described here as screening tests because they

do not claim to provide a detailed description of the aphasic disorder but rather to check for and focus the direction on the problem if aphasia is present. Screening tests have been developed by Fitch-West and Sands (1987), Keenan and Brassell (1975), Lecours, Mehler, Parente, and Beltrami (1988), and Orzeck (1964; Inglis & Lawson, 1981), but have found only limited use. The Frenchay Aphasia Screening Test is a British measure that has led to some additional investigations (Al-Khawaja, Wade, & Collins, 1996; Enderby & Crow, 1996).

APHASIA SCREENING TEST

The Reitan (1991; Reitan & Wolfson, 1985; Wheeler & Reitan, 1962) version of the Halstead-Wepman screening test is designed to determine whether the patient can perform such simple tasks as spelling a word or naming an object. The Aphasia Screening Test (AST) procedures are such that the clinician should elicit the patient's best performance. A broad array of language functions is briefly assessed by one or two items each. For example, the patient is required to draw a shape, name it, and spell it; to read (e.g., "See the black dog"); to do a single pencil-and-paper and a single "in-head" arithmetic problem; and to demonstrate object use and picture drawing.

The test takes approximately 20 min to complete. The test manual provides many illustrative examples of performances. The test is usually given within the context of a complete neuropsychological test battery intended to assess the full range of psychological deficits after brain damage. Interrater reliability has been reported as high (Barth, 1984). The screening efficiency of the test as a single measure (discrimination between aphasic and nonaphasic brain-damaged patients) has been reported as 80% correct (Krug, 1971). Ernst (1988) reported normative data for elderly subjects.

It should be noted that Barth (1984), Goldstein and Shelley (1984), and Werner, Ernst, Townes, Peel, and Preston (1987) reported significant correlations between the AST and IQ as well as educational level in large neuropsychiatric populations. In a study by Snow (1987), only 1 of 33 items of the AST differentiated between right- and left-hemisphere lateralized tumor and stroke patients.

SKLAR APHASIA SCALE

The Sklar Aphasia Scale (SAS) (Sklar, 1973) provides a brief assessment of the aphasic patient's abilities along four dimensions: auditory decoding, visual decoding, oral encoding, and graphic encoding. Each of the four subtests is represented by five areas that each comprise five items. The SAS is constructed within a framework of a decode (input), transcode (process), and encode (output) model of language and its disabilities.

Each response on the SAS is scored on a 5-point scale, from a "correct" response (0) to no response (4). An impairment score for each test is obtained by finding the mean value of the four subtest impairment scores (0 = no impairment, 100 = full impairment). The total impairment index may be used prognostically in terms of potential benefit of therapy if modified by both the recency of the impairment and the patient's overall state of health. However, such prognostication is based only on a simple index score (e.g., an aphasic performing with a score of 70 has a better prognosis than an aphasic scoring 15), and psychometric evaluations of the prognostic significance of the SAS have not yet been presented.

The SAS test items were standardized on a sample of only 20 adults ranging in age from 29 to 78 years. The test author reported high correlations between SAS performance and performance on Eisenson's aphasia examination, Schuell's short version of the Minnesota Test for Differential Diagnosis of Aphasia (MTDDA), and the Halstead-Wepman AST in a sample of 12 patients. Although reliability data are not presented, the author describes five studies on the validity of the SAS as an instrument to assess language ability in aphasics.

Tests of Specific Aspects of Language Behavior

Several tests have been constructed for the detailed assessment of a specific language function. Such tests make no claim to cover all aspects of aphasia but do provide a relatively thorough assessment of the function in question. Because such functions are usually central to the aphasic disorder, however, these tests may also provide a reasonable discrimination between aphasic and nonaphasic patients in general. Some of these tests have been used as screening devices because of their good discrimination, although this was not necessarily the intent of the test authors.

AUDITORY COMPREHENSION TEST FOR SENTENCES

The Auditory Comprehension Test for Sentences (ACTS) (Shewan, 1988) provides an examination of auditory comprehension. The sentences are spoken by the examiner, and the patient must point to one of four visual stimuli to indicate which correctly represents the spoken sentence. Four training trials are permitted, which also serve as a screening device to determine whether the patient is too impaired to perform the entire task. These trials are followed by the 21 test items, which vary along parameters of sentence length, vocabulary difficulty, and syntactic complexity. Pass–fail scoring and qualitative error analysis are possible; the use of each system is made easy by the clear and concise ACTS response sheet. An average of 10 to 15 min is required to complete the ACTS.

A score of 18 of 21 (i.e., approximately 2 *SD* below the mean) is considered to be the lower bound of normal limits for adults with at least an elementary school education. Shewan (1988) reported an internal consistency correlation of .82, as well as a test–retest reliability of .87. Flanagan and Jackson (1997) recently reconfirmed the test–retest stability of the measure in a sample of neurologically intact individuals aged 50 to 76 years. Two measures of validity are provided in the manual. First, a correlation of .80 was obtained between the ACTS and an 8-point clinical rating of functional auditory–verbal comprehension. Second, when compared with established tests, there were correlations of .52 between the ACTS and the BDAE auditory comprehension section, and .89 with the WAB comprehension section. The lower ACTS–BDAE correlation was attributed to the wider range of abilities assessed by the BDAE compared with the ACTS.

Information about the ACTS standardization sample of 150 aphasics and 30 normal controls is provided in the manual. Means and standard deviations of the performance of aphasic subtypes (diagnosed clinically) are included: Wernicke's aphasics performed the poorest, followed in turn by Broca's aphasics, aphasics with anomia, and normal controls. The ACTS (along with the WAB) has been used to examine recovery and the differential impact of treatment in a subtyped sample of 100 aphasics (Shewan & Kertesz, 1984); improvements over time were similar for treated and untreated patients.

The ACTS is brief, easy to administer and score, and requires only simple nonverbal responses by the patient. The test has demonstrated reliability and validity, and appears to be a promising test of comprehension for sentence-length material with systematic variations of sentence length, difficulty, and complexity.

BOSTON NAMING TEST

The Boston Naming Test (BNT) (Kaplan & Goodglass, 1983; Kaplan, Goodglass, & Weintraub, 1978) has emerged as a popular test of visual confrontation naming not only for aphasia but also in dementia and other geriatric work. The current 60-item version has several variants: the original 85-item experimental form (Kaplan et al., 1978), the short 15-item version which is part of the CERAD screening battery for dementia (Morris, Mohs, Highes, Van Belle, & Fillenbaum, 1989), two 42-item equivalent versions (Huff, Collins, Corkin, & Rosen, 1986), and four 15-item short versions, drawn from the full-length, 60-item test (Mack, Freed, Williams, & Henderson, 1992). A Spanish adaptation is available (Taussig, Henderson, & Mack, 1988; Ponton et al., 1992). Morrison, Smith, and Sarazin (1996) used the test with normal French-speaking subjects in Quebec, Canada. The test may be suitable for children from age 4 years.

The BNT stimuli are line drawings of objects with increasing naming difficulty, ranging from simple, high-frequency vocabulary (*tree*) to rare words (*abacus*). Administration requires a spontaneous response within a 20-sec period; if such a response is not made, two kinds of prompting cues (one phonemic, one semantic) may be given. Rules allow for discontinuation and for starting the test at an advanced level, thus saving considerable time for subjects without obvious impairment. Scoring counts the number of spontaneously produced correct responses, the number of cues given, and the number of responses after phonemic cuing and after semantic cuing. M. Nicholas, Obler, Au, and Albert (1996) developed a rating of BNT errors based on relatedness to the correct stimuli.

Reliability has been assessed in a number of independent settings. Test–retest reliability after 8 months in 51 adult intractable epileptics was reported as .94 (Sawrie, Chelune, Naugle, & Zuders, 1996). Henderson, Mack, Freed, Kemperer, and Andersen (1990) reported an 80% response consistency for both uncued and cued responses in Alzheimer's disease (AD) patients after 6 months. Huff et al. (1986) divided the original BNT into two equivalent forms and obtained between-forms correlations of .81 in healthy control subjects and .97 in patients with AD. Thompson and Heaton (1989) compared the 85-item form with the standard 60-item form, and with the two nonoverlapping 42-item versions in 49 clinical patients. They found correlations of .96 to .84. The authors recommend the use of the short forms, as they may be more suitable if repeat testing is required. Another study constructed an "odd-item" and "even-item" version, as well as an experimental version of the BNT and found that all three short versions successfully discriminated AD, other dementing diseases, and normal older (mean age 73.7) subjects (Williams, Mack, & Henderson, 1989). Another 30-item version, developed for a Chinese population, showed a sensitivity between 56 and 80%, and a specificity between 54 and 70% in separating demented and nondemented subjects of low and high educational background (Salmon, Jin, Zhang, Grant, & Yu, 1995).

Age stability is a common finding in healthy elderly subjects (Ganguli, Seaburg, Ratcliff, Belle, & DeKosky, 1996; Mitrushina & Satz, 1995). However, Lansing, Randolph, Ivnick, and Cullum (1996) examined various short forms with a population of 717 controls and 237 AD subjects in the age range of 50 to 98 years and found significant correlations with age and education as well as gender effects for all forms, including the original full-length version. Correct classification rates varied from 58 to 69% for AD patients, and from 77 to 87% for normal controls. The authors used a discriminant function analysis to develop an empirical 15-item version balanced for gender. K. A. Hawkins et al. (1993) also found correlations between .74 and .87 between the Gates–McGinite Reading Vocabulary Test

and the BNT across normal and clinical adult populations; they demonstrated that norms for the test may lead to many false-positive rates for naming deficit, and that corrections should be applied, especially for subjects with a lower-than-average reading level. Concurrent validity with the Visual Naming Test of the Multilingual Aphasia Examination (MAE) (Benton, Hamsher, Rey, & Sivan, 1994) was described by Axelrod, Ricker, and Cherry (1994).

The manual provides means for aphasics with a BDAE severity level of 0 to 5, which are well below the level for normal adults. However, the range for aphasics with severity levels 2 to 5 extends well into the range for normals. This is not surprising, because naming is not necessarily impaired in all types of aphasia. Sandson and Albert (1987) found that aphasic patients made more perseverative errors than patients with right-hemisphere lesions; furthermore, perseverations were more frequent in patients with posterior rather than frontal lesions.

Knopman, Selnes, Niccum, and Rubens (1984) reported good measurement of recovery of naming after strokes of small volume in the left posterior superior temporal–inferior parietal and the insula–putamen areas. Welsh et al. (1995) found that semantic errors and circumlocutions in AD patients were associated with left mesial and lateral temporal lobe metabolism, as measured by positron-emission tomography (PET) and fluoro-D-glucose (FDG) emission techniques. The left anterior temporal area has also been implicated (Tranel, 1992). However, Trenerry et al. (1995) reported that carefully limited anterior right or left temporal lobectomy in 31 left and 24 right lobectomy patients with left-hemisphere language lateralization did not impact positively or negatively on BNT performance. The BNT was also not sensitive as to side of epileptic focus in a study of patients with idiopathic epilepsy (Haynes & Bennett, 1990) and in patients with anterior temporal lobectomy (Cherlow & Serafetinides, 1976). The test is sensitive to subcortical disease (multiple sclerosis and Parkinson's disease), even when global mental status is only mildly affected; in addition, responses were slower than in normal controls (Beatty & Monson, 1989; Lezak, Whitham, & Bourdette, 1990).

As with other tests, visual–perceptual integrity should be checked if errors occur. Kaplan and Goodglass (1983) noted that, particularly in patients with right frontal damage, "fragmentation responses" may be made (e.g., the mouthpiece on a harmonica is interpreted as a line of windows in a bus; Lezak, 1995).

CONTROLLED ORAL WORD ASSOCIATION

The Controlled Oral Word Association (COWA) provides a fast, efficient assessment of verbal fluency. The task requires the patient to produce in a

limited time period (usually 1 min) as many oral word associations as possible to a specific letter of the alphabet or to some category, such as animals or foods. The test does not measure verbal fluency of conversational speech. Deficits in aphasic patients are common, but defective COWA has also been studied in patients with dementia, in nonaphasic patients with anterior left-hemisphere lesions, and in patients with frontal lobe pathology manifesting disorders of executive functions. Several standardized tests are available. The first, developed by Spreen and Benton (1969, 1977), was introduced as a subtest of the NCCEA, but has been widely used as a single test. Other versions were created by Benton et al. (1994) as part of the MAE, by Goodglass and Kaplan (1983) as part of the BDAE, and by R. T. Wertz (1979).

Spreen and Benton's version requires the patient to say as many words as possible that begin with the letters F, A, and S within 1-min time periods for each letter. Proper names and words that differ only in suffix are not acceptable; performance is gauged in terms of the sum of admissible words in all three trials. Normative data, as well as corrections for age and level of education, are available (Spreen & Benton, 1977; Spreen & Strauss, 1998).

The letters used in the NCCEA version are of the "easy" level. Borkowski, Benton, and Spreen (1967) examined the number of associations by normal adult females for the letters of the alphabet. The number of associations was related to the difficulty, as defined both by the Thorndike–Lorge (1944) word count (.80) and the number of words per letter in *Webster's New Collegiate Dictionary* (.74). The authors also reported that a heterogenous sample of brain-damaged patients performed less well than normal adults at all levels of severity, lending validity to the testing method. Patients with low IQ scores were better differentiated with easy-level letters, whereas patients with high IQ scores were better distinguished with more difficult letters.

Interscorer reliability is very high, and 1-year test–retest reliability has been reported as .70 (Snow & Tierney, 1988). Concurrent validity has been established in several studies, generally indicating better validity for letters than categories (Coelho, 1984), although in dementia and the subcortical damage category (semantic) fluency tends to be more affected than letter association (phonetic fluency) (Butters, Granholm, Salmon, Grant, & Wolfe, 1987; Monsch et al., 1994; Monsch, Bondi, Butters, & Salmon, 1992). Correlation with age, education, WAIS-R Verbal IQ, and Performance IQ is quite low (Yeudall, Fromm, Reddon, & Stefanyk, 1986). FAS-COWA was part of the computerized Alzheimer's Disease Assessment Battery (Branconnier, 1986) and discriminated well between AD and normal control subjects. Consistent with clinical–anatomic expectations FAS–COWA corre-

lates with glucose metabolic rates in left prefrontal regions in poststroke aphasic patients (Karbe, Kessler, Herholz, Fink, & Heiss, 1995). Benton et al.'s MAE version (1978; Benton, 1994) differs from the NCCEA in using three letters of progressively increasing associative difficulty, otherwise the testing is quite similar. Two equivalent versions (CFL and PRW) are available for repeated or follow-up administrations.

The BDAE uses category (animals) COWA, a format traditionally used in child evaluation settings. Patients are instructed to say as many different animal names as possible. A 90-sec recording period is allowed, with responses recorded in 15-sec blocks. The best 60-sec period (usually the first 60 sec) is scored. COWA tests are quick and simple. The versions described here have proven discriminative validity. COWA tests may not be very sensitive in distinguishing at lower levels of ability, but they are capable of screening for the presence of less severe disability.

DISCOURSE COMPREHENSION TEST

The Discourse Comprehension Test (DCT) (Brookshire & Nicholas, 1997) arose out of psycholinguistic research (L. E. Nicholas & Brookshire, 1995), which suggested that word and sentence comprehension tests are unlikely to predict comprehension of multiple-sentence messages and are unable to examine contextual aspects of such messages, that is, the ideas that are implicit across sentences. The authors also cite work by Brownell (1988) that suggests that word and sentence comprehension measures may underestimate comprehension problems in patients with right-hemisphere lesions.

The DCT has a narrative format. Paragraph-length stories are played for the patient on audiotape. The patient is queried after each story. Eight yes/no questions are divided as follows: two deal with the story's main ideas, two with implied main ideas, two with stated details, and two with implied details. The authors found that regardless of group, normals and aphasics score higher on main ideas than on details, and higher on stated than implied material. Ten stories are divided into two equivalent 5-story sets. After one or two practice stories, one set is administered; if performance is variable, the second set is given.

Scores on the DCT include an overall score and scores for each type of question. The test uses cutoff scores to denote defective performance, set at the 5th percentile of the normal sample. Standardization was based on 40 normal adults, aged 55 to 75 years, with a range of education from 8 through 18 years. Performances by 20 aphasics, 20 patients with right-hemisphere lesions, and 20 patients with traumatic head injury were reported. Overall scores correlated minimally with age and mildly with education. Test–retest reliability after 2 to 3 weeks was .87 for aphasics and

.95 for patients with right-hemisphere lesions. Studies of content validity are reported in the manual. Criterion-related validity ranged from .54 to .76 for the DCT with the BDAE Auditory Comprehension subtests, and with the four-subtest short version of the PICA (DiSimoni, Keith, & Darley, 1980).

The manual includes instructions for a silent-reading comprehension administration of the DCT with the same stimuli. The authors' Sentence Comprehension Test is also included, which is used to determine whether consistent yes/no responses can be elicited.

The DCT fills a niche in the assessment of comprehension at the level of narrative discourse which is only approximated in other assessment batteries. The authors report that results may generalize to the level of highly structured communicative dialogue, but it remains unclear whether it can be generalized to less structured dialogue. In addition to helping describe the communicative skills of brain-damaged patients, the DCT offers a potentially useful research instrument at a level between concise measures of single word and sentence comprehension (e.g., the ACTS) on the one hand, and subjective functional communication rating scales on the other. A developmental study (Newton, 1994) reported that with young children the use of a picture that depicts the initial circumstances of a narrative story helps them maintain a functional mental model to supplement comprehension as the story progresses. The use of such pictures for the DCT might provide a useful treatment-oriented adjunct, as well as an opportunity to compare patient performance with and without such visual aids.

The question remains whether the length of the stimuli and the number of questions per story introduce an undesirable attentional–memory component into the test which may influence performance. For example, Tompkins, Bloise, Timko, and Baumgaertner (1994) commented on the relationship between memory capacity and discourse passages that require a revision of the plot as the story unfolds.

The weakness of the DCT is the relatively small size of the standardization sample. It remains open whether the cutoff scores would remain stable with a larger sample of normal controls. Larger samples of both aphasic and control subjects would also permit a more refined percentile scoring system.

"IOWA-GROUP" TESTS

In addition to developing two comprehensive aphasia batteries, Benton and his colleagues have developed a number of specific-function tests for the evaluation of aphasic patients. Some of these tests are described briefly.

The SOUND RECOGNITION TEST (SRT; Spreen & Benton, 1974; Spreen &

Strauss, 1998), presented on audiotape, examines auditory object recognition (e.g., train whistle) by requiring the identification of familiar sounds. The original form of the SRT presented two equivalent forms of 13 items each. Three modes of administration were offered: verbal response, pointing to one of four multiple-choice names, and pointing to one of four multiple-choice line drawings. Varney (1980, 1984b) modified the format such that all 26 items are presented and multiple-choice line drawings are used as the response format. Scoring standards are detailed in Spreen and Strauss (1998). Spreen and Benton (1974) described norms for normal adults and children, and the performance of brain-damaged adults. Impairments of sound recognition are typically limited to aphasic patients and are frequently associated with aural comprehension deficits. Aphasics with aural comprehension deficits during the acute stage of recovery from cerebrovascular accident but with normal sound recognition show rapid and near complete recovery of aural comprehension, whereas patients with acute aural comprehension and sound recognition deficits show a much poorer outcome (Varney, 1984b). Defects in sound recognition are associated with a number of lesion sites, none of which appear specific for the manifestations of the defect (Varney & Damasio, 1986).

The PANTOMIME RECOGNITION TEST (Benton, Hamsher, Varney, & Spreen, 1983), presented on videotape, shows a male pantomiming different activities (e.g., using a telephone). The patient is presented with four multiple-choice responses: one correct, one semantic error, one neutral error, and one odd error. Defective pantomime recognition is an infrequent finding, observed most frequently in aphasic patients, and appears to be related to associated defects in reading comprehension level. Demented patients may perform defectively on the task. Taking another approach, Records (1994) designed an experimental test evaluating the impact of gestures on comprehension in aphasic patients.

The PHONEME DISCRIMINATION TEST (Benton et al., 1983) is a brief test of the discrimination of phonemic sounds. The test consists of 30 pairs of one- or two-syllable nonsense words on audiotape. The patient has to indicate whether the pair members are the same or different. Practice or pretraining is encouraged to determine whether the patient can make same–different responses reliably. Normative data and samples of 100 aphasic left-hemisphere damaged and 16 nonaphasic right-hemisphere damaged patients are reported in the test manual. Comparisons of the performance of aphasic patients on the Phoneme Discrimination Test with the MAE Aural Comprehension Test are also reported (see also Varney & Benton, 1979). Varney (1984a) presented longitudinal data on the Phoneme Discrimination Test in the context of evaluating additional measures of comprehension.

PEABODY PICTURE VOCABULARY TEST-III

The Peabody Picture Vocabulary Test-III (PPVT-III) (Forms A and B, 3rd ed., Dunn & Dunn, 1997) assesses auditory comprehension of picture names, and is also part of the Florida Kindergarten Screening Battery (Satz & Fletcher, 1982). It was initially constructed as a test of hearing vocabulary in children, but has since been standardized for adults and used with a variety of clinical populations. The test requires the subject to choose one of four items displayed on a card as depicting the word spoken by the examiner. After 5 training items, 204 items of increasing difficulty can be given, but usually only 35 to 45 items need to be administered if a suitable entry point (six consecutive correct) is chosen; the test is discontinued after consecutive failures on 6 out of 8 items. It has been restandardized for the age range of $2\frac{1}{2}$ to 90+ years of age. The time required is about 10 to 20 min. In the new edition, a number of items have been revised or added to correspond well to the negatively accelerating growth curve of vocabulary with age. A Spanish version is available.

The new edition has been standardized on a sample of 2725 subjects representative of the 1994 U.S. census estimate, ranging in age from $2\frac{1}{2}$ to 90 years. Canadian norms are available from the distributor (Psycan). Kamphaus and Lozano (1984) note that in 6- to 11-year-old children with Spanish surnames (about one half of whom spoke Spanish at home), standard scores were about 12 to 13 points below the national norms, although the scores showed regular, expected increases with age. Sattler (1988) recommends special care in the interpretation of scores of ethnic minority children because their scores tend to be lower and reflect their verbal and experiential differences rather than ability.

The score on this test is simply the number of items passed including the items before the entry point. The manual allows translation of these scores into "age equivalents," "standard score equivalents," stanines, and percentiles. The authors have added a "true confidence band," indicating the range of scores in which the subject's true score can fall 68 times out of 100.

Split-half reliability has been reported as ranging from .61 to .88 in children and adolescents, and .82 in adults. The reliability of alternate forms ranged from .73 to .91 (Stoner, 1981; Tillinghast, Morrow, & Uhlig, 1983). Retest reliability with the alternate form after a minimum of 9 days showed a median correlation of .78. In children, retest stability over 11 months has been reported as .84 for the revised PPVT (Bracken & Murray, 1984), .81 in retarded children over a period of 7 months (Naglieri & Pfeiffer, 1983), and .71 in a mixed clinical neuropsychiatric population after $2\frac{1}{2}$ years (Brown, Rourke, & Cicchetti, 1989). Internal consistency ranged from .96 to .98 in

children 6 to 11 years old (Kamphaus & Lozano, 1984). Construct validity of the test as a measure of scholastic aptitude is good (Hinton & Knights, 1971). Bracken and Murray (1984) report a predictive validity of .30 with spelling, .54 with reading recognition, .58 with reading comprehension, and .59 with the total Peabody Individual Achievement Test (PIAT) for the revised PPVT; similar values were reported for the first edition (Naglieri, 1981) and for mentally handicapped children with the revised edition (Naglieri & Pfeiffer, 1983). Concurrent validity with similar tests—the Bracken Basic Concept Scale, the preschool version of the Boehm Test of Basic Concepts and its revised version—were .68, .65, and .62, respectively (Zucker & Riordan, 1988). Because vocabulary is the single most important subtest of most intelligence tests, the test also correlates with the WISC-R, although the results of several studies are somewhat contradictory. In children, correlations with measures of verbal (.87), performance (.80), and full-scale IQ (.88) have been reported (Crofoot & Bennett, 1980). The PPVT also correlates highly with the McCarthy Scales in children (Naglieri, 1981), with the 1986 Stanford-Binet Intelligence Scale (Carvajal, Gerber, & Smith, 1987), and with the achievement scale of the Kaufman Assessment Battery for Children in a learning-disabled population (.78; D'Amato, Gray, & Dean, 1987). However, Faust and Hollingsworth (1991) found a correlation of only .34 with the WPPSI-R Full-Scale IQ, and of .30 and .31 with the PIQ and VIQ, respectively, in normal preschoolers. Altepeter (1989; Altepeter & Johnson, 1989) found only modest correlations in healthy adults with the WAIS-R (.47 with full-scale IQ) and warns that in this age range the test tends to overestimate IQ in the lower ability ranges and to underestimate IQ in the higher ability ranges; in a cross-tabulation of IQs in 10-point steps, less than one half of the clients were classified correctly. Price, Herbert, Walsh, and Law (1990) reported similar discrepancies in adult psychiatric inpatients.

Hollinger and Sarvis (1984) also stress the role of perceptual–organizational ability in PPVT performance of school-age children, and L. J. Taylor (1975) reached the same conclusion for preschool children based on a factor analysis of the WPPSI and the ITPA as well as the PPVT. Children with impaired oral language production (Rizzo & Stephens, 1981) and nonpsychotic, emotionally disturbed adolescents (Dean, 1980) tend to produce variable results. Elliott et al. (1990) found also that the PPVT results in children between 6 and 11 years of age with normal pure-tone hearing were strongly influenced by the ability to make fine-grained auditory discriminations, with consonant–vowel stimuli varying in timing and place of articulation. The PPVT showed significant differences between 28 young adults with specific language impairment and 28 controls (means of 82.36 and 92.79, respectively; Records, Tomblin, & Buckwalter, 1995). Das,

Mishra, Davison, and Naglieri (1995) demonstrated that the PPVT is sensitive to mental decline (dementia) in older (age greater than 50 years) Down's syndrome patients, with results parallel to those in a dementia rating scale.

The test is relatively nonthreatening and requires little verbal interaction; because it also allows gestural or pointing responses, the test is suitable for language-impaired as well as autistic or withdrawn patients. The auditory and visual–perceptual integrity of the patient should be carefully considered in interpreting the results of this test. Considering the extensive research with the PPVT and its continuing revision, it is clearly the preferred measure of vocabulary for children and is now valid for adult and geriatric populations.

REPORTER'S TEST

De Renzi used the stimuli and most of the commands from the Token Test to construct the Reporter's Test, a test for expressive deficits in aphasics (De Renzi, 1980; De Renzi & Ferrari, 1979). The Reporter's Test was designed to meet two specific goals: (a) to elicit organized speech and (b) to limit the range of what the patient is expected to say. Although picture description tasks (e.g., the BDAE "Cookie Theft" card) adequately fulfill the first goal, they fail on the second. Other task-description tests have been used in nonaphasic clinical settings (e.g., the "dice game"; McDonald & Pearce, 1995), but lack the range limitations of the Reporter's Test. The patient is required to act as a "reporter"; that is, she or he must report the actions of the tester to an imaginary third person. For example, if the examiner were to touch the large red circle, the patient must verbalize the relevant information necessary for a third person to reproduce the tester's actions ("Touch the large red circle"). The Reporter's Test begins with several sample items to acquaint the patient with the task. The test comprises five sections; the first four are taken from the Token Test. De Renzi recommended that it be used after the Token Test, so that the patient is acquainted with the stimuli and the required commands.

De Renzi (1980) described initial findings for the Reporter's Test in discriminating 24 aphasic patients from 40 hospitalized, nonaphasic, nonbrain-damaged controls. In this study, an actual third person sat next to the patient and performed the instructions given by him or her. Years of education, but not age, were significantly related to performance; scores were corrected to account for education. Using a cutting score expected to produce 5% false positives, a 97% hit rate was obtained. Classification accuracy was higher for the Reporter's Test than for four other tests of verbal expression: visual naming, oral fluency, sentence repetition, and story telling.

De Renzi and Ferrari (1979) described aphasic performance using the

original pass–fail scoring, partial credit for correct performance after repetition, and a weighted scoring (1 point for each bit of information on a trial but without credit for repetition). Aphasic patients, nonaphasic left brain-damaged patients, and nonaphasic right brain-damaged patients were discriminated with an 82% hit rate. The authors recommended the use of both scoring systems to offset the weaknesses of each: lower classification for the weighted system and overly severe evaluation with the pass–fail scoring. Unfortunately, the test has not been used as widely in clinical settings and psychometric research as the Token Test.

TOKEN TEST

The Token Test (TT) was introduced as a brief test by De Renzi and Vignolo in 1962 to examine subtle auditory comprehension deficits in aphasic patients, by having patients respond gesturally to the tester's verbal command. Since its inception, the original TT has been used, modified (De Renzi, 1980; De Renzi & Faglioni, 1978), and included in some batteries (Benton et al., 1994; Spreen & Benton, 1977). The original test has spawned many variants: short forms (Boller & Vignolo, 1966; Spellacy & Spreen, 1969; Van Harskamp & Van Dongen, 1977), a concrete-objects version (Martino, Pizzamiglio, & Razzano, 1976), a format with both auditory and visual presentation of commands (Kiernan, 1986), a TT "battery" (Brookshire, 1978), and a version with expanded linguistic examination (McNeil & Prescott, 1978). Equivalent versions in several languages are available (e.g., Italian, German, Portugese; Fontanari, 1989; Kannada; Vena, 1982).

The TT is a portable test that, in most versions, contains 20 plastic token stimuli of two sizes (large and small), two shapes (square/rectangular and round), and five colors. The tokens are laid out in front of the patient, typically in a standard 4×5 matrix. The test has a variable number of sections that increase in sentence length and linguistic complexity (e.g., from "Point to a square" to "Put the small red square on the large blue circle"). The McNeil and Prescott (1978) version provides the most complex commands.

Some authors have reported age (Emery, 1986) and level of education (De Renzi, 1980; De Renzi & Faglioni, 1978) differences on certain versions of the TT. Gallaher (1979) reported day-to-day retest reliabilities for one version of the TT and its subsections to be greater than .90. Validation studies have shown the TT to be a strong and accurate discriminator between the performance of aphasic patients and that of normal hospitalized adults (De Renzi, 1980), nonaphasic right-hemisphere-damaged adults (Boller & Vignolo, 1966; Swisher & Sarno, 1969), and nonaphasic diffuse and focal brain-damaged adults (Orgass & Poeck, 1966). Morley, Lundgren, and Haxby (1979) found the TT to discriminate particularly well between normals and aphasics with high levels of ability compared with discrimina-

tions on the BDAE comprehension section and the Porch Index of Communicative Ability (PICA). Poeck, Kerschensteiner, and Hartje (1972) demonstrated independence of TT performance and the fluency–nonfluency dimension in aphasic patients.

Cohen, Kelter, and Shaefer (1977), and Cohen, Lutzweiler, and Woll (1980) studied construct validity and other aspects of TT validity. The memory component of TT performance was examined by R. Lesser (1976), Cohen, Gutbrod, Meier, and Romer (1987), and Gutbrod, Mager, Meier, and Cohen (1985), who concluded that the test measures deficits in the short-term storage of highly specific information in aphasics. In contrast, Riedel and Studdert-Kennedy (1985) claimed that a general cognitive deficit is responsible for poor TT performance. In AD patients, Swihart and Panisset (1989) found that a short version of the test correlated only weakly with other simple auditory–verbal comprehension tasks, but correlated highly with the Mini-Mental-State Examination because of strong perseverative tendencies found in that patient population.

The TT has maintained consistent popularity as both a clinical and an investigative instrument, and has been examined for use as a therapeutic tool (Holland & Sonderman, 1974; West, 1973). Two major compilations of work with the TT are available (Boller & Dennis, 1979; McNeil & Prescott, 1978), and at least three English-language versions are commercially available (Benton et al., 1994; McNeil & Prescott, 1978; Spreen & Strauss, 1998).

The TT's advantages lie in sound discriminative validity, portability, and brief administration time. Brookshire's (1973) early advice remains valid: The clinician should keep in mind that although it is a sensitive indicator of comprehension deficits, the TT relies on a limited stimulus array. Other tests of auditory comprehension (e.g., the ACTS) may be used to supplement the TT. Rao (1990) also points out that the test introduces a somewhat artificial test situation and therefore has less "eco-validity" than other, functional communication measures. Other comprehension tests include Lexical Understanding with Visual and Semantic Distractors (LUVS; Bishop & Byng, 1984), which focuses on semantic comprehension, and an object-manipulation test designed to measure syntactic comprehension (Caplan, 1987).

Rating Scales

Rating scales assume a position somewhere between clinical assessment and psychometric tests. The clinician who assigns a label of mild, moderate, or severe to the symptoms of a patient is actually providing a basic rating of severity. Ratings are frequently used (a) as a summary judgment of severity of any symptom or syndrome, (b) as a specific judgment of aspects

of a patient's behavior that cannot be readily measured, (c) to rate the "quality of life" (QOL), or (d) to represent an entire class of functioning. A specific example of (a) is a professional's summary judgment in disability or worker's compensation cases. The second application has its origin in ratings of "activities of daily living" (ADL), frequently used by occupational therapists to rate numerous aspects of the patient's behavior and to measure the progress of the patient's general behavioral impairment during therapy. Some aspect of the patient's speech behavior—for example, ability to communicate in the home setting—is rated on a scale of levels or points. QOL ratings, application (c), have also become an important aspect of the success of rehabilitation (M. T. Sarno, 1997). In this context, QOL addresses the question of "how a person experiences aphasia, how its meaning influences his/her behavior and interaction . . . A total of human response to an unexpected and unwanted life event" (M. T. Sarno, 1997, p. 675), and includes psychosocial factors such as loneliness, difficulty making friends, lowered self-esteem, and depression. Specific instruments have been designed to measure QOL (Caregiver Burden Interview, Zarit, Reever, & Bach-Peterson, 1980; Functional Life Scale, J. E. Sarno, Sarno, & Levita, 1973; Geriatric Evaluation of Relative's Rating Instrument, Schwartz, 1983).

The last application is best demonstrated by the increasing use of functional independent measures (FIMs) (Frattali, 1993; Hamilton, Granger, Sherwin, Zielezny, & Tashman, 1987) in rehabilitation units, which include items ranging from sphincter control and feeding to communication. Such functional communication ratings have gained further importance in recent years because they are often used for public policy and reimbursement issues (Frattali, 1992, 1993). Progress in aphasia rehabilitation is now frequently defined as progress in functional communication, although Crockford and Lesser (1994) state that the actual use of such scales by practicing clinicians seems to be limited. Such measures sometimes attempt to reduce entire cognitive domains, such as communication, to a single scale of independence and various levels of dependence. It should be noted that single FIMs representing entire cognitive domains such as language or memory would seem to be of questionable value and validity.

Several comprehensive batteries, such as the BDAE, MAE, and Western Aphasia Battery (WAB), include qualitative rating scales as part of their set of subtests. Usually, a rating scale should not exceed seven points (from "normal" to "very severe"), since it has been demonstrated that the use of more than seven points does not enhance the accuracy of the ratings but merely provides a false impression of greater accuracy.

Rating scales should be subjected to careful interjudge reliability studies. Reliability can be improved if careful descriptions of each rating point

are provided. For example, instead of marking the lowest point as "normal," and the highest as "very severe," each point should be illustrated in as much detail as possible with examples. Rating scales are no substitute for psychometric testing, but they are extremely valuable if the information being rated cannot be readily tested or is too complex to be documented in test scores. Ratings of communicative ability in the home or in a conversational setting are often made by an informant (e.g., a relative or a member of the nursing staff) rather than by the clinician who sees the patient only in a highly structured, isolated, and somewhat artificial examination or therapy situation. The Functional Communication Profile (M. T. Sarno, 1969) is an example of a standardized rating scale of such communicative features.

The trend toward using rating scales for functional communication, particularly for program evaluation, has been criticized by Sacchett and Marshall (1992). They argue that material that has been "squeezed" out of a patient by role-playing or verbal prompts does not reflect natural conversational abilities, and that ratings of functional communication or QOL, for the sake of justifying speech therapy costs, may lead to forms of intervention that merely promote abilities in the areas of speech subject to rating. Instead, they advocate a case study approach along psycholinguistic lines, arguing that this will "repair" or improve language processing, generalizing to overall language use.

Communication Profiles

A distinction between selective language elicited in the typically structured test setting and the patient's ability to communicate in everyday environments has intuitive value to the speech clinician. The Functional Communication Profile (FCP) was the first standardized attempt to assess the functional usefulness of language ability in the everyday life of the aphasic patient. The Communicative Abilities of Daily Living (CADL; Holland, 1980) was the second psychometric measure to index the degree of disability faced by the patient in attempting to communicate in daily life, but using observations of actual interactions. The Communicative Effectiveness Index (CETI; Lomas et al., 1989) relies mainly on the patient's communicative interaction with spouses or significant others in 16 different situations, and correlates with the improvements shown on the WAB. The Functional Assessment of Communication Skills (FACS; Frattali, Thompson, Holland, Wohl, & Ferketic, 1995) returns to the FCP method by using the observations of both the informant and the speech clinician. Other, infrequently used instruments (e.g., Communicative Competence Evaluation Instrument; Houghton, Pettit, & Towey, 1982) are summarized and re-

viewed by Manochiopinig, Sheard, and Reed (1992). A new British instrument, the Assessment of Communicative Effectiveness in Severe Aphasia (Cunningham, Farrow, Davies, & Lincoln, 1995) uses standard open-ended conversational questions, objects, and pictures. It is fully described in the first publication, but has so far only been examined for interrater and test–retest reliability with 10 aphasic and 10 normal speakers. The rating of functional language and verbal processing skills in daily living has also become a common feature in the many geriatric rating scales (see the review by DeBettignies & Mahurin, 1989), and is available as separate language ratings (Patient Functional Communication Screening Instrument, FCS; Toner, Gurland, & Gasquoine, 1984; Toner, Gurland, & Leung, 1990) or in mental competence batteries (e.g., the Cognitive Competency Battery; Wang, Ennis, & Copland, 1986). However, these omnibus measures for use with the elderly often lack suitable standardization and item range for use with aphasic patients. Le Dorze, Julien, Brassard, Durocher, and Boivin (1994) reported on the development of a scale for use with long-term care patients, including those with aphasia and/or dementia, based on 196 statements about communicative acts in daily living.

The CADL, FACS, and FCP defer obtaining pure, isolated samples of specific language behaviors (as obtained by diagnostic tests) in favor of sampling complex communicative behaviors, such as the ability to communicate on the telephone, handle money, read newspapers and product labels, and ask for, correct, and impart significant information to and from others. As such, the information gauged on these profiles is a unique contribution to the overall assessment of the aphasic patient, providing the clinician with descriptive information about the communicative status of the individual, which can be treated as a second dimension of information not directly obtained from diagnostic testing procedures (M. T. Sarno, 1984a).

COMMUNICATIVE ABILITIES OF DAILY LIVING

The Communicative Abilities of Daily Living (CADL) (Holland, 1980) was designed to measure the functional communicative ability of aphasic patients. Much of the test involves patient performance during simulated, cue–context daily activities, such as dealing with a receptionist, communicating with a doctor, driving, shopping, and making telephone calls. Given its inherent focus on communicative rather than language ability per se, accurate communication whether oral, written, gestural, or by any other modality is acknowledged as significant.

The CADL has 68 items, which are scored as either "correct" (2 points), "adequate" (1 point), or "wrong" (0 points). For example, at one point the examiner asks the patient, "Your first name is _____, right?" (filling in a

fictitious name). If the person's response includes both a negative response ("No," headshake, written response, etc.) and his or her correct name, 2 points are given. If the patient simply replies with a negative response without further elaboration, the response is considered adequate but not fully appropriate, and is scored 1 point. If the patient responds affirmatively, perseverates, echoes the question, responds incoherently, or simply does not respond, no points are allotted. Requests for repetition are considered legitimate communicative statements and are not penalized. However, if the patient fails to respond within 5 sec, only partial credit (1 point) is allowed for a correct response. Given the generally slowed psychomotor and information-processing speed that frequently accompanies acquired brain damage, one wonders whether this time restriction is overly strict and does not indeed violate one of the goals of the CADL, which is to score communication success regardless of transmission method. Testing time ranges from 30 to 90 min.

The test situation is deliberately informal, and the examiner is instructed to act in various roles as much as possible (e.g., by changing his or her voice, introducing humor, etc.) to increase the "contextual richness" of the situations created during the test. The total score is the sum of all points scored on the 68 items (maximum score is 136). Holland reported reliability for 20 subjects retested after 1 to 3 weeks by a different examiner as .99, and internal consistency as .97. Concurrent validity was .84 with the BDEA, .93 with the PICA, and .87 with the FCP. Correlations for 23 ratings by staff and family were .67. Criterion validity was established by comparing CADL performance with behavior during a 4-hour observation period. Furthermore, the distribution of scores for different types of aphasia followed the clinical impression of their severity. Global aphasics showed the poorest CADL performance. Wernicke's aphasics performed more adequately than did the global aphasics, but less well than Broca's aphasics. The anomic group had near-normal scores. Aphasics living at home consistently had higher scores than those living in an institution.

Normative data are based on a sample of 130 normal adults (fluent English speakers without history of mental disorder or brain damage, or vision or hearing impairment). The differences between occupational levels, institutionalized versus noninstitutionalized, and between males and females were not significant. However, a slight decline with aging (over 65 years) was significant. The performance of 130 aphasic patients is also described in the manual.

CADL performances have been examined in patients with Wernicke's aphasia, with normal controls, with AD, and in depressive patients (Fromm & Holland, 1989). Aphasics had markedly different profiles compared with AD patients, who showed performances corresponding to AD

severity. Depressed patients also showed lower scores than controls, but tended to show incomplete responses rather than the irrelevant, vague, or rambling responses seen in AD. A group of adult mentally retarded subjects with IQs between 50 and 80 obtained scores in the aphasic range; IQ and CADL score correlated .72 (Holland, 1980). A group of hearing impaired subjects (with hearing aids) showed near-normal scores (Holland, 1980).

The CADL is an excellent supplement to other aphasia examinations because it allows an estimate of the patient's communication ability rather than the accuracy of language. The "staged" quality of some sets of items requires a certain amount of acting ability on the part of the examiner, and may not always be successful with patients who refuse or cannot enter into such simulated interactions. It is not clear from the manual how this affects scores, but it is probably wise to take note of a patient's inability to follow the play-acting, and to make allowance for this in the interpretation of the total score. These interactions, created by specific instructions in the CADL, would also seem to require the patient to show a certain amount of new learning during the test situation which is not required for the FACS or FCP ratings. Apraxic patients may also be problematic (R. Wertz, LaPointe, & Rosenbek, 1984), although the test may serve as a supplementary instrument in such a population. Davis (1993) warns that the CADL is "still a test and does not provide for observing natural interactions" (p. 65).

Italian (Pizzamiglio et al., 1984), Spanish (P. Martin, Manning, Munoz, & Montero, 1990), and Japanese (Watamori et al., 1987) versions are available. A new edition, the CADL-2, will soon be published (Holland, Frattali, & Fromm, 1998). This edition eliminates redundant items and avoids role-playing problems by substitution of hypothetical situations (A. Holland, personal communication).

COMMUNICATIVE EFFECTIVENESS INDEX

The Communicative Effectiveness Index (CETI) (Lomas et al., 1989) uses a 16-item questionnaire. Each question is concerned with an everyday situation and paired with a visual analogue scale (a line marked "Not at all" at one end, and "As able as before the stroke" on the other), which the relative or other caregiver has to mark. For example, one question asks: "Does he *intentionally* let you know how he is feeling rather than you 'reading' his emotion?" A template is used to convert each mark into a 10-point scale. This measure is designed to use little of the therapist's time. The authors applied the scale to 11 recovering (6 to 10 weeks postonset) and 11 stable (more than 15 months postonset) aphasics. The recovering group showed, with the exception of two patients, the same or more recovery as the WAB; and with the exception of three patients, the CETI showed the

same or less change compared with the WAB for the stable group. Crockford and Lesser (1994) found, however, that the scale has limited potential for planning appropriate intervention. In a follow-up of eight aphasics, retested after 3 months, the CETI was less effective as a measure of stability or change of communicative effectiveness.

The interrater reliability has been reported as .73 for the combined group, and as .94 for the stable group only. Test–retest reliability was .94, and the difference between first and second testing for recovering aphasics was significant. Internal consistency was .90. Concurrent validity was .61 with the WAB Aphasia Quotient on first testing, and .52 on second testing. Correlation with a global rating of aphasia severity by caregivers was .79 on first testing and .62 on second testing. However, correlation with a speech questionnaire was only between .46 and .43 for speaking, and between .47 and .56 for understanding. The authors (Lomas et al., 1989) ascribe this finding to the emphasis on language in the speech questionnaire, whereas the CETI stresses functional communication.

FUNCTIONAL ASSESSMENT OF COMMUNICATION SKILLS FOR ADULTS

The Functional Assessment of Communication Skills for Adults (FACS) (Frattali et al., 1995) uses 43 items rated on a 7-point scale. The ratings are based on the observation of the therapist, but may include observations reported by others. Ratings for communication independence cover Social Communication (e.g., refers to familiar people by name, understands facial expressions); Communication of Basic Needs (recognizes familiar faces, makes needs to eat or to rest known); Reading, Writing, and Number Concepts (makes basic money transactions, fills out short forms); and Daily Planning (tells time, follows a map, keeps scheduled appointments). In addition, each domain is rated on a 5-point scale for qualitative dimensions of communication: Adequacy (understanding of message), Appropriateness (communication is relevant and done under the right circumstances), Promptness (responds without delay and in an efficient manner), and Communication Sharing (burden on the communication partner). A combined communication independence score can be calculated. The ratings for each domain of communication independence and for the qualitative dimensions may be entered into profiles which facilitate plotting of the patient's progress during therapy. Each rating point is well-defined, as reflected in interrater reliability between .88 and .92 for communication independence scores (total .95) and quality of communication (.72 to .84, total .88) measured during a field test involving 31 examiners and 185 adult patients (Frattali et al., 1995). Internal consistency averaged .82 within domains, and .78 between items and overall score.

The external validity of the overall score for the aphasic group was .73, measured against the WAB Aphasia Quotient, and .78 for the qualitative dimension, against the Functional Independence Measure (State University of New York, 1993). Construct validity, measured with a principal component factor analysis, resulted in one major factor, with minor factors representing the four domain scores. Overall ratings of functional communication by clinicians and family members correlated with FACS scores at a level between .58 and .63.

Normative data are not appropriate for the FACS since a client with normal language would obtain perfect scores on all ratings. However, means for the aphasic and the traumatic brain injury (TBI) group are given and show the sensitivity of the FACS. This is further documented by an account of the discrimination of the scores between groups of patients with different levels of severity of impairment.

In comparison with the CADL, the FACS uses ratings based on general observations rather than on observations under specific conditions of testing.

FUNCTIONAL COMMUNICATION PROFILE

The Functional Communication Profile (FCP) (M. T. Sarno, 1969; M. L. Taylor, 1965) is designed to measure natural language use in everyday communication. The FCP attempts to index the aphasic patient's ability to use language in common situations, relative to the patient's estimated premorbid level of ability. "Normal" performance on the profile is defined by the clinician's skilled estimation of the patient's previous language ability based on available evidence (e.g., education level, occupation, interviews with family members). The effectiveness of a clinician-based rating scale of this type is directly related to the experience and skill of the user; therefore, the FCP is not recommended for use by testing technicians or clinicians with limited experience, or in settings where few adult aphasics are likely to be seen. Its usefulness may also be limited in situations where little premorbid information is available.

The clinician's primary role is to create an informal rapport with the patient that allows the clinician to observe the patient's natural communicative behavior without resorting to formal testing. Forty-five behaviors are rated on a 9-point scale of current ability as a proportion of estimated former ability. The scale ranges from "normal" (100%) to "absent" (0%) ability. Examples of functional abilities include the abilities to indicate yes or no, to read newspaper headlines, and to make change. The 45 behaviors are clustered into five categories: Movement, Speaking, Understanding, Reading, and Miscellaneous (e.g., calculation and writing) abilities. Over-

all cluster scores are obtained by determining the mean rating of the items in a cluster.

Despite the subjective clinical nature of the scoring system, M. T. Sarno (1969) reported interrater reliability coefficients larger than .87 for each of the five FCP categories.

Gains on psychometric testing do not automatically imply improved functional abilities, and, conversely, functional gains may not alter diagnostic classification. The distinction between functional ratings and psychometrically measured language functioning was examined by J. E. Sarno et al. (1971). Measurements of improvement were determined by comparing original and follow-up performances on the NCCEA Visual Naming and Identification by Sentence (i.e., TT) subtests and the FCP Speaking and Understanding subscales. Only a modest relationship was found between the original and follow-up scores on each of the two speech measurements (i.e., NCCEA Visual Naming and FCP Speaking), and no correlation was found between score changes on the two comprehension measures (NCCEA Identification by Sentence and FCP Understanding). On the other hand, M. T. Sarno and Levita (1981) reported some concordance between NCCEA TT performance and FCP Understanding in the examination of global aphasics at 1-year follow-up examinations.

The information obtained from the FCP is not designed to replace a comprehensive examination of the aphasic patient's language abilities and disabilities. Rather, its goal is to provide information about natural communication capacity (M. T. Sarno, 1984a). The information yielded by a properly administered FCP may well translate more easily into a description of the patient's everyday capabilities than the information provided by a standard comprehensive examination. It is not a diagnostic test. For example, M. T. Sarno, Buonaguro, and Levita (1987) found that scores for fluent (Wernicke's) and nonfluent (Broca's) aphasics on the FCP were highly similar after age, education, and time of onset were controlled. When properly used, the FCP may provide information on the functional consequences of the patient's aphasic condition that is not otherwise (except anecdotally) available. Studies examining the accuracy of medical personnel and family members in estimating or predicting the aphasic patient's performance levels (e.g., McClenahan, Johnston, & Densham, 1992) indicate the potential educational value of the results of the FCP and similar measures in counseling the family and in fine-tuning therapist and caregiver communicative interactions with the patient. Repeated FCP administration may provide information on the recovery process of functionally relevant communicative ability (Sands, Sarno, & Shankweiler, 1969; M. T. Sarno, Buonaguro, & Levita, 1985; M. T. Sarno & Levita, 1979).

None of the diagnostic tests discussed later in this chapter provides di-

rect measurements of functional communication. Hence, any of the described communication profiles would make a significant addition to aphasia assessment in association with a comprehensive diagnostic battery.

Comprehensive Examinations

Comprehensive examinations of the aphasic's language ability seek a diverse sampling of performances at different levels of task difficulty along all dimensions or functions that the test author deems relevant to language disability. Examples of dimensions common to most of these tests include naming, oral expression, auditory comprehension, repetition, reading ability, and writing ability. Other dimensions vary according to the theoretical orientation of the authors.

For practical purposes, we limit the current review to instruments designed for and/or primarily used in English. Tests developed and available mainly in another language (e.g., Aachen Aphasia Battery; Willmes, Poeck, Weniger, & Huber, 1983) are excluded. We also omit an older, infrequently used instrument (Appraisal of Language Disturbance; Emerick, 1971).

ARIZONA BATTERY FOR COMMUNICATION DISORDERS OF DEMENTIA

The Arizona Battery for Communication Disorders of Dementia (ABCD) (Bayles & Tomoeda, 1990) is designed as a comprehensive assessment of language and other communicative functions in patients with dementia, and includes four screening tasks: Speech Discrimination, Visual Perception and Literacy (reading sentences), Visual Field (circling all *A*'s on a page of randomly scattered letters), and Visual Agnosia (naming or describing pictured objects). These tasks are designed to alert the examiner to other disorders that may interfere with communicative functioning. The main test consists of 14 subtests. A number of these subtests reflect traditional items found in most batteries, such as confrontation naming, category (animal) fluency, yes/no comprehension, and repetition. The battery also includes assessment of mental status (i.e., orientation), free-speech description of three pictured objects, a subtest similar to the Wechsler Vocabulary task, figure copying, and object drawing. Importantly, the ABCD differs from more typical aphasia batteries by measuring immediate and delayed recall of a story containing 17 pieces of information.

The response record provides relatively clear scoring guidelines, allowing 1 point for each item or part of an item. The authors suggest converting raw scores into "summary scores" between 1 and 5 to make subtest

scores comparable to each other. The summary scores are based on the performance of 50 healthy subjects of age (mean = 71 years), premorbid IQ, and education similar to that found in dementing patients. A summary score of 5 was achieved by most normal subjects, a score of 3 to 5 by patients with mild dementia, and a score of 2 to 5 by patients with moderate dementia. A construct summary score can be obtained by averaging summary scores across subtests contributing to each of five major areas (Mental Status, Episodic Memory, Linguistic Expression, Linguistic Comprehension, Verbal Visuospatial Construction). An overall performance score can be calculated by averaging across all subtests.

The ABCD has been used in only a few published studies. The manual reports 1-week retest reliability for 20 patients with AD as ranging from .01 (Reading Comprehension, Words) to .86 (Figure Copying), and concordance rates from .65 to .87. Internal consistency is high for all subtests (.63 to .98). The test has been successfully used in the United Kingdom without a change of items (Armstrong, Borthwick, Boyles, & Tomoeda, 1996).

Criterion validity has been demonstrated by highly significant correct classification rates between 50 normal and 50 AD subjects. Correlational validity of individual subtests with several measures of the severity of dementia in 50 AD patients ranged from .59 to .85. The test has also been used in a study of patients with multiple sclerosis (Wallace & Holmes, 1993), and in a comparison of semantic and phonetic (letter) word fluency by Bayles, Salmon, Tomoeda, Jacobs, & Caffrey, 1989).

The ABCD was standardized on 50 healthy elderly subjects with a mean age of 71 and slightly higher than average education and premorbid IQ. These subjects also form the basis for transformations into summary scores. It is not clear whether the test is subject to age, education, or gender effects, or whether a profile of subtest scores may contribute to differential diagnoses.

The current version of the ABCD is obviously still in the research stage and requires further work and independent confirmation. The test provides a wide range of measures similar to the WAB or the NCCEA, but also includes a general measure of mental status, several measures of memory, and diverse screening measures for visual perception, visual fields and/or neglect, auditory discrimination, and visual agnosia. These additional features are suitable for a quick, superficial assessment of the status of mental functions of dementing patients in the context of speech therapy. However, neuropsychologists may wish to use more detailed and better validated tests for these areas. In fact, one could use the MMSE, the BNT, FAS-COWA, and a list-learning task in half the time and obtain information similar to that tested in the ABCD. Although designed for elderly patients with dementia, the ABCD may be suitable for a larger range of pa-

tients, or for differential diagnosis with other dementing conditions or specific aphasia syndromes.

BOSTON ASSESSMENT OF SEVERE APHASIA

The purposes of the Boston Assessment of Severe Aphasia (BASA) (Helm-Estabrooks, Ramsberger, Moyan, & Nicholas, 1989) are to provide a full assessment of language and other communicative functions in severely aphasic patients, and to identify those abilities that might form the initial steps of direct rehabilitation. It consists of 61 items in 15 subtests: Social Greetings and Simple Conversation; Personally Relevant Yes/No Questions; Orientation to Time and Place; Bucco-Facial Praxis; Sustained "Ah" and Singing; Repetition; Limb Praxis; Comprehension of Number Symbols; Object Naming; Action Picture Items; Comprehension of Coin Names; Famous Faces; Emotional Words, Phrases, and Symbols; Visuo-Spatial Items; and Signing One's Name.

Several items can be administered in a relatively informal manner or as part of a conversation. The test takes approximately 30 to 40 min. Items are scored at several levels of performance, depending on the task demands, from no verbal or gestural response and partially communicative gestural response, to fully communicative verbal response and verbal response with affective quality. For each subtest, exact scoring criteria are provided. Correct responses are summed across five item clusters (auditory comprehension, praxis, oral–gestural expression, reading comprehension, gesture recognition + writing + visuo-spatial tasks). The total of correct responses forms the BASA total score. The total of other responses can also be summed across clusters and for the total test.

Internal consistency is high (between .72 and .89 for the five areas and .94 for the total score, slightly lower in global aphasics). Test–retest reliability after 2 months ranged from .52 to .73 for the area scores, and was .74 for the total score. Interrater reliability ranged from 80 to 100% in two patients. Concurrent validity with the BDAE is modest to adequate. This is probably to be expected because of the low difficulty range of the BASA subtests and the wide range of the BDAE. The correlation for 43 patients between BASA total score and BDAE aphasia severity rating was .67. Cluster total score correlations ranged from .44 to .76, suggesting some independence of each area score. A factor analysis showed expressive, visuospatial, and comprehensive language factors.

The BASA samples a wide range of communicative functions at a relatively low level, and lends itself to bedside examination of severely language-impaired patients. The scoring of affect and perseveration, and of partial verbal and gestural responses can provide useful information for the examiner and others, but so far these have not been further investigat-

ed. Townes (1995) states that the inclusion of gestural communication is a unique feature of the BASA, but this also makes extra demands on the examiner's attention during testing.

Norms are presented as preliminary and are based on the performance of 111 patients with severe aphasia, including 47 with global aphasia. To use the norms, raw scores for the five areas are converted into standard scores, which in turn can be used to assign a percentile rank within this aphasic population. Norms for normal healthy speakers are not provided because they would presumably achieve a perfect score. Effects of age, gender, or education are not reported.

The BASA is a useful addition to the other comprehensive batteries in that it provides the opportunity for a broad communication assessment in severely aphasic patients by extending the range of items in the lowest range of performance. So far, research with this relatively new instrument has been limited.

BOSTON DIAGNOSTIC APHASIA EXAMINATION

The original Boston Diagnostic Aphasia Examination (BDAE) (Goodglass & Kaplan, 1972) has been revised (Goodglass, Kaplan, & Weintraub, 1983). The primary focus of the BDAE, which remains one of today's most popular aphasia examinations (e.g., Beele, Davies, & Muller, 1984), is the diagnosis of classic anatomically based aphasic syndromes. This diagnostic goal is attained by comprehensive sampling of language components that have previously proven themselves valuable in the identification of aphasic syndromes.

Goodglass and Kaplan stated that the design of their instrument is based on the observation that various components of language function may be selectively damaged by central nervous system (CNS) lesions; this selectivity is an indication of the anatomical neural organization of language and the functional interactions of various parts of the language system. A number of studies have validated this stated purpose (e.g., Meffer & Jeffrey, 1984; Naeser & Hayward, 1978).

The BDAE is divided into five language-related sections: (a) conversational and expository speech, (b) auditory comprehension, (c) oral expression, (d) understanding written language, and (e) writing. Each section contains a variety of subtests. Each subtest attempts to measure the specific function in as purely isolated a manner as possible.

The detailed manner of examining conversational and expository speech remains an important and relatively unique aspect of the BDAE. A "speech characteristics profile" is derived from samples of both free-conversational speech and narrative speech in the description of a line drawing (the "cookie theft" card). The profile indexes verbal prosody (melodic

line), fluency, articulation, grammatical level, paraphasias, and word-finding difficulties. Repetition and auditory comprehension are also rated, but ratings are derived from subtest performances. Finally, an overall "severity rating scale" can be determined from conversational speech samples, ranging from "no usable speech or auditory comprehension" (a score of 0) to "minimal discernable speech handicaps" (a score of 5). The reliability of the speech characteristic profile was examined for the original BDAE by three judges who rated the tape-recorded speech samples of 99 patients. The lowest correlations were .78 and .79 for word-finding difficulties and paraphasias, respectively; the other dimensions had coefficients of at least .85. Other interrater agreement studies have also shown satisfactory results (Davis, 1993).

Auditory comprehension is tested by word discrimination (pointing to objects, etc., on cards), body-part identification, commands (e.g., "make a fist"), and complex ideational material ("will a stone sink in water?").

Oral expression is gauged by oral agility (mouth movements); verbal agility (rapid repetition of words); automated sequences (e.g., reciting days of the week); recitation, singing, and rhythm (e.g., reciting nursery rhymes); repetition of words of increasing length and difficulty; and repeating phrases and sentences of high and low probability. Oral expression also includes word reading, responsive naming ("what do we tell time with?"), visual confrontational naming, animal naming, and oral sentence reading.

Understanding written language is assessed by symbol and word discrimination, word recognition, comprehension of oral spelling, word–picture matching, and reading sentences and paragraphs. Writing includes a 4-point rating of the mechanics of writing, serial writing (alphabet and numbers), primer-level dictation (letters, numbers, and words), spelling to dictation, written confrontation naming, narrative writing ("cookie-theft" card), and sentences to dictation.

In addition, several optional supplementary language tests explore psycholinguistic aspects of language, for example, comprehension of prepositions of location, passive subject–object discrimination, possessive relationships, expression of indicative, interrogative, and conditional verb and tense usage; specific repetition tasks for conduction aphasia; naming by touch; instructions for exploring minor hand agraphia; and the BNT as an extended naming test. Supplementary nonlanguage tests (spatial–quantitative tests, formerly called the Boston Parietal Lobe Battery) are included as a separate chapter as well as on the profile summary. These include constructional apraxia (drawing to command), finger agnosia, right–left orientation, acalculia, and ideational apraxia, as well as commands to test for bucco-facial apraxia.

Internal consistency ranges from .98 for visual confrontation naming to .68 for body-part identification. Test–retest reliability is not reported.

Knowledge of the "Boston school" approach to aphasia classification is necessary to interpret the BDAE (e.g., Benson, 1979a). The speech characteristics profile and the severity ratings are central to diagnostic decision making with the BDAE; particularly important is the fluency–nonfluency dimension. More detailed diagnoses may incorporate corroborative information from the profile sheet delineating subtest performances.

The original BDAE was standardized on a sample of 207 aphasic patients with relatively distinct cerebrovascular accidents (CVAs) and isolated, well-defined symptoms. Although standardization on such a large sample of patients is psychometrically useful, the clinician whose referrals do not reflect this select sample (i.e., referrals with a different bias in symptomatology and severity) may not be able to reference her or his patients directly to the BDAE sample. The revised BDAE was standardized on a new sample of 242 patients. As in the first sample, selective aphasias produced by CVAs predominate. However, the authors report that this sample included more patients with larger lesions and more severe aphasic symptoms.

The BDAE was also standardized on 147 healthy normal adult subjects (Borod, Goodglass, & Kaplan, 1980) to provide cutoff scores at 2 *SD* below the mean and include age and education corrections. These means, ranges, and suggested cutoff scores are included in the manual.

Emery (1986) found only a minimal, insignificant decline of scores on all subtests when comparing 20 healthy adults aged 30 to 42 years with a similar group aged 75 to 93 years. Whitworth and Larson (1989) found no significant effects for gender and education in their sample. Heaton, Grant, and Matthews (1991) presented norms for the comprehension of complex material of the BDAE in scaled scores, corrected for gender, education, and age, that are based on 553 normal subjects. However, these norms should be used with caution, because the number of subjects in some of the cells is quite small. Rosselli, Florez, and Castro (1990) presented norms based on 180 normal Spanish speakers from Colombia, broken down by educational level and three age ranges (16 to 30, 31 to 50, 51 to 65 years). These data show a significant effect of educational level for most tasks, and an age effect for some of the tasks. Norms for the parietal lobe battery in older subjects (ages 40 to 89) were presented by Farver and Farver (1982).

Borod, Carper, Goodglass, and Naeser (1984) applied the battery to 163 right-handed aphasics and found four factors: construction, visual schemata, verbal components of the Gerstmann syndrome, and visual finger recognition. Impairment was strongest in patients with lesions in both left parietal and frontal areas. The spatial–quantitative tests, together with

the WAIS, were applied to right- and left-handed aphasics: left-handed aphasics were significantly poorer on both, especially on tasks involving visuospatial construction, suggesting that in left-handers, the left hemisphere is typically dominant for tasks usually thought of as right-hemisphere specific (Borod, Carper, Naeser, & Goodglass, 1985).

Construct validity has been examined by reviewing the intercorrelation matrix of the 43 language and 23 nonlanguage measures of the BDAE and by factor analysis. In an earlier analysis (Goodglass & Kaplan, 1972), a strong general language factor emerged, as expected, with other factors covering the spatial–quantitative–somatognostic domain, as well as articulation/grammatical fluency, auditory comprehension, and paraphasia domains. A second factor analysis (Goodglass & Kaplan, 1983), omitting ratings and nonlanguage tests, resulted in five factors (comprehension/reading/naming, recitation/repetition, writing, oral agility/singing/rhythm, auditory comprehension). When rating scales were included, three additional factors emerged (fluency, reading, paraphasia). The addition of the spatial–quantitative tests resulted in a 10-factor solution, including a strong spatial–quantitative factor, a finger identification factor, and a factor labeled "freedom from paraphasia." Discriminant validity between cases of Broca's, Wernicke's, conduction, and anomic aphasia was optimal when the following tests were entered into the equation: body-part identification, repetition of high-probability sentences, paraphasia rating, word-finding rating, phrase-length rating, and verbal paraphasias.

Divenyi and Robinson (1989) reported correlations of .86 and .93 of the auditory comprehension measured in the BDAE with the TT and with the respective part of the PICA. However, the BDAE auditory comprehension subtest was not an adequate predictor of auditory paragraph comprehension in independent standardized material (Brookshire & Nicholas, 1984); a second study (L. E. Nicholas, MacLennan, & Brookshire, 1986) showed that both aphasic and healthy subjects were able to answer a similar number of questions about a paragraph without having actually read the passage, suggesting a high passage dependency of this test. This dependency applied not only to the BDAE, but also to similar tasks in the MTDDA and the WAB. Dyadic interaction measures also did not correlate well with the BDAE (Behrmann & Penn, 1984). Decision rules for the "diagnosis" of the individual subtypes are not always clearly defined, although Reinvang and Graves (1975) attempted such clarification. Crary, Wertz, and Deal (1992) tried to isolate subtypes of aphasia empirically by means of a Q-type factor analysis for the BDAE and the closely related WAB; the resulting seven patient clusters (labeled Broca, anomic, global, Wernicke, conduction, and two unclassified clusters) agreed only poorly (in 38% of 47 patients) with the classification obtained using the classification rules of the test it-

self; the results were even worse for the WAB. The study, aside from its limited subject population for factor analytic studies and the use of a somewhat dated cluster-analysis technique, suggests that BDAE classification rules are based on clinical rather than construct validity. Similarly, Naeser and Hayward (1978) and Reinvang (1985) pointed out that scale profiles can aid in the classification but do not firmly classify patients into subtypes of aphasia. The test authors acknowledge that 30 to 80% of aphasic patients are not classifiable; this is also consistent with clinical experience that a majority of aphasic patients show mixed rather than pure symptomatology.

The BDAE predicted progress in therapy (Helm-Estabrooks & Ramsberger, 1986; Davidoff & Katz, 1985). In aphasics, word reading (Selnes, Niccum, Knopman, & Rubens, 1984) and confrontation naming (Knopman et al., 1984) showed striking improvement 6 months postinsult. Specifically, Marshall and Neuberger (1994) found that a measured pretreatment effort in self-correction (but not success of self-correction) and good auditory comprehension were related to improvement during treatment, as measured by the BDAE and the PICA. Narrative response to the Cookie-Theft card has been examined as a method of gleaning additional information about patients' self-monitoring and self-correction of errors. Nonaphasic and nondemented healthy elderly self-correct 72 to 92% of their errors in the description of this card (McNamara, Obler, Au, Durso, & Albert, 1992).

The test is lengthy (90 to 120 min) and probably more useful for assessments in detailed studies of aphasia and aphasia rehabilitation than as a routine language test included in a general assessment. The somewhat unwieldy number of tasks is clearly the result of trying to fit classical clinical–neurological testing into a psychometric format. The test includes useful directions for observing and recording many specific types of errors (e.g., paraphasias) found in aphasia, demonstrating the Boston process approach. However, even if the full-length test is not used, a number of subtests can be useful additions to clinical assessment depending on the presenting symptoms of the patient.

A Spanish (Goodglass & Kaplan, 1986), French (Mazaux & Orgogozo, 1985), and Hindu version (Kacker, Pandit, & Dua, 1991) are available. Computerized scoring and interpretation software is also available (Code, Heer, & Schofield, 1990).

MINNESOTA TEST FOR DIFFERENTIAL DIAGNOSIS OF APHASIA

The Minnesota Test for Differential Diagnosis of Aphasia (MTDDA) (Schuell, 1995, 1973) was ranked among the more popular batteries in 1984 (Beele et al., 1984), although few studies with the test have been published in recent years. It is a comprehensive examination designed to observe the

level at which language performance is impaired in each of the principal language modalities at different levels of task difficulty. To Schuell, the goal of a careful and comprehensive description of impairment in the aphasic patient is to provide a guide for effective therapeutic intervention.

The current version of the MTDDA is the result of numerous systematic revisions of the original experimental version of the late 1940s. The author employed empirical factor analytic techniques (Schuell, Jenkins, & Carroll, 1962) as well as clinical experience to construct and revise the test. The construction of the MTDDA reflects Schuell's theoretical view of aphasia as a unitary reduction of language that crosses all language modalities, which may or may not be complicated by perceptual or sensorimotor involvement, by various forms of dysarthria, or by other sequels of brain damage (Schuell, 1974b; Schuell & Jenkins, 1959; Schuell et al., 1964). The MTDDA is composed of five sections: auditory disturbances (represented by 9 subtests), visual and reading disturbances (9 subtests), speech and language disturbances (15 subtests), visuomotor and writing disturbances (10 subtests), and numerical relations and arithmetic processes (4 subtests). Within each section, the subtest order is generally arranged from the least difficult to the most difficult. Each section may be started at an estimated level of difficulty corresponding to the patient's ability, and then continued to the point where the patient fails 90% or more of the items. Both the test manual (Schuell, 1965) and the companion monograph (Schuell, 1973) describe supplementary tests that should be considered, as well as the factor and intercorrelation structure for the tests.

Differential diagnosis with the MTDDA identified five aphasia syndromes: simple aphasia, aphasia with visual involvement, aphasia with sensorimotor involvement, aphasia with scattered findings compatible with generalized brain damage, and an irreversible aphasia syndrome (Schuell, 1974a). Schuell (1966, 1973) also added two additional "minor syndromes": mild aphasia with persistent dysfluency (dysarthria), and aphasia with intermittent auditory imperception. However, as Zubrick and Smith (1979) pointed out, the MTDDA was not designed to deal with broader issues of aphasic differential diagnosis (e.g., distinguishing aphasia from memory loss, dementia, severe hearing loss, and confusional state). The test has been successfully used to measure language recovery after stroke and head trauma, and to show that language recovery is relatively independent from intelligence (Bayley & Powell, 1981; David & Skilbeck, 1984). Armstrong and Walker (1994) tested older adults with an MTDDA short version to examine any gender differences, but no differences were found.

The length of the MTDDA presents a problem for the user of the test. Short forms (Schuell, 1957) and "very short" forms (Powell, Bayley, &

Clark, 1980) have been created, reducing the 43 subtests to as few as four. Schuell herself was not impressed with the role of short examinations in the diagnosis of aphasic disorders. The large number of subtests includes many functions that exceed what some authors would consider the assessment of speech and language functions, and extend into material that has been a traditional component of many intelligence tests. Schuell's factor analysis may, on closer inspection, seem to reflect a major first "general" factor that is closely related to the *g* obtained in factor analyses of intelligence tests. Schuell and Jenkins (1959), however, considered this factor a general language factor, supporting their assumption about the unitary nature of language.

In summary, the MTDDA is an extensive examination. At the time, the test represented a major breakthrough in the development of comprehensive aphasia test instruments that met the requirements of both standardization and objectivity. Great care was taken in its construction, which used both clinical expertise and empirical technique. Potential users of the MTDDA should consider whether its length will be prohibitive in clinical settings. The user should also examine the theoretical bases of the MTDDA relative to the user's own conception of the nature of aphasic deficits, and balance its breadth against practical needs.

MULTILINGUAL APHASIA EXAMINATION

The benefits of having equivalent versions of a single aphasia examination for several language communities has been well stated by Benton (1967, 1969). The Multilingual Aphasia Examination (MAE) has been developed through the efforts of Benton and his collaborators to meet the requirements of a multilingual examination (Benton, Hamsher, Rey, & Sivan, 1994). The MAE is available in English (Benton et al., 1994) and Spanish (MAE-S; Rey & Benton, 1991). Chinese, French, German, Italian, and Portuguese versions of this test are being prepared. The different language versions of the MAE are functionally equivalent in content rather than simple translations. For example, COWA uses letters that have corresponding levels of difficulty in each language rather than identical letters. Hence, performance of the task in each language is functionally equivalent.

The MAE, a shortened and highly modified relative of the NCCEA, consists of seven subtests: Visual Naming, Sentence Repetition, COWA, Spelling, a version of the TT, Aural Comprehension of Words and Phrases, and Reading Comprehension of Words and Phrases. Two MAE rating scales are included. The first is a scale of speech articulation based on verbal performance throughout the test session. Ratings range from 0 ("speechless or usually unintelligible speech") to 8 ("normal speech"). The second scale encodes writing praxis, scored when possible by performance

on tasks of writing to dictation (from the MAE Spelling subtest); scores range from 0 ("illegible scrawl") to 8 ("good penmanship").

A practical and distinctive feature of the MAE is that alternate versions of Sentence Repetition, COWA, Spelling, and TT are available for repeat assessment of the patient. Hermann and Wyler (1988) provided a research example of the utility of MAE alternate forms in an examination of language behavior before and after temporal lobectomy in epileptic patients. Visual Naming requires naming of line drawings (whole objects and object details), which is more difficult than the naming of actual objects. COWA presents three letters of progressing association difficulty. The Spelling subtest permits oral spelling, writing to dictation, or block-letter spelling. The TT is composed of 22 commands at two levels of complexity, and is scored pass–fail. Aural and Reading Comprehension are administered in a multiple-choice format. Scoring adjustments for age and educational level are provided in the test manual.

The MAE manual provides standardized test instructions and normative information from a sample of 360 normal Iowa adults (aged 16 to 69 years) without a history of or evidence of neurological disability. A second validation sample of 50 aphasic patients is included, which may be used to discern aphasic subtypes. Normative information for 229 children (aged 6 to 12 years), based on a study by Schum, Sivan, and Benton (1989), is included in the manual. The MAE-S manual includes normative information from a sample of 234 normal Spanish-speaking adults (aged 18 to 70 years) from Texas and Puerto Rico. The Mayo Older Americans Normative Studies (MOANS; Ivnik, Malec, Smith, Tangelos, & Petersen, 1996) reported results with a large sample of normal adults between 55 and 97 years of age for two MAE subtests (COWA and TT). Because the Naming subtest may be especially sensitive to cultural experience, separate normative data have been obtained for urban inner-city Black residents (Roberts & Hamsher, 1984). The latest edition of the manual reports unpublished data by R. L. Schum and A. B. Sivan about the MAE performance of 60 healthy control subjects, aged 70 to 89 years. Stable performances were noted for subjects in their 70s, but relative performance decrements were found for those in their 80s, particularly on the Sentence Repetition and TT subtests. These studies were conducted with well-educated adults; the authors speculate that subjects with less education may show a steeper decline with age. Rather than looking for poor performance in depressed elderly, La Rue, Swan, and Carmelli (1995) emphasized good COWA performance in elderly adults scoring high on a measure of "zestfulness," suggesting that this is an example where COWA levels may show better than expected performance.

A study of discriminative validity between 115 normal and 48 aphasic

subjects with six of the MAE tests found that with the suggested cutoff scores, between 2.6 and 7.0% of normals, and between 14.4 and 64.6% of aphasics were misclassified by individual subtests. With failure on one subtest as a cutoff, 15% of controls and no aphasics were misclassified; with failure on two or more subtests, the misclassification rates were 3 and 4%, respectively (Jones & Benton, 1995). The TT proved to be the most divergent between the two groups; the two comprehension subtests were the least discriminative. Patients with left temporal lobe epilepsy also showed significant impairment on the MAE compared with epileptics with right hemisphere impairment (Hermann, Seidenberg, Haltiner, & Wyler, 1992; Hermann & Wyler, 1988). Concurrent validity of the Visual Naming subtest, which has 30 items, and the 60-item BNT (.86) was reported by Axelrod et al. (1994) in a diagnostically mixed sample of 100 adult patients with neurological or psychiatric histories.

Research shows that failure on the MAE Token Test is a sensitive indicator of the presence of acute confusional states (delirium) in nonaphasic medical inpatients (G. P. Lee & Hamsher, 1988). Levin and colleagues (Levin, Grossman, & Kelly, 1976; Levin, Grossman, Sarwar, & Meyers, 1981) have used the MAE to examine the linguistic performance of patients with closed head injuries. They documented a high frequency of naming errors, defective associative word-finding, and TT, and revealed the correlation of these with the severity of brain injury. A factor analysis of 16 aphasia battery subtests (including subtests of the MAE, NCCEA, and WAB) given to healthy Taiwanese volunteers (Hua, Chang, & Chen, 1997) suggested a major factor of verbal comprehension (including TT, Sentence Repetition, Digit Repetition, Visual Naming, Reading, and Aural Comprehension). A second factor was labeled effortful writing, and a third factor involved mainly verbal expression and word production.

In summary, it is hoped that the successful deployment of the MAE in a number of language communities will facilitate direct cross-community comparisons of case and sample data. Regardless of this research goal, clinical use of the English-language MAE suggests that it is an effective instrument which requires a relatively brief (usually under 45 min) administration time. In addition, the use in general clinical practice of individual subtests (viz., Visual Naming, TT, COWA) can serve as a good exploratory examination as to the presence of language deficits.

NEUROSENSORY CENTER COMPREHENSIVE EXAMINATION FOR APHASIA

The Neurosensory Center Comprehensive Examination for Aphasia (NCCEA) (Spreen & Benton, 1977; Spreen & Strauss, 1991) was designed to provide a comprehensive assessment of language comprehension, lan-

guage production, reading, and writing. Other stated goals of the NCCEA are to provide subtests that are sufficiently complex so that the clinician can obtain a relatively exact measure of performance level; to standardize and score performances such that necessary corrections for age, sex, and education can be made; to include nonlinguistic subtests to ensure valid interpretation of performance deficits on language tests as either linguistic in nature or due to other dysfunction; and to include specific subtests that could be used in aphasiology research (Benton, 1967).

The NCCEA is composed of 20 subtests that focus on the language function stated above and four "control" subtests of visual and tactile functioning. The test is designed to yield a description of the patient's profile of abilities and disabilities. NCCEA subtests include stimulus presentation in the visual, auditory, or tactile modality. The subtests measure visual object naming, description of object use, tactile object naming for each hand, sentence repetition, sentence construction, object identification by name, oral reading of names and sentences, oral reading of names and sentences for meaning, writing of object names, writing to dictation, copying sentences, and articulation. The sequential order of subtests provides a meaningful grouping into tests of name finding, immediate verbal memory, verbal production and fluency, receptive ability, reading, writing, and articulation. Eight of the 20 language subtests and three out of the four control subtests require the use of four sets of eight common objects displayed on trays. These objects are presented in order of difficulty, from least to most difficult. The four sets are matched for item difficulty and are equivalent in mean and distribution of difficulty for aphasic patients (mean percent correct is approximately 63% for all trays) and young children (mean acquisition age of names approximately 5:8 years); they are rotated throughout the battery.

Several subtests provide a set of items for initial testing, as well as a second set of items to be used only if errors occurred on the first set. This feature tends to shorten test administration in areas in which a patient has no difficulties. The second set of items provides more detailed quantitative information on problem areas. Isolated errors due to poor attention or other irrelevant causes will be reduced in importance if the second set of items is then passed correctly. The complete test takes between 45 and 120 min to administer.

The range of item difficulty is limited. In an attempt to avoid highly specialized or low-frequency vocabulary, the authors used only common objects for their object naming, identification, and similar tasks. As a result, the test has a rather low ceiling on some of the subtests, with the effect that very mild aphasic symptoms in highly educated patients may be missed. Other subtests, however, are "open ceiling" tests for which this limitation does not apply.

Scores on the NCCEA are determined by response correctness. Incorrect responses and mispronounced correct responses are recorded verbatim to yield qualitative performance information. An individual's performance on the NCCEA, when corrected for the effect of age and educational level, can be converted into percentile scores to yield relative levels of performance on each subtest and can be ranked on three different profile sheets, comparing the patient's performance to samples of normal adults (Profile A), aphasic patients (Profile B), and nonaphasic brain-damaged patients (Profile C). The aphasic and nonaphasic brain-damaged samples consist of consecutive referrals for neuropsychological evaluation in acute-care hospital settings.

Because the NCCEA was primarily designed to assess patients with aphasia or aphasia-type complaints, patients without language problems and normal controls tend to obtain ceiling scores. Therefore, the test cannot be used to measure language ability in normal adults, although the language development in children has been successfully measured with most subtests up to a ceiling age from 8 to 13 years (Gaddes & Crockett, 1975).

An empirical study with 353 children, aged 5:5 to 13:5 (Crockett, 1974), found that seven factors described the content of the NCCEA in that population: reading/writing, verbal memory, name finding, auditory comprehension, syntactic fluency, reversal of digits, and repeating digits.

One-year retest reliability in older adults for selected subtests has been reported as satisfactory (Word Fluency, .70; Visual Naming, .82; TT, .50; Snow & Tierney, 1988).

Construct validity was examined in two studies by Crockett (1976, 1977). The first examined the discrimination of groups of aphasic patients based on ratings of verbal productions and divided on the basis of the Howes/Geschwind two-type, and the Weisenberg/McBride three-type typologies. Neither of the two models showed significant multivariate differences. The second study showed significant multivariate differences on the NCCEA between four types of aphasia empirically derived from ratings of verbal production by hierarchical grouping analysis. Two of the four types appeared to be similar to Howes' two types, a third appeared to reflect Schuell's single dimension of language disorder, and a fourth seemed to be characterized primarily by memory impairment. Concurrent validity with the WAB was demonstrated by Kertesz (1979). Concurrent validity for changes in language functioning during therapy was reported by Kenin and Swisher (1972).

Predictive validity was established in a study by Lawriw (1976), who also presented a successful cross-validation between patient groups from Iowa City, New York City, and Victoria, British Columbia. Kenin and Swisher (1972) and Ludlow (1977) investigated patterns of recovery from aphasia; improvement was best reflected in writing from copy and in tests

of comprehension, whereas expressive performance showed the least improvement. Single-word reception or production was more readily recovered than that of longer verbal units. The authors mentioned that reading, writing, and oral production items were not sufficiently difficult for patients at an advanced stage of recovery. M. T. Sarno (1984b) described significant differences between aphasic, dysarthric/subclinical, and subclinical aphasic patients on Visual Object Naming, Sentence Repetition, Word Fluency, and the TT. Patients with AD scored significantly lower in the areas of verbal expression, auditory comprehension, repetition, reading, and writing compared with age-matched, nonneurological controls (Murdoch, Chenery, Wilks, & Boyle, 1987). M. T. Sarno and Levita (1981) described recovery from global aphasia during the first 3 months after stroke.

The 1977 NCCEA normative data for an aphasic reference group are based on 206 unselected referrals to hospital and clinic services in Iowa City, New York City, and Victoria. Although the concept of "averaging" across aphasic patients, also used in the BDAE, disregards the different types of aphasia, this procedure allows the profiling of individual patients against that reference group; that is, an individual patient's subtype will stand out more clearly. However, when patients from another referral source (e.g., patients in rehabilitation or patients with residual aphasia) are seen, the reference group may no longer be appropriate.

Normative data for most subtests remain stable through the age span up to age 64. Results of studies with a geriatric population on some of the tests (Montgomery, 1982) showed only a minor decline of 1 or 2 points, which has been incorporated into the age and education correction rules. A study by Tuokko and Woodward (1996) of elderly subjects in Vancouver, British Columbia, for example, showed a mean for Visual Naming of 16 (ceiling score) for subjects below 60 years of age to be maintained up to age 79, and drop to a mean of 15.44 for subjects 80 years and older. Similarly, Description of Use showed a ceiling score of 16 up to the age of 74; the mean for subjects 75 years and older was 15.78. Tactile Naming (right hand) showed a mean of 15.66 for subjects up to the age of 79, and 14.89 for subjects 80 years and older. Tactile Naming (left hand) showed a mean of 16 for subjects under 60, and means between 15.14 and 15.57 for subjects between the ages of 60 and 79 years, but a mean of 12.44 for subjects 80 years and older.

Normative data for children between the ages of 6 and 13 are presented. The means were merged from studies by Gaddes and Crockett (1975) and Hamsher (1980), as the differences between the two sources (Victoria and Milwaukee) were minimal.

The test has also been adapted into Italian, Japanese, and Spanish. Writing from Dictation and Writing from Copy were used in a Chinese study (Hua et al., 1997).

In summary, the NCCEA provides a comprehensive assessment of language functions for aphasic patients without the use of a specific language model and without applying a specific approach to delineate diagnostic types of aphasia. Psychometric development of the test has been slow, and few new studies from the last decade are available. The development of three different profile sheets for score evaluation is a distinct asset. On the other hand, the low ceiling of some of the subtests suggests that some aspects of language functioning in mildly or borderline aphasic patients cannot be adequately measured.

PORCH INDEX OF COMMUNICATION ABILITY

The Porch Index of Communication Ability (PICA) (Porch, 1967, 1973, 1981) is designed to assess verbal, gestural, and graphic responsiveness subsequent to brain damage. Unlike classificatory instruments, the PICA is designed to categorize the nature of the aphasic's ability to respond, modality of response, and quality of response to task demands. A prime use of PICA has been in assessing patient performance on multiple occasions postonset to determine recovery trends. The PICA has 18 subtests: four verbal, eight gestural, and six graphic. A high degree of homogeneity among subtests is established through the repeated use of 10 common, everyday objects of equal difficulty (e.g., a key, a cigarette) for a majority of the subtests. This allows examination of fluctuations of performance over time (but may also introduce a practice effect). Subtests were created to conform to a model of language functioning involving several possible input modalities and outgoing responses in several possible output modalities. Five modalities are assessed: auditory comprehension, visual comprehension, written expression, verbal expression, and pantomime. The test takes $\frac{1}{2}$ to 2 hr to administer.

A persistent problem in recording responses involves assessing and quantifying the given response as one of a wide variety of possible responses. As a compromise between two possible extremes (i.e., longhand notation of response characteristics and simple pass–fail dichotomies), the PICA uses a 16-point multidimensional scoring system, an attempt by the author to integrate the strengths of the two approaches while minimizing their weaknesses.

A given response to a PICA test item is evaluated along five dimensions: accuracy, responsiveness, completeness, promptness, and efficiency. A scoring system that considers all possible permutations of these five dimensions is, for all practical purposes, impossible and meaningless. Hence, 16 categories have been identified that represent various relevant combinations of the five dimensions, resulting in a 16-point ranked scale from "no response" (1) to "complex response" (16). For example, any at-

tempt by the patient to perform on the task is scored at least a 6; all accurate responses are scored at least an 8. Additional points are given for a correct response after repeated instructions, for self-corrected responses, responsive ease, completeness, promptness, and efficiency. Porch (1967) reported the viability of the rank ordering of the 16 categories by pointing out the high agreement between PICA category ordering and ranking of categories by 12 speech pathologists. The individual item scores (180 possible) are transformed into an overall performance score, several modality scores, and individual subtest scores. The overall performance score is considered as the best single index of the patient's general communicative ability. Modality scores yield information on the relative capacity for verbal, gestural, and graphic communication. Use of mean values require that statistical and conceptual assumptions of equal intervals between category levels be met. Whether this assumption is legitimate for the PICA has been the subject of debate (e.g., Lincoln & Pickersgill, 1981; A. D. Martin, 1977; McNeil, 1979). Although single-item responses can be categorized on the 16-point scale (i.e., a score of 12 represents an incomplete response), mean values cannot be so categorized. Hence, mean scores cannot categorize the method by which the patient generally communicates, but can only represent a performance level relative to other normative values or to the patient's derived scores.

Data indicating that the PICA shows high interrater reliability as well as high test–retest reliability are provided in the manual. Holland (1980) reported concurrent validity of the PICA as .93 with the CADL, .86 with the FCP, and .88 with the BDAE. Lendrem and Lincoln (1985) found that the PICA given at 4 weeks postonset successfully predicted spontaneous recovery at 6 months in 32 male stroke victims between 48 and 80 years of age. Age, but not type of aphasia, was related to rate of recovery in 87 stroke victims between the ages of 38 and 92 years (Lendrem & McGuirk, 1988). However, Lincoln and McGuirk (1986), examining 124 patients between the ages of 38 and 92, 4 weeks and 34 weeks after stroke, noted that groups with and without treatment did not reach the level of recovery predicted by the PICA. They concluded that such predictions are not accurate enough for clinical practice.

Percentile data for all principal transformed scores from normative samples of 357 left hemisphere damaged patients, 96 right hemisphere, and 100 bilaterally damaged patients are presented in the manual (Porch, 1981). In addition to providing relative information for these transformed scores, percentiles are also used to determine a given patient's "aphasia recovery curve," by factoring in the overall test percentile, the mean percentile of the nine highest scored subtests, and the mean percentile of the nine lowest scored subtests. Predictions on the scope of recovery can be attempted

from this curve (Porch, Collins, Wertz, & Friden, 1980). Studying 110 aphasic patients with the PICA, Marshall and Tompkins (1983) found that age, health, etiology, and time since onset were good predictors of improvement during the course of treatment. Training patients in functional communication efficacy, on the other hand, did not show improvements on the PICA, although improvement was found on the CADL (Aten, Caligiure, & Holland, 1982).

DiSimoni, Keith, Holt, and Darley (1975) found a high degree of redundancy among PICA subtests and concluded that a shortened form of the test may be more useful. DiSimoni, Keith, and Darley (1980) described two short versions of the PICA that require only one-third of the time used for the full-length test. A preliminary Portuguese (Brazilian) version is available (Gunther, 1981).

In summary, the PICA is a well-developed and standardized test instrument that has been extensively used in rehabilitation settings to track recovery, although we found few research studies with the test in recent years. The multidimensional scoring system has become the most criticized aspect of the test, only partly remedied in the second edition. Two other shortcomings of the PICA include the paucity of sampling auditory comprehension and the misleading labeling of several subtests as gestural when they entail other specific behaviors. McNeil (1979) suggested that such criticism should not turn clinicians away from the PICA as a test instrument, but rather should make them more cautious interpreters of PICA results.

WESTERN APHASIA BATTERY

The Western Aphasia Battery (WAB) (Kertesz, 1979, 1982) is a close relative of the BDAE and shares with it the diagnostic goal of classifying aphasia subtypes and rating the severity of the aphasic impairment. The examination comprises four language and 3 performance domains. Syndrome classification is determined by the pattern of performance on the four language subtests, which assess spontaneous speech, comprehension, repetition, and naming. Weighted performance on these language subtests yields an overall measure of severity of aphasia, the Aphasia Quotient (AQ). Stepwise regression analysis has shown that, of the AQ constituents, the Information Content rating is most highly correlated with the AQ (Crary & Rothi, 1989). The three performance areas—reading and writing, praxis, construction, and the Raven's Colored Progressive Matrices–yield a second summary measure: the Performance Quotient (PQ). Finally, the AQ and the PQ are summed to form a Cortical Quotient (CQ). Criteria for the classification of eight classic aphasic syndromes are described based on the language subtest performances of 375 aphasic patients with various eti-

ologies (mostly CVA) and 162 normal individuals. Classification is forced into one of these syndromes; unclassifiable aphasia subtypes or mixed presentations are not addressed. In this respect, WAB classification is more rigid than in any other diagnostic battery. This rigidity may have relatively more appeal to researchers than clinicians.

Spontaneous speech is assessed both in response to questioning and in the patient's description of a line drawing, similar to the BDAE. Speech is rated on two 10-point scales: information content and fluency (the fluency scale incorporates both grammatical competence and the presence of paraphasias). Comprehension is assessed by yes/no questions that may be answered in either verbal or nonverbal fashion, by word recognition, and by performance to sequential commands. Repetition has 15 items that are scored as correct, in phonemic error (partial credit), or as an error. Naming is composed of object naming (without cuing or, if necessary, with tactile and/or phonemic cuing), word fluency (animals), sentence completion, and responsive speech. Test items were selected to provide a wide enough range of difficulty for assessing all levels of severity.

An uncommon feature of this test's structure is the dissociation from language performance of reading and writing abilities, which, along with nonverbal measures, form part of the PQ. Shewan (1986) reunited the spoken language section (i.e., the AQ tests) with reading and writing as part of a scale called the Language Quotient (LQ), and provided a detailed account of reliability and validity for this addition to the original WAB format. The LQ is weighted so that 60% reflects spoken language performance, and 40% reflects written language performance. Shewan's report emphasized the relation between the LQ measure and the severity of the aphasic disorder. Crary and Rothi (1989) demonstrated internal consistency for the 10 language subtests with protocols of 100 aphasic patients: All subtests correlated highly with the AQ.

WAB standardization information and reliability, and validity data were provided by Kertesz and Poole (1974) and then updated by Kertesz (1979) and Shewan and Kertesz (1980). The WAB clearly meets standard rules of test construction, although it ranked last in a review of nine aphasia tests by Skenes and McCauley (1985). The WAB manifests good internal consistency and high interrater and intrarater reliabilities. High test–retest reliability has been reported for a sample of 38 chronic aphasic patients. Successful criterion validity has been described by the author. Aphasics were differentiated from non-brain-damaged adults in their WAB performance; the AQ distinguished aphasics from non-brain-damaged controls. Construct validity was assessed in a sample of 15 patients who were examined with both the WAB and the NCCEA; there were high intercorrelations between corresponding subtests ranging from .82 for spontaneous speech

subtests to .95 for comprehension subtests. The WAB line drawing, used to elicit spontaneous speech, generated more enumerations, produced at a slower rate, than the pictures in the BDAE or the MTDDA (Correia, Brookshire, & Nicholas, 1990). McClenahan, Johnston, and Densham (1992) compared the accuracy of estimation of comprehension problems, using the WAB and the FCP, with estimates made by doctors, nurses, and relatives. The comprehension sections of the two tests usually overestimated the patient's ability compared with the judged estimates, but was more accurate for patients with mild problems. Interestingly, length of relationship or educational background did not affect the correctness of judgment, but high or low level of confidence in the judgment by the individual making it did have an influence. One study examined the validity of cutoff scores on the CQ (Fromm, Greenhouse, Holland, & Swindell, 1986; Fromm & Holland, 1989); a cutoff score of 90 points showed good sensitivity and specificity. The authors state that the language subtests can be administered in approximately $1\frac{1}{2}$ hr, but the full WAB might require at least two 2-hr sessions to complete. The test manual is far less detailed than Kertesz' book (1979), which remains a good, detailed introduction to the WAB.

The WAB has established itself as a useful classificatory research instrument, helped by its inclusive objective classification rules and its summary measures. The main subtypes obtained with the WAB are global, Broca's, Wernicke's, conduction, and anomic aphasias (Shewan & Kertesz, 1980). Studies with the test include novel cluster analytic taxonomies of aphasic syndromes of different etiologies over time (Kertesz & Phipps, 1980), and the evolution of aphasic syndromes during the course of recovery (Kertesz, 1981) and during therapy (R. Lesser, Bryan, Anderson, & Hilton, 1986; Shewan & Kertesz, 1984). Crary et al. (1992) used cluster analysis with a small group of 47 patients and found that cluster membership corresponded only poorly (30% of cases) to the classification suggested by the WAB classification rules. Other research investigated the role of activation of the nondominant hemisphere during word repetition (as shown in PET studies; Ohyama et al., 1996), the relation between aphasia and nonverbal intelligence (Kertesz & McCabe, 1975), the relation between language and praxis (Gonzales-Rothi & Heilman, 1984; Kertesz & Hooper, 1982), comparative diagnostic classification between a Portuguese version of the WAB and a Portuguese aphasia examination (Ferro & Kertesz, 1987), and the efficacy of aphasia treatment. A study with 193 Norwegian aphasics found good agreement (85%) between classifications made with the WAB and the Norwegian Basic Aphasia Test, but only after a large number of mixed or otherwise unclassifiable patients were excluded (Sundet & Engvik, 1985); a cluster analysis of this group showed four types of aphasia—global, Wernicke, Broca, and anomia—each at a major and minor im-

pairment level. Butterworth, Howard, and McLoughlin (1984) found that semantic errors were not related to diagnostic grouping but to severity of aphasia. Semantic errors occurred on both naming and auditory comprehension, suggesting that both have a common underlying deficit.

Like the BDAE, the WAB offers a measure of spontaneous speech. The WAB measure of spontaneous speech, however, appears to be less comprehensive than the BDAE method; for example, fluency, grammatical competence, and the extent of paraphasic errors are combined into a single scale on the WAB, whereas they are assessed independently on the BDAE. Shewan and Donner (1988) also noted that the WAB spontaneous speech subtest does not provide comprehensive information compared with other tests designed to evaluate this aspect of language. The repetition test does not appear to be as encompassing or as well structured as other repetition tasks (e.g., NCCEA or MAE Sentence Repetition). Controlled associative fluency is measured as category (animal), not phonetic (letter), naming.

Mark and Thomas (1992) found auditory–verbal comprehension, as measured on the WAB, to be strongly related to outcome. This study did not find strong relationships between neuroradiological measures and outcome, whereas Metter and colleagues (1990; Metter & Jackson, 1992) found significant relations between outcome and PET scan findings in the left temporo-parietal cortex (left angular, supramarginal, postero-supero-temporal gyri). L. E. Nicholas et al. (1986) found that passage comprehension could be answered correctly better than chance without reading the sentences which the items purported to test, a finding that also applied to passage comprehension in the BDAE and the MTDDA.

In summary, the primary purpose of the WAB, like the BDAE, is diagnostic: the classification of aphasic performances into traditional aphasic syndrome subtypes. Explicit decision rules about which classification applies in an individual case are provided, but the test operates on the assumption that all cases can be clearly classified as one of eight basic types. Such clear-cut classification has limited meaning for the "mixed" aphasias that occur much more often in clinical practice than this classification system suggests. The WAB has also been used successfully in studies of recovery and treatment. It offers an additional choice for aphasia assessment which is on par with contemporary research in the field, and has found continuing use in research studies. The inclusion of a "cortical quotient" is new and unusual in an aphasia battery. The concept of a CQ, using a mixture of language and performance measures (including the Raven Progressive Matrices), seems to compete with the traditional concept of IQ (general intellectual functioning) without providing the solid psychometric and theoretical foundations for an intelligence test.

Assessment of Aphasia in Children

The major obstacle encountered in designing assessment methods for children is that language ability increases with chronological age in the normal child and there is relatively high variability from child to child within a given age level. Full language competency is not reached until 12 to 14 years of age (depending on the definition of competency); after this age, further development takes place in terms of increased vocabulary, grammatical complexity, awareness of rules of generative grammar, and so on. For these reasons, any assessment method for children requires the establishment of normative data for each year (or half-year) of age. Because of somewhat different rates of growth of language abilities in boys and girls, separate norms for each sex are also required. Obviously, the construction of suitable tests for children requires much more extensive psychometric work than does the construction of tests for adults.

Several tests of normal language development in children are available, such as the Illinois Test of Psycholinguistic Abilities (ITPA; Kirk, McCarthy, & Kirk, 1968), but few have been constructed or restandardized for children for the specific purpose of aphasia assessment (see review by Eisenson, 1972; Sattler, 1988). The Pediatric Evaluation of Disability Inventory (PEDI; Haley, Coster, Ludlow, Haltiwanger, & Andrellos, 1992) provides a first functional profile of ratings designed for children. It includes ratings based on observation or parent report for self-care, mobility, and social function; the social function domain includes detailed ratings of comprehension of word meaning, comprehension of sentence complexity, functional use of expressive communication, and complexity of expressive communication.

Among the brief or specific-purpose assessment methods, adaptations are common. Several adaptations of the sentence repetition method have been attempted. One experimental technique, which used 24 sentences that varied according to grammatical complexity, was used in a population of congenitally aphasic children (Bliss & Peterson, 1975). Adaptations of the COWA change from words starting with a given letter to animal names or similar categories or to words beginning with a specific sound ("*sh*-words") for children who cannot be expected to have a sufficient knowledge of spelling. DiSimoni (1978) published an adaptation of the Token Test for Children, standardized with 1304 children from preschool age 3 to grade 6 (age 12:6) and drawn from a mixed suburban population. The test manual also reviews several other studies investigating the scoring criteria, as well as aspects of concurrent validity with other tests of auditory comprehension, including the ITPA and the PPVT. The TT has also been investigated as a discriminator between aphasic and other brain-damaged

children, and in relation to socioeconomic status of the home, an important aspect of language development in children (Gutbrod & Michel, 1986), and in relation to speech training in language-delayed children (Alexander & Frost, 1982). Syntactic comprehension in children was also examined with the TT and the BDAE Auditory Comprehension subtest, and compared with adult forms of aphasia (Naeser, Mazurski, Goodglass, & Peraino, 1987). Other tests of auditory comprehension not specifically designed for the assessment of aphasia but potentially useful are the Assessment of Children's Language Comprehension (Foster, Giddon, & Stark, 1973) and Carrow's (1972) Test for Auditory Comprehension of Language (Tallal, Stark, & Mellits, 1985). A frequently used test of receptive vocabulary, the PPVT-III, described earlier in this chapter, is appropriate for children.

Comprehensive examinations designed for children include the already mentioned ITPA (which has been used for aphasia assessment in some studies, e.g., Paul & Cohen, 1984); the Reynell Developmental Language Scale, designed for children from 1 to 6 years (Reynell & Gruber, 1990; Reynell & Huntley, 1971); the Northwestern Syntax Screening Test (Arndt, 1977; L. L. Lee, 1970); and the Utah Test of Language Development (Mecham, Jex, & Jones, 1967). No specific studies of children with acquired aphasia are available for these tests.

Adaptations of comprehensive examinations for aphasia for use with children have been presented for the NCCEA, the MAE (Schum et al., 1989), and the children's revision of the AST (Tramontana & Boyd, 1986). The NCCEA adaptation (Gaddes & Crockett, 1975) merely provides norms for children between ages 6 and 13 for each of the NCCEA subtests, but has not been used in research studies with aphasic children. The presented norms show an acceptable gradual increase with age for some subtests, whereas other subtests show a rapid increase within a limited age span, after which the test scores remain at ceiling level.

The Porch Index of Communicative Ability in Children (PICAC; Porch, 1981) contains a "basic battery" for 3- to 6-year-olds, and an "advanced battery" for 6- to 12-year-olds. With the exception of some floor effects, score progression with age is satisfactory. Reliability data are provided, but so far no validity studies with aphasic children have been reported. As with the PICA, the multidimensional scoring system poses problems and requires extensive training.

The Clinical Evaluation of Language Fundamentals (CELF-3; Semel, Wiig, & Secord, 1995) provides a comprehensive assessment of language development in school-age children. It consists of three receptive and three expressive language tests which differ by age group (6 to 8 years, 11 months; 9 to 21 years, 11 months). In addition, the test includes two supplementary tests. Approximately 30 to 45 min are required for the complete

test. The *Technical Manual* provides full details of the revision of the test, as well as information regarding reliability and validity. The test is designed primarily for use by the school psychologist and child speech therapist. A preschool version (Wiig, Secord, & Semel, 1992) is also available.

The CELF-3 has been well standardized with 2450 children representative of the United States in terms of region, age, sex, and ethnicity. Rules on the interpretation of dialectical variants (including "Black English") for Word Formation (morphology) and Sentence Structure (syntax) are provided in the manual. The authors stress the need for local norms to be developed by the test user, as they can differ considerably from the norms presented in the manual. One particular strength of the manual is the provision of sources for additional testing and instructional resources throughout the *Examiner's Manual* for each subtest. The constant reminders of confidence intervals for each test and for differences between tests on the summary page are also a welcome addition.

No studies of specific aphasiological interest have been presented so far. However, the CELF-3 provides downward extensions or modifications of at least three tests which have been extensively used with aphasic populations: Word Associations (Category Word Fluency), Recalling Sentences (Sentence Repetition), and Concepts and Oral Directions (TT). When testing children, the examiner may wish to use these as welcome substitutes with a good normative database. The full-length CELF-3 is suitable for the exploration of both developmental and acquired language deficits in children. Although it may not satisfy everybody's model of language functions, CELF-3 covers the major areas of syntax and semantics in both the receptive and expressive mode. It does not cover phonological (articulatory) problems.

Assessment of Aphasia in Clinical Practice

This final section presents some general considerations regarding the assessment of aphasia in clinical practice. In particular, we discuss the decision-making process before, during, and after the clinical assessment for questions of diagnosis, treatment planning, and prediction of recovery. Such decisions cannot be made by an assessment procedure, no matter how well constructed or "comprehensive" it may be, but remain the responsibility of the clinician in cooperation with related professionals involved with the individual patient.

Decisions about the Presence or Absence of Aphasia

In clinical practice, some patients are referred with an obvious presentation of aphasia. In a fair number of patients with mild or questionable

language disorder, however, a decision that rules out aphasia should be made before proceeding to other questions. On the surface, it would seem that well-validated tests of a more comprehensive nature, or even of a screening type, would be sufficient to determine whether aphasia is present. It should be remembered, however, that no test has a discrimination accuracy of 100%, and that they gray area of false-positive and false-negative decisions encountered with any given test lies necessarily in the borderline area between mild (or residual) aphasic features and normal language. Relying solely on cutoff points provided by test authors in patients with borderline impairment would, in effect, not be much better than random guessing.

The presence of disordered language, however, need not indicate the presence of aphasia. Significant nonaphasic language changes or deficits can be observed in other syndromes, such as an emerging dementia or acute confusion, and even in psychosis. Because the traditional aphasia subtypes are best seen when the etiology is a nonhemorrhagic cerebrovascular accident, language deficits may display different features when there are different etiologies, such as diffuse and severe traumatic brain injury.

The clinician must use informed judgment to arrive at her or his own diagnosis that significant language changes are present, and that they actually represent an aphasic disorder. Language disorders are frequently seen in dementing diseases and may be described by many linguistic parameters, although not necessarily as aphasic disorders (e.g., Bayles, 1984; Bayles, Boone, Tomoeda, Slauson, & Kaszniak, 1989; Bayles & Tomoeda, 1983; Fromm & Holland, 1989). Nondefective language changes may be observed in the population of normal, healthy elderly (e.g., Obler, Nicholas, Albert, & Woodward, 1985). Language and communication deficits are also common after traumatic head injury, again in the absence of frank aphasia (e.g., Hagen, 1981; Levin et al., 1976; Marquardt, Stoll, & Sussman, 1988; M. T. Sarno, Buonaguro, & Levita, 1986).

Premorbid Language Function and Intelligence

One major consideration in making an informal diagnosis is the determination or estimation of a given patient's language ability and intelligence before the onset of illness. Because test results before the onset of illness are rarely available, a careful evaluation of the patient's educational history, occupational background, language, and reading and writing habits must be made. Relatives may be consulted with regard to this information, and their judgment may be invited as to whether any language impairment is noticeable to them. The judicious clinician often can arrive at a reasonable estimate of the premorbid level of intellectual functioning by obtaining demographic information, such as years of education, age,

sex, and race, and using it in specifically developed formulas (Barona, Reynolds, & Chastain, 1984; Krull, Scott, & Scherer, 1995). Such estimates of premorbid functioning are, however, of limited value in the presence of premorbid illiteracy (Lecours, Mehler, Parente, & Caldeira, 1988).

A related, but more difficult consideration concerns the sociocultural habits of the home and job environment of the patient. The need for verbal expression varies greatly from one setting to another, and ethnic influences tend to affect such factors as verbal fluency, general fund of information, vocabulary, articulation (and intelligibility), and prosody.

Bilingualism

Patients whose first language is not English pose a special problem in the assessment of aphasia. For such patients (e.g., Hispanic/Latino-Americans, French-Canadians), the judgment of premorbid English language ability becomes difficult. Moreover, the matter of a differential impairment in the two languages requires investigation. Various theories have proposed that the "older," the "more affectively favored," or the "most frequently used" language is less affected by aphasia, whereas other studies point out either that little difference actually exists between languages (Albert & Obler, 1978), or that the language environment during recovery from brain damage is the crucial factor. It is sensible to refrain from such generalizations and establish premorbid language competence and assess impairment for both languages.

Frequently, the examination in the second language is carried out by using the same assessment methods with or without the use of an interpreter. Although this provides seemingly close comparability of the assessment in the two languages, such comparability may be tenuous at best. Often, an "instant" translation of this type only poorly approximates the difficulty level of vocabulary and grammar because of basic differences in the frequency of word use and grammatical structure in the two languages. Language tests such as COWA and even nonverbal tests are frequently affected (Jacobs et al., 1997). The MAE, described earlier, addresses these problems and attempts to provide fully equivalent forms in several languages. A bilingual test, however, can be used to best effect only when the examiner is fluent in the two languages. More broadly, any translated or interpreted verbal performance on an aphasia evaluation is subject to bias on the part of the translating resource, whether technical (i.e., quality of translation) or interpersonal (i.e., a family member who despite best intentions may "normalize" the aphasic patient's speech).

Individual tests have been deliberately constructed for the assessment of bilinguals (e.g., the Bilingual Aphasia Test; Paradis, 1987). Translations

TABLE 4.3
Tests Available in Translation or Adaptation

Test	Language
Bilingual Aphasia Test	French and other languages (Paradis, 1987)
Boston Diagnostic Aphasia Examination	Norwegian (Reinvang & Graves, 1975), Spanish (Garcia-Albea et al., 1986)
Boston Naming Test	Spanish (Taussig et al., 1988)
Communication Abilities in Daily Living	Italian, Japanese (Pizzamiglio et al., 1984; Sasanuma, 1991; Watamori et al., 1987)
Controlled Oral Word Association	Spanish (Taussig et al., 1988)
Mutlilingual Aphasia Examination	Chinese, French, German, Italian, Portugese, Spanish (Rey & Benton, 1991)
Token Test	Italian, German, Portugese
Western Aphasia Battery	Portugese

or adaptations of several other tests are available (Table 4.3), but many are still at an experimental stage or without adequate psychometric studies. Unless adequate adaptations are available, it is far more preferable to use tests developed in foreign countries. Two examples of well-developed foreign language tests are the Aachen Aphasia Battery (in German or Italian; De Bleser, Denes, Luzatti, & Mazzucchi, 1986; Willmes & Ratajczak, 1987) and the Standard Language Test of Aphasia (in Japanese; Kusunoki, 1985).

Motivational, Affective, and Attentional Considerations

Language is not an isolated cognitive function. Patients who still show the acute aftereffects of a cerebrovascular accident are frequently apathetic, drowsy, and uncooperative. Patients may also show considerable emotional reaction to their neurological impairments. Depressed mood, for example, is frequently observed during the phase of neurological stabilization, when the patient begins to realize the full extent of her or his disabilities. Other patients frankly deny their deficits or are unwilling to submit to testing procedures. These emotional reactions are not limited to patients with aphasia or with left-hemisphere lesions (e.g., Gass & Russell, 1986). Patients with accompanying acute confusion, another frequent manifestation after the onset of neurological disease, may be too disoriented or agitated to yield valid performances (e.g., Lipowski, 1980). Confused patients typically show impaired levels of verbal comprehension, regardless of whether or not they are aphasic. For these reasons, it is quite common that willingness or ability to communicate is drastically reduced, and test per-

formances are defective due to lack of motivation, attentional defects, or changes in consciousness. Interpretation of test findings will call on the judgment of the clinician rather than on blind reliance on test results.

The Nature of the Speech and Language Deficit

After a diagnosis of aphasia has been made, the description of the exact nature of the deficit becomes of paramount importance. Does the presentation conform to a known clinico-anatomic subtype? What exactly is it that the patient cannot do? What degree of impairment is present in each of the areas under examination? How much will the impairment interfere with day-to-day communication? A description of areas of strength is as important as the description of areas of deficit, because the approach to treatment relies on both types of information.

Information about the nature of the deficit continually influences the process of assessment. As the clinician finds out about specific areas of weakness, a more detailed description of that area and related deficits will be required. Special testing procedures may be added to gather this information. Occasionally, it is necessary to continue the examination in this manner after the initial assessment results have been obtained.

Diagnostic subtyping of aphasia has led many test authors to develop a test pattern for each type, either empirically or descriptively. As was pointed out earlier, the range of types of aphasia described varies from test to test, depending on the theoretical orientation of the authors. It is perhaps obvious from the preceding text that fitting a patient into a particular type on the basis of test results is of only preliminary value. Types of aphasia have been related to location of lesion as well as to rate and stage of recovery, but, as with the borderline between aphasic and normal language functioning, the gray area between types presents serious problems. Perfect fit of individual patients into such types is rare, and general or mixed impairment defying any typology is the norm. However, even if a subtype diagnosis is elusive or untenable, the attempt may still benefit the patient and family, as Benton (1994) suggests, by focusing professional decision making and by sometimes identifying unsuspected or nascant syndromes. For all of these reasons, the description of the nature of the language deficit must proceed beyond a typology and produce an individual profile for each patient.

Comprehensive Assessment

The need to refer to results from general intelligence tests has already been mentioned, as have affective considerations. Similarly, the results of other cognitive, sensory, perceptual, attentional, and motor tests are nec-

essary to appreciate fully the consequences of the underlying neurological disease process that generated the patient's aphasia. Certain language functions are likely to show severe deficits if the patient experiences distortions of visual perception, hearing, or basic ability to maintain attention. Patients are not likely to produce valid responses on tactile naming, for example, if impairment of motor functions or stereognosis is present. Comprehension may be impaired by attentional losses as well as by language deficits. Some authors of aphasia tests have built in supplementary tests for such functions that are automatically administered if the patient fails on specific language tasks; in other tests, the clinician must ascertain the basic abilities of the patient without such guidance. In multidisciplinary settings, clinical neuropsychologists provide comprehensive evaluations of cognitive status (Lezak, 1995; Spreen & Strauss, 1998) which may be interpreted in conjunction with results from aphasia tests. Regardless of the route, the description of the aphasic deficit will be clearly modulated if an examination is not restricted to features of language performance, and if ancillary and additional deficits are formally considered (e.g., Benton, 1982). The neuropsychological evaluation can also be used to explore the presence of acquired deficits in new learning and memorization, and to appreciate the residual learning capacity of aphasic patients, features of considerable importance in treatment planning and community reentry.

Recovery and Treatment of Aphasic Disorders

Another chapter in this volume is devoted to a discussion of the treatment of aphasia. However, because many patients are referred for evaluation mainly to explore treatment options, the issue of treatment should be considered briefly in relation to assessment. Increasing numbers of research studies address the question of recovery from aphasia and the most effective ways to evaluate recovery (e.g., Basso, 1992; Kertesz, 1981; M. T. Sarno & Levita, 1981; Shewan & Kertesz, 1984). One goal of clinical research has been to discern assessment findings with predictive (prognostic) value or with value relative to the recovery process (e.g., Naeser, Helm-Estabrooks, Haas, Auerbach, & Srinivasan, 1987; Varney, 1984b).

Obviously, if exploring treatment options is the purpose of the assessment, the choice of instruments will differ from the choice made for diagnostic purposes. However, many of the features described in earlier parts of this chapter still need to be considered (e.g., the presence of concurrent neuropsychological or sensory deficits, motivational status) for their impact on the recovery process and for identification of treatment modalities. Situational circumstances, family support, and other factors also may have to be assessed. Most important in this context is an assessment of the patient's relearning capacity. Existing aphasia tests do not provide an ade-

quate opportunity to judge this capacity. The clinician may resort to self-made verbal tasks carefully calibrated in difficulty to the residual capacity of the patient. For this reason, therapists may prefer to "lift" whole sections of an existing test in the appropriate area of deficit and amplify such tests with additional material of their own choosing in order to establish a baseline of performance at the beginning of therapy. For example, learning can be assessed with a brief series of items (e.g., word-finding to pictures or objects) that is repeated until all items are fully learned; the measure in this case would be the number of trials needed to reach criterion (e.g., complete naming or description of use for five items). A repetition of the same procedure on the following day will indicate the patient's "gain" or "carry-over," that is, how many fewer trials are needed for relearning the same items. However, a wide variety of standardized new-learning and memorization tests that minimize verbal demands are available (Lezak, 1995) to assess residual verbal and nonverbal learning skills and capacity.

The methodology for the development of progress evaluation or criterion-based techniques during therapy has been well established by authors in the behavior modification field (e.g., Lahey, 1973). The "test–teach–test" approach in both education and speech therapy has the advantage of being directly relevant to the material being taught or to the language problem under training; no inferences from a general sampling of language behavior are necessary. Repeated examination after specified periods of training will then allow a plotting of any change over time and, if a criterion is specified, a determination of whether significant progress has been attained.

Conclusion

Choice of Tests

No formal battery of tests can or should be recommended as sufficiently comprehensive to arrive at an optimal description of the nature of the speech and language deficit for an individual patient. In clinical practice, we, as well as many other clinicians, tend to use a flexible approach for which a comprehensive test battery is only the beginning. Complete reliance on a given test battery tends to introduce an element of rigidity that may result in failure to explore fully the patient's problem.

The choice of instrument will depend on the purposes of the assessment, as well as on individual preference and theoretical orientation. The test chosen should be supplemented with other test procedures: specific-purpose tests (or parts of another comprehensive battery), a functional com-

munication assessment, a clinical examination of specific problems, and, if possible, specially constructed tasks suitable for retraining.

The approach advocated here requires full knowledge of all available instruments as well as clinical skills and judgment. Although parts of the examination are likely to be conducted in many settings by a trained psychometrist, the full involvement of the experienced clinician is necessary. Even though computerized test administration and scoring are becoming available for many tests and can be time-saving, computer programs for test *interpretation* should be used with considerable caution. The need for the clinician to review the test scores and interpret the test protocol remains.

Other considerations in the choice of assessment methods are (a) psychometric adequacy of a test, (b) portability of the test material, and (c) time requirements. The more a test meets the ideals of a psychometrically well-developed test, the more likely it is that valid and reliable results are obtained. Portability tends to be of no major concern in a hospital-based clinic or evaluation service, but it does become a problem if bedside examinations are frequently carried out. In this latter case, one would prefer a handy portfolio of pictures rather than a suitcase full of objects, even though any pictured item tends to lose some value on a "reality" dimension. Time is a crucial consideration in many facilities with heavy patient loads; however, time requirements should be carefully weighed against the information that might be gleaned from a given test. Brevity is no virtue if crucial information is not collected. In fact, the approach advocated here suggests that time requirements should be of secondary importance, and that experimental variants and additional exploratory procedures that may be of benefit in the long run should be used in the course of the assessment. If, on the other hand, brief screening is the only goal of assessment, then many of the short tests or screening devices deserve consideration.

Assessment is not an end in itself, but must be considered in relation to its potential value to the patient and to the treatment and management of the patient's deficits. As Messick (1980) pointed out, the adequacy of a test is not dictated solely by psychometric soundness. Rather, the concept of construct validity should include the "ethics" of assessment; that is, it must provide a rational foundation for prediction and relevance as well as take into account the implications of test interpretation per se.

Interpretation of Assessment Results

Every clinician has his or her own model of how best to survey a summary sheet of assessment results, with frequent glances at the actual test records and notes on the behavior of the patient during testing. Many of

the comprehensive tests provide, of course, their own grouping of the test information and hence a suggested approach to interpretation (e.g., summary scores for dimensions such as auditory comprehension and verbal expression). Other test authors leave the interpretation open to the clinician using the test. Our own approach (and that of many other clinicians) tends to be "syndromatic" in the sense that we tend to focus first on the most seriously defective scores in the assessment record and then scan the record for related information and corroborative test findings. For example, if the patient's most serious problem is on a test of word finding, we scan all related test results, as well as information about the patient's ability to find words in conversational settings, for higher order performance on verbal and nonverbal memorization/new-learning tests and so forth. This allows a better description of the deficit, that is, whether the deficit is generalized or specific to the test setting, whether it is related to a specific sensory modality, whether it is a secondary manifestation of a nonaphasic amnestic or attentional disorder, and so forth. Additional assessment procedures may well be necessary to evaluate fully this first "syndrome."

We then proceed to the next syndrome that appears to be reasonably independent of the first, and again search for associated task failures and other corroborative evidence. In this manner, we can move toward the least deviant score on the assessment record, keeping in mind the estimated premorbid intelligence of the patient. Such syndromes may or may not be related to each other; they may or may not reflect a "classical type" of aphasia with localizing significance. Our primary purpose is to gain a detailed picture of the patient's deficits in order of severity and in the context of other deficits. We then proceed in the opposite direction, searching for the best score in the test record or the best preserved function until the information in the assessment record is exhausted.

Finally, we reexplore the noted syndromes by evaluating the actual behavior of the patient on individual tests or other assessment procedures. This step results in a fuller description of the patient's performance. Interpretation of findings in the broader context of the patient's level of adjustment to his or her current deficits, the patient's awareness of the deficits, family cohesiveness and ability to provide support, and appreciation for individualized community reentry needs are all likely to influence the clinician's understanding of the patient. For instance, we would no longer describe "anomia for visually presented real objects," but now include details of whether this deficit is part of a fuller diagnostic syndrome, what the associated impairments are, how the deficit affects the patient and his or her family, and how treatment might approach the deficit by building on strengths and working on weaknesses.

The approach described here is highly idiosyncratic in a deliberate at-

tempt to avoid preconceived models of language and brain functions. However, until a more generally accepted model of language disorders and standards of procedure for standard questions are developed—and little progress has yet been made in that direction—this outline of objective procedures for interpretation may provide the fullest utilization of assessment results at the present state of knowledge.

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5

Phonological Aspects of Aphasia

SHEILA E. BLUMSTEIN

Introduction

Phonology is the study of the sound structure of language. In both speaking and understanding, it provides the medium by which meaning is conveyed. This chapter explores the nature of the deficits in the sound structure of language that are found among the adult aphasias. Although the focus of this chapter is on the sound structure of language, it is worth emphasizing that studying phonology in aphasia does not imply that other linguistic abilities are necessarily normal. In fact, selective deficits affecting only one aspect of language processing (speaking or understanding) and one component of the linguistic grammar (phonology, syntax, or lexicon) are extremely rare. Moreover, as will be noted in the course of this chapter, the processes involved in mapping between meaning and sound are inextricably linked and interdependent.

It is the goal of this chapter to characterize the nature of the phonological deficits in aphasia. To this end, we consider a number of general questions:

- Do phonological deficits reflect impairments of representation or the processes involved in access to and implementation of sound structure.
- Do phonological deficits reflect impairments at a linguistic level or do they reflect impairments that are more properly characterized as low-level phonetic, that is, articulatory in speech production or auditory in speech perception.
- To what extent do phonological deficits in aphasia respect the classical dichotomy between left anterior brain structures, as largely in-

volved in language/speech production, and posterior brain structures, as largely involved in language/speech comprehension.

- To what extent are the speech production and speech perception impairments similar or different among the clinical types of aphasia, and what do these results suggest for the nature of the underlying neural mechanisms subserving the sound structure of language.

We first discuss speech production deficits in aphasia and examine the dichotomy between phonological and phonetic deficits which seem to underlie retrieval of lexical representations and planning processes, on the one hand, and articulatory implementation, on the other. We then turn to speech perception and explore how speech perception deficits may relate to both auditory processing deficits and auditory comprehension deficits. We also consider the processes involved in the mapping from sound structure to the lexicon. As a preliminary step, it is useful to provide a working framework for the study of the sound structure of language and to review the classical approaches to the clinical–neurological bases of language disorders.

The Sound Structure of Language: A Theoretical Framework

The sound structure of language is shaped not only by physiological constraints of the speech apparatus (the vocal tract) in speech production and the auditory system (the auditory pathway) in speech perception, but also by constraints and principles that are unique to language itself. Every language has its own inventory of sounds and its own rules of how these sounds can combine to form words. Sound units called sound segments are typically analyzed in terms of two levels of representation: PHONOLOGICAL and PHONETIC. The phonological level defines the way in which the sound properties of language may be defined as well as their organizational principles. One fundamental unit is the PHONEME, which is defined as the minimal sound unit of language that contrasts meaning; for example, in English the sounds /p/ and /b/ differentiate words such as *pear* and *bear.* Every language has an inventory of phonemes, and in addition, has its own rules of combination of those phonemes. For example, *brick* is a word in English; *blick* is a possible but nonexistent word in English; and *bnick* is an unacceptable potential word in English.

Although phonemes are considered the minimal "meaningful" sound units of language (considered meaningful because they distinguish among potential words of the language), they are further divisible into smaller

components called PHONETIC FEATURES. Phonetic features characterize phonemes in terms of either articulatory or acoustic characteristics that make up the identity of the phoneme. For example, the phoneme /p/ is [+ consonantal] (it is produced with an obstruction in the vocal tract); [+ stop] (it is produced with a complete closure in the vocal tract followed by an abrupt release); [+ bilabial] (the closure occurs at the lips); [− voice] (the vocal cords do not begin to vibrate until after the release of the stop closure). The phoneme /b/ shares the same phonetic features as /p/ except for the voicing feature. For /b/, the vocal cords begin to vibrate either prior to or close in time to the release of the stop closure, and thus /b/ is [+ voice].

The phonological level of representation also characterizes the stress and intonation patterns of language. Individual words have different stress patterns, and combinations of words may have differing intonation patterns. Together, stress and intonation comprise SPEECH PROSODY and are defined as suprasegmentals because they overlay the domain of the individual sound segments. Stress corresponds to the patterns of accentuation of words, for example, *prósody* not *prosódy,* and intonation refers to the pitch or melodic pattern of a sentence, distinguishing in English, for example, the two sentences "he is here!" versus "he is here?"

Ultimately, the phonological level must be realized in physical reality. The phonetic level provides the detailed physical characteristics of the phonological representation of language, specified either in terms of their articulatory parameters for speech production or in terms of their acoustic properties for speech perception. For example, the phoneme /p/ is realized differently phonetically as a function of the environment in which it occurs—in initial position, it is produced with aspiration as in the word *pill;* whereas after /s/, it is produced without aspiration, as in the word *spill.*

The phonological and phonetic levels of representation define the linguistic principles that are used to characterize the sound structure of language. These levels and principles are also incorporated into models of language production and language comprehension. Figure 5.1 shows such a working model, which includes not only the levels of representation but also the "processes" contributing to both speech production and speech perception. As the figure shows, a single lexicon (words of the language) is shared by both speech production and speech reception mechanisms. That is, words to be produced or perceived ultimately contact a common representation. The nature of that representation is in terms of segments, phonetic features, and rules for their combination. In addition, all auditory speech input ultimately accesses the lexicon. Thus, as depicted, there is no separate mechanism for the processing of nonsense syllables indepen-

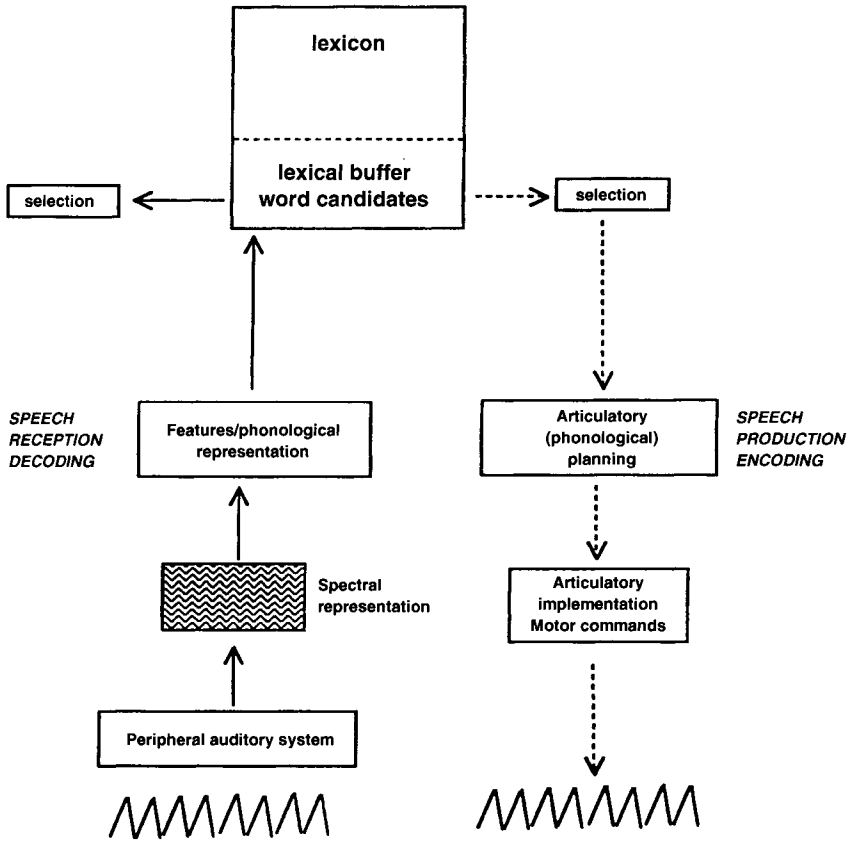

FIGURE 5.1. *A working model of speech production and speech perception. From Blumstein (1994).*

dent of the lexicon. Nonwords presumably access the lexicon, but because they do not map onto any lexical entry, they are rejected as words. A similar assumption is made in speech production. As will be discussed, processing real words and nonsense syllables respect similar patterns of performance, although usually the level of performance is worse with nonsense syllables than with real words.

Nonetheless, language/speech production and language/speech reception ultimately use different mechanisms for implementing and processing the physical properties of speech. A lexical representation ultimately must be transformed into a set of articulatory commands in the processes involved in language production. In contrast, an acoustic input ultimately must be mapped onto a lexical representation in the processes

involved in language comprehension. Thus, the interface of the production and perception mechanisms with the lexicon requires a different set of operations.

It is a matter of considerable debate as to the nature of the cognitive architecture that underlies the speech/lexical processing system. Without entering that debate, we are assuming that the internal structure of the representation of a word or a feature is coded in terms of patterns of neural activity of units, and that both lexical representations and their underlying sound structure are themselves part of a network that is "connected" by means of patterns of excitation and inhibition between and among these units. Thus, the identity of a sound or a word is distributed in the pattern of activation of a number of units. This assumption about the cognitive architecture of speech and language makes some very important predictions about the effects of brain damage on speech and language processing. Most particularly, if the representation of a word or a feature is coded in terms of patterns of neural activity, then it is unlikely that a strict localizationist approach will ultimately be able to characterize patterns of impairment in the sound structure of language. In addition, brain damage will not likely have an all-or-none effect on the processing of speech/language, as only part of a network (either units, their connections, and/or connection strengths) will likely be affected (cf. McClelland & Rumelhart, 1986; Wood, 1982). As will be shown, results from studies of aphasia are consistent with these assumptions.

Speech Production

As Figure 5.1 shows, the production of a word or words involves the SELECTION of a word candidate or candidates from the lexicon, the ENCODING of the abstract phonological representation of the word in terms of the proper order of the segments and in terms of the phonological context in which they appear (articulatory planning), and then the IMPLEMENTATION of this phonetic string into a set of motor commands or motor programs to the vocal tract. A number of models of speech production propose that these word candidates are scanned into a short-term buffer to account for the fact that the ultimate production of a sequence of words or an utterance is influenced not only at the segmental level but also at the prosodic level by the phonological context of neighboring words, and, ultimately, the syntactic role that the individual lexical item plays in the utterance string (cf. Levelt, 1989, for discussion). For example, the auxiliary *have* may be reduced in certain syntactic contexts and appended to the preceding word, for example, "I have eaten" may be produced as "I've eaten."

The study of speech production deficits in aphasia have typically distinguished between analyses of the phonological patterns of production, that is, the selection of a lexical item and the planning of its production, and the phonetic patterns of speech production, that is, its articulatory implementation. Thus, for example, if a patient produces a wrong sound segment, but its phonetic (articulatory) implementation is correct, that is, for *cat* the patient says *gat*, then it is inferred that the basis of the patient's output error is phonological and is related to either the selection or planning stages of speech output. In contrast, if a patient produces the correct sound segment but its phonetic implementation is incorrect, that is, for *cat* the patient produces an initial /k/ that is overly aspirated [k^{hh}], then it is inferred that the basis of the patient's disorder is phonetic and is related to the articulatory implementation stages of speech output.

Recently, phonological output disorders have been looked at within a broader framework, not just focused on sound structure per se, but on how phonological form may be instantiated in the processes of word (lexical) retrieval. The essential features of the working model proposed in Figure 5.1 are implemented within an interactive spreading activation framework (cf. Dell, 1989; Schwartz, Saffran, Bloch, & Dell, 1994). It is beyond the scope of this chapter to present the details of these models. Nonetheless, the working model in Figure 5.1 is compatible with these models, and, more importantly, the overall conclusions drawn from the results of phonological disorders presented here are similar.

Phonological Patterns of Speech Production

Clinical evidence shows that nearly all aphasic patients produce phonological errors in their speech output. These errors can be characterized according to four main types:

1. Phoneme substitution errors, in which a phoneme is substituted for a different phoneme in the language, for example, *teams* → /kimz/.
2. Simplification errors, in which a phoneme or syllable is deleted, for example, *brown* → /bawn/.
3. Addition errors, in which an extra phoneme or syllable is added to a word, for example, *papa* → [paprə].
4. Environment errors, in which the occurrence of a particular phoneme is influenced by the surrounding phonetic context. The order of the segments may be changed, for example, *degree* → [gədri], or the presence of one sound may influence the occurrence of another, for example, *Crete* → [trit].

Within each of the four categories of errors, there are systematic patterns that have been observed among the aphasic patients studied and provide

clues as to the basis of the deficit. The majority of phoneme substitution errors are characterized by the replacement of a single phonetic feature. For example, patients may make errors involving the phonetic feature [voice], for example, *peace* → [bis], the phonetic feature [place of articulation], for example, *pay* → [tei], or manner of articulation such as [nasal], for example, *day* → [nei]. Rarely do they make errors involving more than one phonetic feature. Moreover, there is a hierarchy of phoneme substitution errors, with a greater preponderance of errors involving place of articulation, then voicing, and fewest, manner of articulation. The overall pattern of sound substitutions is consistent with the view that the incorrect phonetic features have been selected or activated, but they have been correctly implemented by the articulatory system. Most simplification errors and addition errors result in what is believed to be the simplest and thus the canonical syllable structure of language, Consonant Vowel. For example, consonants are more likely deleted in a word beginning with two consonants, *sky* → *ky*, and are more likely added in a word beginning with a vowel, *army* → *jarmy* (Blumstein, 1990). And finally, environment errors which occur across word boundaries preserve the syllable structure relations of the lexical candidates. That is, if the influencing phoneme is at the beginning of the target word, so is the influenced phoneme, for example, *history books* → *bistory books*. If the influencing phoneme is at the end of the target word, so is the influenced phoneme: *roast beef* → *roaf beef*.

The stability of these patterns is evidenced by their occurrence across languages: French (Bouman & Grunbaum, 1925; Lecours & Lhermitte, 1969), German (Bouman & Grunbaum, 1925; Goldstein, 1948), English (Blumstein, 1973; Green, 1969), Turkish (Peuser & Fittschen, 1977), Russian (Luria, 1966), and Finnish (Niemi, Koivuselka-Sallinen, & Hanninen, 1985). Despite the systematicity and regularity of these phonological errors, their particular occurrence cannot be predicted. That is, sometimes the patient may make an error on a particular word, and other times she or he will produce it correctly. Moreover, the pattern of errors are bidirectional (Blumstein, 1973; Hatfield & Walton, 1975). A voiced stop consonant may become voiceless, /d/ → /t/, and a voiceless stop consonant may become voiced, /t/ → /d/.

Taken together, these results suggest that the patient has not "lost" the ability to produce particular phonemes or to instantiate particular features. Rather, his or her speech output mechanism does not seem to be able to encode consistently the correct phonemic (i.e., phonetic feature) representation of the word. As a consequence, the patient may produce an utterance that is articulatorily correct but deviates phonologically from the target word. On other occasions, the patient may produce the same target word correctly. These results are consistent with the view that the underlying phonological representations are intact, but there are deficits in accessing

these representations (Butterworth, 1992). As such, these patients have a selection or phonological planning deficit (Blumstein, 1973, 1994; cf. also Nespoulous & Villiard, 1990). To return to the model for speech production in Figure 5.1, a word candidate is selected from the lexicon. To produce the word requires that its sound properties (i.e., its segments and features) be specified so that they can be "planned" for articulation and ultimately translated into neuromuscular commands relating to the speech apparatus. Phonological deficits seem then to relate to changes in the activation patterns of the nodes corresponding to the phonetic representations themselves (e.g., features, syllable structure) as the word candidate is selected, as well as to deficits in the processes involved in storage in the short-term lexical buffer and in phonological planning (cf. also Schwartz et al., 1994; Waters & Caplan, 1995).

The similar patterns of performance are particularly striking given the very different clinical characteristics and neuropathology of the patients investigated. The groups studied have included both anterior and posterior patients. Anterior aphasics, especially Broca's aphasics, show a profound expressive deficit in the face of relatively preserved auditory language comprehension. Speech output is nonfluent in that it is slow, labored, and often dysarthric, and the melody pattern is often flat. Furthermore, speech output is often AGRAMMATIC. This agrammatism is characterized by the omission of grammatical words, such as *the* and *is*, as well as the substitution of grammatical inflectional endings marking number, tense, and so forth.

In contrast to the nonfluent speech output of the anterior aphasic, the posterior patient's speech output is fluent. Among the posterior aphasias, Wernicke's and conduction aphasia are perhaps the most studied in relation to phonology (cf. Ardila, 1992; Buckingham & Kertesz, 1976; Kohn, 1992; Schwartz et al., 1994). The characteristic features of the language abilities of Wernicke's aphasia include well articulated but paraphasic speech in the context of severe auditory language comprehension deficits. Paraphasias include LITERAL PARAPHASIAS (sound substitutions), VERBAL PARAPHASIAS (word substitutions), or NEOLOGISMS (productions that are phonologically possible but have no meaning associated with them). Speech output, although grammatically full, is often empty of semantic content and is marked by the overuse of high-frequency "contentless" nouns and verbs, such as *thing* and *be*. Another frequent characteristic of this disorder is LOGORRHEA, or a press for speech.

Conduction aphasia refers to the syndrome in which there is a disproportionately severe repetition deficit in relation to the relative fluency and ease of spontaneous speech production and to the generally good auditory language comprehension of the patient. Speech output contains many literal paraphasias and some verbal paraphasias.

The results of the studies of the phonological patterns of speech production challenge the classical view of the clinical/neurological basis of language disorders in adult aphasics. The classical view has typically characterized the aphasia syndromes in broad anatomical (anterior and posterior) and functional (expressive and receptive) dichotomies (cf. Geschwind, 1965). To a first approximation, the anterior/posterior anatomical dichotomy corresponds well with the functional expressive/receptive dichotomy as anterior patients are typically nonfluent and posterior patients are typically fluent, and anterior patients typically have good comprehension and posterior patients typically have poor comprehension. Nonetheless, the similar patterns of performance across these aphasic syndromes indicates that both anterior and posterior brain structures contribute to the selection of phonological representations as well as to phonological planning in speech production.

An interesting syndrome from the perspective of phonological output disorders is jargon aphasia (those Wernicke's aphasics who produce NEOLOGISMS or JARGON, which are defined as the production of nonwords that do not derive from any obvious literal paraphasia or phonologically distorted semantic paraphasia). Phonological analyses reveal that neologisms follow the phonological patterns of the language. They respect the sound structure, stress rules, syllable structure, and phonotactics (allowable order of sounds). Although it is not clear what the source of these jargon productions are, their phonological characteristics are consistent with the general observation that the processes of lexical activation and retrieval are the source of the problem, not the more abstract phonological shape or organizational principles of the lexicon (Christman, 1994; Hanlon & Edmondson, 1996; Kohn, Melvold, & Smith, 1995).

Phonetic Patterns of Speech Production

As Figure 5.1 shows, subsequent to the selection of a lexical candidate or candidates and the articulatory planning of the utterance, the phonetic string is ultimately converted into a set of motor commands to the articulatory system. There is a wide range of speech production deficits that reflect impairments to the motor commands or motor programs to the vocal tract system. For the purpose of this chapter, we limit discussion to those phonetic disorders occurring in the context of a language impairment, that is, aphasia. Thus, we will not consider the dysarthrias, which are speech disorders resulting from damage to the speech musculature itself or to the neural mechanisms that regulate speech movements, or those speech production deficits that involve the descending motor pathways, including subcortical structures, various levels of the brain stem, the extrapyramidal system, the cranial nerves, and so on.

Whereas the processes involved in the selection and planning of the sound structure of language seem to be broadly represented in the left dominant language hemisphere, the neural basis of phonetic disorders seems to be more localized. And yet, as will be discussed, the language production apparatus also seems to be a highly interconnected system, involving the contribution not only of anterior speech motor areas, but also potentially of posterior areas as well.

A long-held observation is that anterior aphasics produce phonetic errors. The implied basis for these errors is one of articulatory implementation; that is, the commands to the articulators to encode the word are poorly timed and impaired. A number of studies have explored these phonetic patterns of speech by investigating the acoustic properties or the articulatory parameters underlying the production of particular phonetic dimensions.

Studies of speech production in anterior patients have shown that these patients have difficulty producing phonetic dimensions that require the timing of two independent articulators. These findings have emerged in the analysis of the production of two phonetic dimensions, voicing and nasality. In the case of the feature voicing, the dimension studied has been voice-onset time, that is, the timing relation between the release of a stop consonant and the onset of vocal cord vibration. For voiceless consonants, such as /p/, there is a delay in the onset of vocal cord vibration of around 30 ms after the stop consonant is released; whereas for voiced consonants, such as /b/, vocal cord vibration begins either coincident with the release of the consonant or some tens of milliseconds later. The production of nasal consonants also requires appropriate timing between two articulators; in this case, the release of the closure in the oral cavity and the velum opening. For /m/, the velum must be opened when the closure at the lips is released; whereas for /b/, the velum must stay closed while the closure at the lips is released.

Results of analyses of the production of the voicing and nasal phonetic dimensions have shown that anterior aphasics evidence significant deficits (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980; Blumstein, Cooper, Zurif, & Caramazza, 1977; Freeman, Sands, & Harris, 1978; Gandour & Dardarananda, 1984a; Itoh, Sasanuma, Hirose, Yoshioka, & Ushijima, 1980; Itoh et al., 1982; Itoh, Sasanuma, & Ushijima, 1979; Shewan, Leeper, & Booth, 1984). These same patterns emerge across different languages. They occur not only in English and Japanese for which voice-onset time distinguishes two categories of voicing, voiced and voiceless, but also in Thai for which voice-onset time distinguishes three categories of voicing in stop consonants, pre-voiced, voiced, and voiceless aspirated (although cf. Ryalls, Provost, & Arsenault, 1995, for some different findings in French-

speaking aphasics). All of these studies have used acoustic measurements and have inferred the articulatory states giving rise to the acoustic patterns observed. More direct measures of articulatory timing with fiber optics (Itoh & Sasanuma, 1983; Itoh et al., 1979), computer-controlled X-ray microbeams (Itoh et al., 1980), and electromyography (Shankweiler, Harris, & Taylor, 1968) have also shown that the timing relations among the articulators is impaired.

That the anterior aphasics have particular difficulties with the production of two phonetic features, voice and nasal, could indicate that these patients have an impairment that is affecting the articulatory implementation of particular phonetic features (voice and nasal) or, alternatively, the implementation of particular articulatory maneuvers. It is possible to answer this question by exploring the constellation of spared and impaired patterns of articulation associated with the production of voicing in stop consonants. In English, the feature voicing in stop consonants can be cued in several ways. Voice-onset time provides one measure of voicing for stop consonants occurring in initial position. A second measure is the duration of the vowel preceding a stop consonant. Vowels are short before voiceless stops, *write,* and long before voiced stops, *ride.* If patients have a deficit related to the implementation of the feature voicing, then they should display impairments in the production of voice-onset time as well as vowel length preceding voiced and voiceless stop consonants. In contrast, if they have a deficit related to particular articulatory maneuvers, such as the timing of two independent articulators, the production of voice-onset time may be impaired, whereas the production of vowel length may be normal. Results indicate that although these patients show an impairment in the implementation of the voicing phonetic dimension, via voice-onset time, they are able to maintain the distinction between voiced and voiceless stops on the basis of the duration of the preceding vowel (Baum, Blumstein, Naeser, & Palumbo, 1990; Duffy & Gawle, 1984; Tuller, 1984). Thus, these patients do not have a disorder affecting the articulatory production of the feature voicing, but a disorder affecting particular articulatory maneuvers, namely, the timing or integration of movements of two independent articulators.

Consistent with this view are the results from the acoustic analysis of the production of vowels. Differences among vowel sounds such as /i a u/ are determined acoustically by the frequency of the first two resonant peaks, called FORMANT FREQUENCIES. Analyses of the formant frequencies of spoken vowel utterances show that anterior aphasics, including Broca's aphasics, maintain formant frequency characteristics of different vowels, despite increased variability in their productions (Kent & Rosenbek, 1983; Ryalls, 1981, 1986, 1987). The production of vowels requires articulatory

gestures based on the overall shape of the tongue, rather than on the coordination of independent articulators.

Although anterior aphasics show a disorder in temporal coordination, their disorder does not reflect a pervasive timing impairment. Fricative durations do not differ significantly from those of normals (Harmes et al., 1984), and the patients maintain the intrinsic duration differences characteristic of fricatives varying in place of articulation; for example /s/ and /š/ are longer in duration than /f/ and /θ/ (Baum, 1996; Baum et al., 1990). Although overall vowel duration is longer for anterior aphasics than for normals (Baum, 1993; see Ryalls, 1987, for review), these patients do maintain differences in the intrinsic durations of vowels; for example, tense vowels such as /i/ and /e/ are longer than their lax vowel counterparts, /I/ and /E/. In addition, Thai-speaking anterior aphasics maintain the contrast between short vowels and long vowels. In Thai, vowel length is phonemic, in that long and short vowels distinguish words in the language, for example, /hat/ *to practice* versus /haat/ *shoal* (Gandour & Dardarananda, 1984b; Gandour, Ponglorpisit, Khunadorn, Dechongkit, Boongird, & Boonklam, 1992).

In addition to impairment in timing of independent articulators, difficulties for anterior aphasics have also emerged with laryngeal control. They have shown impairments in voicing in the production of voiced fricatives (Baum, 1996; Baum et al., 1990; Harmes et al., 1984; Kent & Rosenbek, 1983), and impairments in voicing influencing the spectral shape associated with place of articulation in stop consonants (Shinn & Blumstein, 1983).

Consistent with the findings that anterior aphasics have impairments involving laryngeal control are studies of INTONATION. Intonation, or the melody of language, is ultimately determined by laryngeal maneuvers. A number of acoustic parameters are used to study intonation. Among the most common is the analysis of fundamental frequency, which relates to the frequency of vibration of the vocal cords. The study of intonation provides important clues to speech planning abilities. That is, different intonation patterns emerge as a function of syntactic complexity and sentence length. Typically, declarative sentences in English have a falling intonation at the end of the sentence, called TERMINAL FALLING f_0 (fundamental frequency), and the final word of the sentence is typically lengthened (Cooper & Sorenson, 1980). For the speaker to produce the appropriate pitch contours and word duration, it is necessary to effectively preplan the sentence, taking into consideration its length and syntactic structure.

Acoustic analyses of two-word spontaneous speech utterances and reading in Broca's aphasics have shown that although these patients have rudimentary control over some features of prosody, in that they maintain a terminal falling fundamental frequency even in utterances in which the

pauses between words may reach durations of as long as 7 sec (Cooper, Soares, Nicol, Michelow, & Goloskie, 1984), they show a restriction in the fundamental frequency range (Cooper et al., 1984; Ryalls, 1982). Restrictions in fundamental frequency range support the clinical impression that these speakers produce utterances in a monotone or with a flattened intonation. However, the fact that they maintain a falling fundamental frequency suggests that they do have a linguistic sense of an utterance and are not simply stringing together lexical items. Nonetheless, these patients do show a number of systematic problems in the production of prosody. They typically do not show utterance final lengthening, but rather show longer durations in word initial position (Danly, de Villiers, & Cooper, 1979; Danly & Shapiro, 1982). An increased threshold for initiating and maintaining the flow of speech may account for these findings.

Another dimension of prosody, tone production, is used in languages such as Thai and Chinese to distinguish among lexical items or words. Only a few acoustic analyses of the production of tone have been conducted. The results suggest that deficits in tonal production may emerge in anterior aphasics; however, it appears that the global properties of the tone, for example, whether the tone is high or falling, is maintained, suggesting that the production deficit is due to articulatory implementation rather than to phonological planning (Gandour, Holasuit, Petty, & Dardarananda, 1988; Gandour, Ponglorpisit, Khunadorn, Dechongkit, Boongird, Boonklam, & Potisuk, 1992).

Kent and Rosenbek (1983) have suggested that the timing problem found for individual segments and their underlying features is a manifestation of a broader impairment in the integration of articulatory movements from one phonetic segment to another. The sounds of speech are affected by the phonetic contexts in which they occur. For example, the production of /s/ and its consequent acoustic characteristics vary depending on whether /s/ is followed by the vowel /i/ or the vowel /u/. When /s/ is followed by the vowel /u/, it is produced with rounding (pursing of the lips) in anticipation of the rounded vowel /u/. No such adjustments are made for /i/. The rounding of the lips lengthens the vocal tract causing a lowering of the formant frequencies for /s/ before /u/ compared with /s/ before /i/. The study of such coarticulation effects provides insights into the dynamic aspects of speech production, and also provides evidence about the size of the planning units that can be programmed in the production of syllables or words.

Investigations of coarticulation effects in anterior aphasics show that they produce relatively normal anticipatory coarticulation (Katz, 1988; Katz, Machetanz, Orth, & Schonle, 1990a, 1990b; Sussman, Marquardt, Hutchinson, & MacNeilage, 1988). For example, in producing the syllable

/s/, they anticipate the rounded vowel /u/ in the production of the preceding /s/ (Katz, 1988). Nevertheless, they seem to show a delay in the time it takes to produce these effects (Ziegler & von Cramon, 1985, 1986), and they show some deficiencies in their production (Tuller & Story, 1986; but see Katz, 1987, for discussion). What these results suggest is that phonological planning is relatively intact, but it is the ultimate timing or coordination of the implementation of the articulatory movements that is impaired. Consistent with this view are results showing that Broca's patients demonstrate impairments in the complex timing relation between syllables (Gandour, Dechongkit, Ponglorpisit, & Khunadorn, 1994; Gandour, Dechongkit, Ponglorpisit, Khunadorn, & Boongird, 1993). For example, they do not show the normal decrease in the duration of a root syllable as word length increases (cf. Baum, 1992), nor do they show a normal ability to increase rate of articulation beyond a certain limit. Such an impairment abnormally affects the production of the segmental properties of speech, such as voice-onset in stop consonants, fricative duration, and vowel duration as a function of speaking rate (cf. Baum, 1993, 1996; Baum & Ryan, 1993; Kent & McNeill, 1987; McNeill, Liss, Tseng, & Kent, 1990).

Several conclusions can be made concerning the nature of the phonetic disorders and their ultimate underlying mechanisms. In particular, the impairment is not a linguistic one, in the sense that the patient is unable to implement a particular phonetic feature. Moreover, the patients have not lost the representation for implementation nor the knowledge base for how to implement sounds in context. They not only adjust their articulatory mechanism in the implementation of a segment to anticipate a neighboring segment and to produce the appropriate timing relations in a consonant–vowel sequence, but also compensate for the fixation of the jaw by a bite block (Baum, Kim, & Katz, 1997). Instead, particular maneuvers relating to the timing of articulators seem to be impaired, ultimately affecting the phonetic realization of some sound segments and of some aspects of speech prosody.

Computerized tomography (CT) scan correlations with patterns of speech production deficits suggest the involvement of Broca's area (slice B and B/W), the anterior limb of the internal capsule (including slice B, B/W, and W; Baum et al., 1990), and the insula of the precentral gyrus (Dronkers, 1997). The lower motor cortex regions for larynx, tongue, and lips (slices W and SM) are also implicated, although less consistently so. Nevertheless, phonetic disorders, as described in this chapter, do not emerge with damage to analogous speech areas in the right hemisphere, suggesting that even though both hemispheres may be ultimately involved in the production of speech, the control site for these mechanisms is in the left hemisphere (Gandour et al., 1994; Kurowski, Blumstein, & Mathison, 1998).

There is another phonetic disorder that occurs rarely and reflects a pat-

tern of deficit different from that previously described. This disorder is called the FOREIGN ACCENT SYNDROME and is characterized by the emergence of what is perceived by the listener as a foreign accent subsequent to organic brain disease (Blumstein, Alexander, Ryalls, Katz, & Dworetzky, 1987; Gurd, Bessel, Bladon, & Bamford, 1988; Ingram, McCormack, & Kennedy, 1992; Kurowski, Blumstein, & Alexander, 1996). Acoustic analyses of the patterns of speech production of these patients provide a potential explanation for why listeners report that the patient speaks as though she or he has a foreign accent. Although the deficit is primarily phonetic in nature, it particularly affects the rhythmic and prosodic patterns of language, including the production of vowels, the syllable structure of words, and the prosody of language (especially hypermelodic), phonetic characteristics which are only minimally affected in anterior aphasics. Even phonetic errors which occur on consonants, typically affect the syllable structure of the output. Thus, unlike the articulatory implementation deficits that characterize anterior aphasics, the phonetic patterns of speech of patients displaying the foreign accent syndrome preserve the patterns of prosody and rhythm that occur in natural language. It is for this reason that listeners are likely to "hear" the speech output pattern of these patients as "foreign." These results suggest that there are multiple mechanisms that result in speech output disorders. A great deal more research is required to determine whether these mechanisms are part of a single output system or whether there are a number of different mechanisms contributing to the articulatory implementation of speech.

Although it is not surprising to find that anterior portions of the left hemisphere, particularly those localized in the vicinity of the motor cortex, are implicated in the production of speech, recent results suggest that posterior areas of the brain are also involved. There is no question that phonetic patterns of speech are qualitatively distinct in anterior and posterior aphasics. Posterior aphasics do not display the timing deficits that anterior aphasics manifest in the production of voice-onset time in stop consonants (Blumstein et al., 1980; Gandour & Dardarananda, 1984a; Hoit-Dalgaard, Murry, & Kopp, 1983; Shewan et al., 1984; Tuller, 1984) or in the production of nasal consonants (Itoh & Sasanuma, 1983). Nor do they show impairments in laryngeal control either for the production of voicing or for those articulatory maneuvers requiring the integration of laryngeal movements and movements of the supralaryngeal vocal tract (Baum et al., 1990; Shinn & Blumstein, 1983). Nevertheless, although clearly distinguished from anterior aphasics, posterior patients do display a subtle phonetic impairment even in the production of single syllables or isolated words in citation form. Most typically, they show increased variability in the implementation of a number of phonetic parameters (Kent & McNeill, 1987; Ryalls, 1986), including vowel formant frequencies (Ryalls, 1986) and

vowel durations (Gandour, Ponglorpisit, Khunadorn, Dechongkit, Boongird, & Boonklam, 1992; Ryalls, 1986; Tuller, 1984). In addition, they show abnormal patterns in the temporal relations of segmental structure within and between words (Baum, 1992; Baum et al., 1990; Gandour et al., 1993, 1994). Finally, they show impairments in the production of a number of phonetic dimensions under different speaking rate conditions, including voice-onset time in stop consonants, fricative duration as a cue to voicing, and vowel duration (Baum, 1993, 1996; Baum & Ryan, 1993; Kent & McNeill, 1987; McNeill et al., 1990). Studies exploring the temporal patterns at the sentence level also indicate deficits in temporal (durational) patterns, perhaps caused by speech planning deficits (Gandour et al., 1994). Because these phonetic impairments are not clinically perceptible but emerge only on acoustic analysis, they are thought to be SUBCLINICAL (cf. Baum et al., 1990; Vijayan & Gandour, 1995).

These subclinical impairments in speech production found in left hemisphere posterior aphasics do not emerge in right hemisphere patients (Gandour et al., 1994; Kurowski et al., 1998). Thus, the increased variability in posterior aphasics is not due to a so-called brain-damage effect. Rather, these impairments suggest that the speech production system is a complex network involving both posterior and anterior brain structures. The role of these brain structures in speech production seems to be different, as shown by the differential patterns of deficits. Nevertheless, both anterior and posterior structures ultimately contribute to the speech production process.

The nature of the posterior mechanism contributing to articulatory implementation is not clear. Several hypotheses may be suggested, but at this point they remain speculative. It is the case that posterior fibers project anteriorly to the motor cortex system, and damage to those fibers could affect the speech implementation system itself. Alternatively, the auditory feedback system normally contributing to the control of the articulatory parameters of speech may be impaired. Finally, the speech planning mechanism allowing for the production of word strings at the sentence level may be shorter than normal. More research is required to determine the nature of the mechanisms involved, but what is clear is that the traditional dichotomy between production, subserved by anterior brain structures, and perception, subserved solely by posterior structures, is not supported.

Speech Perception

A review of Figure 5.1 shows that contact with the lexicon (and, ultimately, meaning) requires the mapping from sound structure to lexical

form. The auditory reception of words involves several potential transformations of the auditory input, including the encoding of the auditory input into a spectral representation based on the extraction of more generalized auditory patterns or properties from the acoustic waveform, the conversion of this spectral representation to a more abstract feature/phonological representation, and ultimately the selection of a word candidate from a set of potential word candidates sharing phonological properties with the target word. Studies exploring the perception of the sound structure of language in aphasia have focused on these different "levels" of analysis to determine whether aphasic patients display impairments in perceiving the phonological patterns of language; whether they show impairments in perceiving the acoustic properties that correspond to the phonetic categories of speech; and whether they show impairments in mapping from sound structure to lexical form. The assumption has been that deficits at any one or all of these levels could potentially underlie or contribute to impairments in auditory language comprehension.

Phonological Patterns of Speech Perception

Similar to production studies with aphasic patients, most studies exploring the role of speech perception deficits in auditory comprehension impairments have focused on the ability of aphasic patients to perceive phonemic or segmental contrasts. Studies on segmental perception have indeed shown that aphasic patients evidence deficits in processing segmental contrasts. These studies have explored patients' abilities to discriminate pairs of words or nonwords, for example, *pear* versus *bear, pa* versus *ba,* or they have asked subjects to point to the appropriate word or consonant from an array of phonologically confusable pictures or nonsense syllables. Although the "classical" view of aphasia proposed that reception abilities primarily lie in posterior brain structures, and hence phonological impairments are more likely found in Wernicke's aphasics (Luria, 1966), results show that nearly all aphasic patients, regardless of clinical type and underlying neuropathology, show some problems in discriminating phonological contrasts (Blumstein, Baker, & Goodglass, 1977; Jauhiainen & Nuutila, 1977; Miceli, Calatgirone, Gainotti, & Payer-Rego, 1978; Miceli, Gainotti, Caltagirone, & Massulo, 1980) or in labeling or identifying consonants presented in a consonant–vowel context (Basso, Casati, & Vignolo, 1977; Blumstein, Cooper et al., 1977). Typically, patients have considerably more difficulty in identification tasks than they do in discrimination tasks (Gow & Caplan, 1996). These problems emerge for the perception of both real words and nonsense syllables.

Although there are more errors in the perception of nonsense syllables

than real words, the overall patterns of performance are similar and essentially mirror the patterns found in the analysis of phonological errors in speech production. Namely, aphasics are more likely to make speech perception errors for consonant contrasts than for vowel contrasts. Most perceptual errors occur for consonants when the test stimuli contrast by a single phonetic feature than when they contrast by two or more features (Baker, Blumstein, & Goodglass, 1981; Blumstein, Baker, & Goodglass, 1977; Miceli et al., 1978; Sasanuma, Tatsumi, & Fujisaki, 1976). Among the various types of feature contrasts, the perception of place of articulation contrasts and the perception of voicing contrasts are particularly vulnerable (Baker et al., 1981; Blumstein, Baker, & Goodglass, 1977; Gow & Caplan, 1996; Miceli et al., 1978). Finally, perceptual performance is influenced by the phonetic position in which the contrast appears. More perceptual errors occur for consonants, for example, in medial and in final position than in initial position. Most patients who show such phonological perceptual deficits display impairments that affect the entire phonological inventory, although there have been a few cases reported in which patients have shown a selective impairment of a particular phonological contrast (cf. Caplan & Utman, 1994).

Interestingly, similar patterns emerge in normal subjects when perceiving speech under difficult listening conditions (cf. Miller & Nicely, 1955). That the patterns of perception for real words and for nonwords is similar among the aphasics is consistent with the view that the organizational properties of the sound structure of language are still intact. The greater impairment for nonwords is consistent with the view that nonwords do not have a lexical representation and thus are not a part of the lexical network; as a consequence, they are particularly vulnerable, because they can only be processed with respect to their sound structure. The similar phonological patterns of misperceptions of words and nonwords suggest that the same processing mechanisms are used in the mapping from acoustic structure to phonological structure (cf. Figure 5.1).

Phonetic Patterns of Speech Perception

What is not clear from many of the studies exploring the perception of segmental contrasts is whether the failure to perceive such contrasts reflects an impairment in the perception of abstract phonetic/phonological features or alternatively an impairment in extracting the acoustic cues from the speech signal which underlie the phonetic/phonological features. To explore this issue, several studies have investigated the perception of the acoustic parameters associated with phonetic features. To this end, subjects are presented with an acoustic continuum in which certain acoustic cues

or acoustic attributes are systematically and parametrically varied. Subjects are asked to either categorize or identify the phonetic category of the stimuli or, alternatively, to discriminate pairs of stimuli from the continuum.

The acoustic cues associated with place of articulation in stop consonants and voicing have been the most extensively studied (Basso et al., 1977; Blumstein, Cooper et al., 1977; Blumstein, Tartter, Nigro, & Statlender, 1984; Gandour & Dardarananda, 1982). For voicing, the acoustic dimension varied was voice-onset time distinguishing [d] from [t], and for place of articulation, the dimension varied was the frequency of the formant transitions appropriate for /b d g/ and the presence or absence of a burst preceding the transitions. Results showed that in general aphasic patients had great difficulty in performing these tasks, and particularly in perceiving synthetic (as compared to natural) speech stimuli (cf. Gow & Caplan, 1996). If aphasic patients could perform either of the two tasks (labeling or discrimination), it was the discrimination task. Most importantly, the discrimination functions were generally similar in shape and the locus of the phonetic boundary was comparable to those of normals, even for those patients who could not reliably identify the stimuli.

The fact that no perceptual shifts were obtained for either the discrimination or the labeling functions for aphasic patients, that the discrimination functions remained stable even in those patients who could not label the stimuli, and that the patients perceived the acoustic dimensions relating to phonetic categories in a manner similar to normals suggests that aphasic patients do not have a deficit specific to the extraction of the spectral patterns corresponding to the phonetic categories of speech. Rather, their deficit seems to relate to the threshold of activation of the phonetic/phonological representation itself or to its ultimate contact with the lexicon. Results consistent with this view have been obtained in recent studies investigating whether aphasic patients are perceptually sensitive, as are normal subjects, to subphonetic, within phonetic category, acoustic differences, and whether such differences affect lexical access.

In contrast to the segmental features of speech, the prosodic cues (i.e., intonation and stress) are less affected in aphasia. Severely impaired aphasics have been shown to retain some ability to recognize and distinguish the syntactic forms of commands, yes–no questions, and information questions when marked only by intonation cues (Green & Boller, 1974), even when they are unable to do so when syntactic forms are marked by lexical and syntactic cues. The perception of word accent in Japanese is less impaired than the perception of segmental cues (Sasanuma et al., 1976), and the perception of stress as a semantic cue distinguishing different lexical items in English is also relatively spared (Blumstein & Goodglass, 1972).

Nonetheless, as with intonation cues, patients' performance is not completely normal. A number of studies have revealed impairments in the comprehension of lexical/phrasal stress contrasts, for example, *hótdog* versus *hotdóg* (Baum, Kelsch, Daniloff, & Daniloff, 1982; Emmorey, 1987), as well as sentential contrasts, for example, "he fed her *dog* bíscuits" versus "he fed her *dóg* biscuits" (Baum et al., 1982). Similar findings emerged for the perception of tone contrasts serving as lexical cues in Thai (Gandour & Dardarananda, 1983) and Chinese (Naeser & Chan, 1980). Importantly, no differences have emerged in any studies between the performance of anterior and posterior aphasics, a finding consistent with the results for the perception of phonemic contrasts.

The Relation between Speech Perception and Auditory Language Comprehension

It does not seem to be the case that speech perception impairments are the basis for auditory language comprehension impairments. That is, there does not seem to be any clear-cut relationship between deficits in perceiving phonological contrasts or in the acoustic cues underlying these contrasts and the level of auditory language comprehension. Patients with good auditory comprehension skills have shown impairments in speech processing; conversely, patients with severe auditory language comprehension deficits have shown minimal speech perception deficits (Baker et al., 1981; Basso et al., 1977; Blumstein, Baker, & Goodglass, 1977; Blumstein, Cooper et al., 1977; Jauhiainen & Nuutila, 1977; Miceli et al., 1980; for general discussion, see Boller, 1978). The patients in these studies have been drawn from a broad range of clinical types and underlying neuropathology, including Broca's aphasics, Wernicke's aphasics, mixed anterior aphasics, and conduction aphasics. For example, Wernicke's aphasics have performed better than mixed anterior patients on a speech discrimination task, despite the fact that the Wernicke's aphasics have the more severe auditory language comprehension deficits (Blumstein, Baker, & Goodglass, 1977).

Thus, although speech perception studies with aphasic patients have supported the view that perceptual impairments reflect the misperception of phonetic features (i.e., the more abstract phonological properties of words or word candidates), they do not support the classical hypothesis that speech perception deficits per se underlie the auditory language comprehension impairments of Wernicke's aphasics, nor do they support the proposal that speech perception impairments are restricted to patients with left posterior brain damage, and in particular, temporal lobe pathology.

Mapping of Sound Structure to Lexical Form

The role of the sound structure of language is ultimately to provide the medium for listeners to contact meaning. Thus, as Figure 5.1 shows, the sound structure of language is ultimately mapped onto lexical form. A question is whether aphasic patients show impairments in the processes and mechanisms responsible for this mapping. Aphasic patients representing a broad array of clinical types (including Broca's, Wernicke's, and conduction aphasics) show an interaction between phonological and semantic factors in lexical access (Baker et al., 1981). In particular, as semantic demands increase (i.e., as subjects are required to process auditorily presented words for meaning), sensitivity to phonological distinctions suffers; as phonological distinctions become more similar, and hence more difficult, semantic processing suffers (cf. also Martin, Wasserman, Gilden, & West, 1975). These results raise the possibility that the auditory language comprehension impairments of aphasic patients could reflect an impairment in the processes of mapping sound structure to the lexicon rather than on impairments in perceiving the sound structure of language per se.

To date, there are only a handful of studies that have explored this question. The results of these studies have shown interesting dichotomies between the performances of Broca's and Wernicke's aphasics. These dichotomies are of particular interest because they are among the first demonstrations of *qualitative* differences between these groups of patients on tasks involving speech processing. Most of these studies have explored priming in an auditory lexical decision task. Some have explored semantic priming and the effects of various types of sound structure distortion on the magnitude of semantic priming (Aydelott & Blumstein, 1995; Milberg, Blumstein, & Dworetzky, 1988; Utman, 1997). Others have explored repetition or rhyme priming and the effects of various phonological factors on the magnitude of priming (Gordon & Baum, 1994; Milberg et al., 1988). In the latter case, the focus is on how sound structure maps onto lexical candidates in the lexicon, rather than on how such lexical activation affects the lexical network more broadly.

The results of these studies can be summarized as follows. Both groups seem to have auditory memory impairments in that they are unable to hold a stimulus in phonological form for a period of time, particularly if the stimulus is a nonword and thus cannot be held in a semantic form. Nonetheless, neither Broca's nor Wernicke's aphasics seem to have deficits that involve the mapping from sound structure to the lexicon. Although their performance is not normal, they show sensitivity to phonological organization (Blumstein et al., 1997; Milberg et al., 1988; but cf. Gordon & Baum, 1994), as well as sensitivity to within phonetic category distinctions

such as differences in voice-onset time (Aydelott & Blumstein, 1995; Utman, 1997). Instead, both Broca's and Wernicke's aphasics seem to have deficits that are lexical in nature and appear to relate specifically to lexical activation.

Broca's aphasics appear to have a reduced level of activation of lexical candidates. Thus, a lexical target fails to activate its lexical node to the same extent as in normals, not only affecting the activation of the lexical node itself but also the activation of its lexical network. As a result, when auditorily presented prime stimuli contain initial stop consonants that are phonologically distorted nonwords (such as *gat*, which is phonologically related to *cat*), Broca's aphasics fail to show *any* priming for semantically related target words such as *dog* (Milberg et al., 1988). They also fail to show semantic priming for words that have a voiced competitor when the initial voiceless stop consonant has been acoustically modified (such as a shortened voice-onset time value for *pear*, which has a voiced competitor *bear*) (Utman, 1997; Utman & Blumstein, 1995). Under the same conditions, normal subjects show significant, but reduced, semantic priming relative to the priming that occurs for a target preceded by an undistorted prime stimulus (e.g., *cat–dog; pear–fruit*). Broca's aphasics are also greatly affected by the lexical status of a stimulus, making many more voiced (or voiceless) responses compared with normals when the voiced (or voiceless) end of a voice-onset time continuum is a word (Blumstein, Burton, Baum, Waldstein, & Katz, 1993).

In contrast to Broca's aphasics, Wernicke's aphasics appear to have a deficit manifested by an overactivation of the lexicon as a result of a failure to inhibit the activation of a lexical candidate and/or alternative lexical competitors. As a consequence, Wernicke's aphasics show semantic priming over a greater range of stimuli than found with normals. They show as much semantic priming for *dog* when it is preceded by a phonologically distorted prime stimulus such as *gat* as when it is preceded by an undistorted prime word such as *cat*. They also fail to show a lexical effect in a phonetic categorization task as do normals, presumably because nonwords are not inhibited as quickly or as reliably as potential lexical candidates (Blumstein et al., 1993).

Taken together, these results suggest that the auditory comprehension deficits of both Broca's and Wernicke's aphasics lie in the processes of lexical activation. The two groups of patients, however, differ, in *how* the processes of lexical activation patterns are affected subsequent to brain damage. Critically, for both groups of patients, their deficits do not seem to reflect impairments in mapping sound structure to the lexicon. What may appear to be perceptual impairments then seem to be primarily manifestations of deficits in lexical activation. As such, although the patients may

appear to have deficits in analyzing the sound properties of language, these deficits are not what underlie their impairments, but rather are secondary to and a consequence of deficits in lexical activation.

Summary

This chapter explored the nature of deficits in the sound structure of language in aphasia. Phonological deficits in aphasia do not appear to reflect impairments of representation, but rather retrieval of or access to lexical form. Thus, in production, all aphasic patients show phonological output impairments that affect the selection and planning of speech. They are unable to retrieve consistently phonological form and plan its output. Such deficits arise from the processes involved in retrieving phonological representations from the lexicon or the short-term buffer and/or planning their production. Only anterior aphasics, and particularly Broca's aphasics, show a phonetic deficit characterized by an impairment in the articulatory implementation of sound structure. The constellation of impairments for anterior aphasics suggests that this phonetic disorder is articulatory, not linguistic, in nature. That is, it affects the implementation of particular articulatory maneuvers, ones that affect the timing of articulators as well as laryngeal control. Posterior aphasics also show evidence of a subtle phonetic output impairment. Qualitatively distinct from the phonetic output disorder of anterior aphasics, the underlying basis for this subclinical deficit is not yet understood.

Studies exploring the perception of the sound structure of language show that all aphasic patients display some impairments in the processing of speech sounds. They have difficulty in perceiving phonological contrasts across a range of tasks. Nonetheless, they do not have a deficit in the extraction of the spectral patterns corresponding to the phonetic categories of speech. Rather, their deficit seems to relate to the threshold of activation of the phonetic/phonological representation itself. Some preliminary studies also suggest that their deficits do not reflect impairments in mapping sound structure to the lexicon. What may appear to be perceptual impairments then seem to be primarily manifestations of deficits in lexical activation. As such, although the patients may appear to have deficits in analyzing the sound properties of language, these deficits are secondary to and a consequence of deficits in lexical activation.

These findings challenge a number of classical assumptions concerning phonological deficits in aphasia. Most specifically, they challenge the relation between the anterior/posterior anatomical distinction and the functional expressive/receptive distinction. The similar patterns of perfor-

mance across the aphasic syndromes in the patterns of phonological output indicate that both anterior and posterior brain structures contribute to the selection of phonological representations as well as to phonological planning in speech production. Moreover, although speech perception studies have supported the view that perceptual impairments reflect the misperception of phonetic features (i.e., the more abstract phonological properties of words or word candidates), they do not support the classical hypothesis that speech perception deficits per se underlie the auditory language comprehension impairments of posterior and particularly Wernicke's aphasics, nor do they support the proposal that speech perception impairments are restricted to patients with left posterior brain damage, and in particular, temporal lobe pathology.

Acknowledgments

This research was supported in part by National Institutes of Health Grant DC00314 to Brown University and DC0081 to the Boston University School of Medicine.

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6

Lexical Deficits

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A Lexical Deficit?

What can it mean to say that a patient has a LEXICAL DEFICIT? There are a number of different things one could mean by such a term. In this chapter, we begin by reviewing several possibilities and then selecting the only interpretation of "lexical deficit" that we believe can provide an adequate basis for understanding a patient's disorder. It should become apparent from this discussion that a pattern of impaired performance can be understood only in the context of a theory that describes the cognitive processes required to perform skills that we want to understand, such as naming, reading, writing, and sentence processing. The importance of a theory of this mental machinery or "cognitive structure" will become apparent as we consider actual patterns of patient data and attempt, in each case, to draw conclusions about the nature of the underlying deficit.

One possibility is that lexical deficit refers, in general, to a DEFICIT IN LEXICAL PROCESSING, that is, to any difficulty in processing words in the reading, spelling, comprehension, and production of single words. A potential problem with this interpretation is that it would include, in the population of patients with lexical deficits, patients who have difficulty in processing words for reasons that may have nothing to do with the words themselves. For example, we might have a patient who demonstrates difficulty in matching an orally presented word with a picture. This performance suggests a deficit in comprehension of the meaning of words. However, it may be the case that this particular patient is not able to discriminate sounds normally and, as a consequence, demonstrates impaired performance on the word–picture matching task. Most would agree

that, although such a deficit might reveal itself as a word comprehension deficit, it would be misleading to conclude that such a patient has a lexical deficit. Therefore, the observation that a patient has difficulty in processing words would clearly be insufficient grounds for concluding that a patient has a lexical deficit.

Another possibility is that the production of LEXICAL ERRORS (e.g., producing one word for another: reading *table* for *chair, chain* for *chair,* etc.) should be considered the hallmark of a lexical deficit. As in the previous case, this criterion does not guarantee that lexical errors are produced as a result of a deficit at a level of processing that is specific to words. An example is provided by certain patients with unilateral neglect who have difficulty in processing the portion of space contralateral to the site of their lesion (i.e., a left-hemisphere lesion might result in difficulty in processing the right side of a stimulus). It has been reported (Behrmann, Moscovitch, Black, & Mozer, 1990; Caramazza & Hillis, 1990; Costello & Warrington, 1987; Ellis, Flude, & Young, 1987; Hillis & Caramazza, 1992; Kinsbourne & Warrington, 1962) that some of these patients make errors such as reading *peach* or *pea* for *pear,* where the right side of the word is deleted or substituted. These would clearly be defined as lexical errors and yet there is no sense in which one would want to characterize the deficit as a deficit in word processing. These patients make comparable errors with pictures and other spatially arrayed stimuli.

Another difficulty that arises from considering the production of lexical errors as the indicator of a lexical deficit is that a patient could, in fact, have damage to mechanisms specific to word processing and yet not produce lexical errors. Thus, for example, a patient, when asked to name pictures, may respond to a picture of a chair by saying /dʃaur/. Although the error would not be classified as a lexical error, it may result from damage to representations of the phonological forms of words—representations that guide subsequent articulatory processes.

Finally, even if we were to ascertain that a patient was having difficulty with words and only words, we could still remain in the dark with respect to the *nature* of the difficulty. For example, we can easily imagine that a patient might produce *peach* for *pear* as the result of confusion regarding the exact meaning of words. However, this would be a very different deficit from the one in which the same error is produced—*peach* for *pear*—but the patient clearly understood the meaning of the word and responded with a word with a similar meaning because he or she was unable to produce the target word. For the first patient, the evidence suggests an impairment in the system that represents word meaning; for the second, a word production system would appear to be implicated. Thus ideally we would want

to know not only that a patient has a deficit restricted to the processing of words, but also which particular aspects of word processing have been affected.

The discussion thus far reveals that it is not possible to understand a deficit if we attempt to interpret *directly* the surface manifestations of the deficit—task performance and error patterns. We have seen that deficits to nonlexical mechanisms might affect the processing of words and even result in lexical errors. Furthermore, we have seen that similar error patterns may result from very different impairments within a system dedicated to the processing of words. It becomes apparent that in order to determine correctly the source, or location within a system, of a deficit we must know first which mechanisms possibly *underlie* performance on a particular task, and second, the functioning and purpose of each mechanism so that we can determine how damage to any one of them would be reflected in task performance and error patterns. Only then are we in the position to explore possible alternative explanations of the performance pattern of a particular patient, test hypotheses concerning the nature of the deficit, and, finally, decide whether the weight of the evidence favors one possible locus of impairment over another.

Thus, for example, in the case of a patient who makes errors in a word–picture matching task, it is only because we have reasons to believe that a mechanism that adequately discriminates speech sounds is required in the performance of such a task that we will go on to devise tests to determine whether or not such a mechanism is intact. How we proceed, therefore, is determined by what we believe to be the relevant underlying mechanisms. Our conclusions regarding the nature of the impairment in the patient will depend on the results we obtain from testing the integrity of this and any other component that we have assumed is required for performing the task.

It would appear that we can most usefully think of a lexical deficit as a DEFICIT TO A LEXICAL COMPONENT, that is, as an impairment to one or more of the mechanisms thought to be involved specifically in the processing of words. In order to do so, however, we must have a theory of what the possible lexical components might be, how they might function, and, as a consequence, the characteristics of performance we might expect to observe when they are damaged. We need a theory of the lexical processing system. Such a theory will be useful to the extent to which it can provide (a) a description of what is often referred to as the functional architecture of the lexical system, a description of the basic components of the system and how they are interrelated; and (b) a description of the internal structure of the individual components (Caramazza, 1988). We will spend some time

developing these points to lay the groundwork for a discussion of specific patterns of patient performance which will illustrate the role of a theoretical framework in the interpretation of performance.

A Functional Architecture of the Lexical System

One widely accepted theory of the functional architecture of the lexical system is that it consists of the set of autonomous yet interconnected components depicted schematically in Fig. 6.1 (Caramazza, 1988; Morton, 1981; Shallice, 1981). According to this theory, a major distinction is drawn between input and output components. Input components are those in-

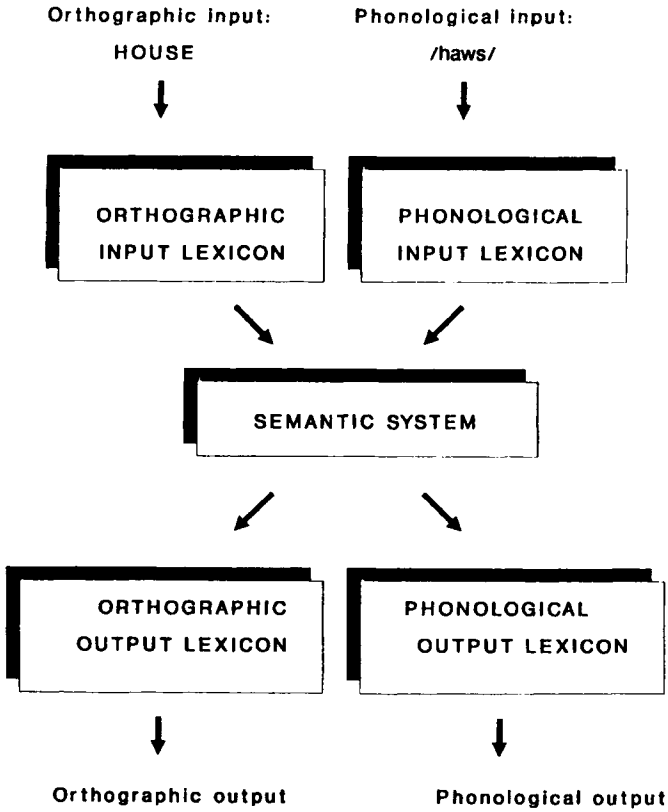

FIGURE 6.1. *A schematic representation of the lexical processing system.*

volved in the comprehension of words, and output components underlie the production of words. A second major distinction is drawn between the modalities of input and output, phonological or orthographic, such that each modality is represented separately for input (comprehension) and output (production). Thus the orthographic input lexicon (OIL), which includes those mechanisms involved in the recognition of written words in reading, is distinguished from the phonological input lexicon (PIL) involved in the recognition of spoken words. Before gaining access to these input lexicons, perceptual mechanisms process the stimulus, visual or phonological as the case may be, in order to represent information regarding the letters or phonemes in the stimulus. The modality of specific input lexicons are further distinguished from their corresponding output lexicons, which represent the orthographic and phonological information involved in the production of written and spoken words. These modality-specific lexical components are interconnected through a lexical semantic system that stores the semantic representations for words.

Each lexicon is thought to contain the information that is necessary to represent a word in a given modality for the purposes of production or comprehension, as the case may be. Thus, the OIL contains information regarding the familiar letter sequences—graphemes—that correspond to words. In reading, it is the OIL that allows one to recognize that the letter sequences *cloud* and *fish* are familiar words, whereas *clowd* and *fislh* are not. The OIL is said to contain lexical orthographic representations that are activated during reading, whereas the PIL contains the information regarding the phoneme sequences that constitute the words of the language that are activated in the course of listening to speech. The output lexicons contain comparable representations that provide the basis for written and spoken production.

The lexical semantic system is the repository of the meaning of words (Jackendoff, 1983; Miller & Johnson-Laird, 1976). In fact, the phonological and orthographic lexicons can be thought of as providing specific forms to the meaning of a word. Expressing a concept (e.g., four-legged domestic animal that barks) involves activation of semantic knowledge. This semantic representation is then given a phonological shape (/dag/) or an orthographic shape (D-O-G) as needed based on the information and mechanisms represented in the output lexicons. In comprehension, the input lexicons identify an orthographic or phonological form so that its meaning can then be activated in the semantic system and comprehension may occur.

We have claimed that patients' performance is best described in terms of impairment to one or more of the underlying processing components. However, because there is no way of examining the integrity of any par-

ticular component directly, we must, instead, examine the available evidence in order to make inferences regarding the condition of the underlying processing system. The evidence that we typically gather is that obtained from observing patient performance on different TASKS. If, for example, we assume that the task of oral reading involves the OIL, the semantic system, and the phonological output lexicon (POL), then a patient's performance in oral reading might provide information regarding these components. The task of silent reading might involve the first two components but not the third, whereas spontaneous speech would involve the semantic system and the POL but none of the input components. By observing the similarities and differences in a patient's performance on the different tasks we can make inferences regarding the integrity of the underlying components. According to such a theory, it is not the case that different components exist to perform different tasks, but rather that each component has a certain FUNCTION and whenever that function is required for any task, the component is utilized. Thus the same phonological output component is recruited in the tasks of reading and spontaneous speech, although the first task involves the orthographic input lexicon and the second does not. An important consequence of this fact is that we would expect that damage to a given component should be reflected in impaired performance on all tasks that require the component.

We present data from the neurologically impaired patient RGB, described by Caramazza and Hillis (1990a), to illustrate what the various components refer to and, at the same time, to show how a theory of cognitive processing can allow us to better understand patterns of impaired performance.

Table 6.1 presents the relevant data regarding RGB's performance. What is immediately striking about this patient's performance pattern is that a comparable level of impairment occurred *only* in the tasks of oral reading, oral naming, and tactile naming. For the three tasks the patient must produce a spoken word—phonological output. However, for the first, the input is orthographic; for the second, pictures serve as stimuli; and for the third, the patient's tactile experience serves as input. The tasks share the semantic processing component as well as a common output mode. The fact that the tasks result in a similar pattern of errors suggests that the patient may have a deficit to some component that the tasks share. In the context of the functional architecture presented earlier, this indicates a deficit either to the semantic system or to the POL (see Figure 6.2).

The functional architecture makes different predictions for the two hypotheses. According to the interrelationship among the components represented in Figure 6.2, a SELECTIVE deficit in the semantic system should be reflected equally in both phonological and orthographic output processes.

TABLE 6.1
Percentage Correct for RGB across Eight Tasks

Task	Percentage correct
Oral reading	69% (131/191)
Oral naming: pictures	66% (126/191)
Oral naming: tactile	64% (30/47)
Written naming: pictures	94% (179/191)
Writing to dictation	94% (179/191)
Auditory word/picture matching	100% (191/191)
Auditory/printed word matching	100% (47/47)
Printed word/picture matching	100% (191/191)

Tasks requiring orthographic output include writing to dictation and written naming of objects. The fact that RGB did not make errors on these tasks comparable to the errors made on tasks requiring phonological output (Table 6.1) allows us to rule out the hypothesis that a semantic deficit was responsible for the errors observed in reading and object and tactile naming. We can therefore conclude that the probable locus of impairment in this patient was the POL, suggesting that the patient was unable to activate normally certain phonological representations in this component of the lexical system. The implication is that for RGB, processing prior to the POL was intact; in reading, for example, the written form activated a representation in the OIL, which allowed the patient to recognize the sequence of letters as familiar, and, furthermore, a semantic representation was activated in the semantic system, permitting the patient to understand the meaning of the word he could not produce. Good reading comprehension was reflected in perfect performance on the written word–picture matching task (Table 6.1). It was only when the semantic representation must activate its corresponding phonological form for output that the patient's performance was affected (for reports of other patients with deficits in spoken but not written naming, see Basso, Taborelli, & Vignolo, 1978; Michel, 1979).

The observation that the POL can be damaged without comparable damage to the orthographic output lexicon (OOL) confirms the claim of this particular architecture that there are modality-specific output systems. RGB made very few errors in writing the names of pictures or the words that were dictated to him and that he could not name. This pattern of results suggests a relatively intact OOL in the face of a damaged POL. The fact that observed patterns of performance can find an explanation within a particular theory increases our confidence in the accuracy of the theory.

In addition to the distinction between orthographic and phonological

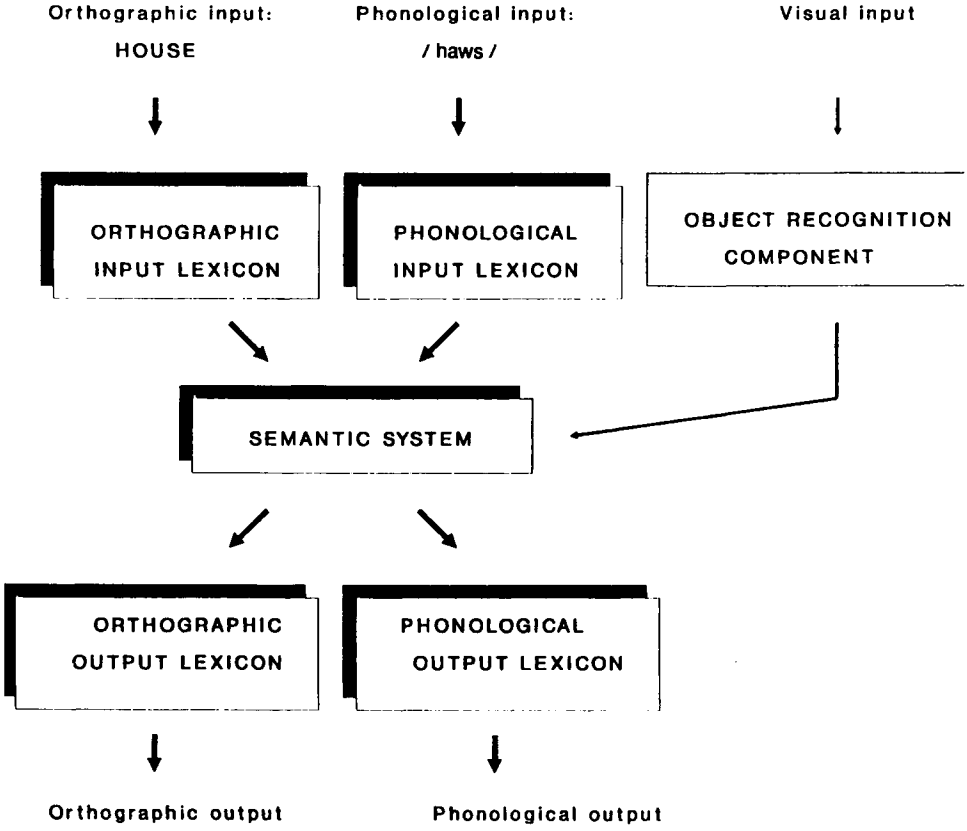

FIGURE 6.2. *The lexical processing system shown in Fig. 6.1, including processing components required for object recognition and naming.*

modalities, the architecture of the lexical processing system also distinguishes between input and output mechanisms. Consequently, it predicts that phonological processing may be impaired for output but not for input, or vice versa. In the case of RGB we have documented an impairment in *output* lexical phonology, coexisting with normal performance on tasks that require intact *input* lexical phonology. Both writing to dictation and auditory word–picture matching involve phonological input—the recognition of spoken forms. In the first case, the patient must write words presented aurally; in the second, the patient must match an aurally presented word to a picture. As shown in Table 6.1, RGB performed very well on both of these tasks. This good performance, in the face of poor performance on

tasks requiring spoken output, supports the postulated distinction between input and output lexicons.

These results not only provide confirmation for the functional architecture of the lexical system shown in Figure 6.1, but they also rule out certain alternative configurations of lexical processing components. For example, a possible theory is one that places the POL before the OOL, thus requiring a phonological representation of a word to serve as the basis for retrieving its orthographic representation for spelling. However, such an architecture would not be able to account for RGB's pattern of performance. It would predict that errors observed in tasks involving the POL should always be reflected in responses involving the OOL. We have seen in the case of RGB, however, that errors involving phonological output were not reflected in tasks requiring written output.

With this example, we hope to have made clear (a) how a functional architecture of the lexical system allows us, on the basis of observed patterns of performance on different *tasks,* to make inferences about damage to *functional components;* and (b) how patterns of impaired performance can, in turn, be used to test and develop models of cognitive processes.

Aspects of the Internal Structure of the Functional Components

Up to this point we have characterized the functioning of the components of the lexical system in general terms, indicating only that the principal lexical components can be classified according to representational type—orthographic, phonological, or semantic—and level of processing—input or output. However, the level of detail at which we can describe these components will determine the level of detail at which we can make predictions regarding the nature of the impairments that result when a particular component is damaged. The more specific the model, the greater our understanding of patterns of impaired performance. We can, in fact, elaborate further on the structure and function of the lexical components. Here, we briefly discuss three aspects of the internal structure of the phonological and orthographic lexical components: differential accessibility, morphological structure, and word class, as well as certain aspects of the semantic component, such as semantic categories, abstractness, and representational format.

Differential Accessibility: Frequency

It has been observed that normal subjects are faster at recognizing that a string of letters corresponds to a legitimate word of the language when

the string corresponds to a frequently occurring word (e.g., *house*) than when a less frequent sequence (e.g., *enzyme*) is presented (Gordon, 1983; Howes & Solomon, 1951; Morton, 1969). This is usually referred to as a FREQUENCY EFFECT. This finding holds true both with orthographic representations in reading and with phonological representations in auditory recognition. These observations have been taken as evidence that the frequency of occurrence of an item in the language is reflected in the ease and speed with which lexical representations are activated.

Specifically, it has been suggested that we can think of each of the lexicons as a set of word recognition units, each unit with a threshold of activation. When appropriate input is presented, the corresponding access unit becomes activated and fires when activation exceeds the unit's threshold level. Thus, the letter sequence H-O-U-S-E will result in the activation of a stored representation of this letter sequence in the OIL; while in the PIL, the phoneme sequence /haws/ serves as the appropriate input for the phonological representation corresponding to *house*. In the output lexicons it is suggested that a semantic representation activates the appropriate output form of an item. For example, a semantic representation such as "a structure in which people can live" will activate the phonological form /haws/ in the POL, which will subsequently serve as the basis for pronouncing the word. Similarly, in the OOL, the same semantic representation will access the orthographic representation of the letter sequence H-O-U-S-E to be used subsequently in writing or spelling. The activation of a representation is thought to raise the resting activation level of the unit, so that upon subsequent presentation of the word, the unit will reach threshold more quickly. Frequent encounters with any given word will permanently lower its threshold, resulting in faster recognition times and, in this way, explaining the frequency effect observed with normal subjects.

In this account, we might expect that some forms of damage to a lexicon could affect the integrity of these word recognition units by elevating the thresholds such that some words will be more difficult to recognize and others may not be recognized at all. On this basis, we might expect that the already higher thresholds of low frequency words may be elevated to such an extent that many of these words will be accessed with much greater difficulty or not at all. This should be reflected in greater errors with low- versus high-frequency items. This is, in fact, observed in patterns of impaired performance. RGB, for example, was able to read 40% of high-frequency words, but only 29% of low-frequency words.

Morphological Structure

A number of theoretical and empirical reasons indicate that the representation of lexical items cannot be simply in the form of an unstructured

string of phonemes or graphemes (e.g., W+A+L+K+I+N+G), but that instead the morphological structure of an item must be represented (e.g., WALK + ING). The most compelling reason for believing that morphological structure is represented in the lexicon is the fact that human language is productive: we can comprehend and produce specific morphologically complex forms that we may not have previously encountered. Thus, although we may have never read the word *chummily* we will know that it must consist of CHUMMY + LY. If *chummy* and *ly* are represented in the lexicon we will know that *chummily* should mean approximately "like friends + in that manner" or "in a chummy manner." Additional support for the notion that morphological information is represented in the lexicon is the observation that words in graphemic or phonological form are normally understood or produced in a sentence context. In such a context, morphological information is critical for a correct syntactic treatment of the sentence. Thus, for example, to produce the sentence, "I *walk* slowly while my dog *walks* quickly beside me," one must have the knowledge that *walk* (1st person) = WALK, whereas *walk* (3rd person) = WALK + S. If every word we encountered consisted of an undifferentiated string of graphemes or phonemes, such abilities would not be easily accounted for.

These observations have led to the suggestion that lexical information is represented in a morphologically decomposed manner, although the exact manner in which such morphological information is represented is still unresolved. Thus some would argue that only root forms and derivation and/or inflection are indicated (e.g., CHUM + Y + LY), whereas others would argue that previously encountered stems are also represented (e.g., CHUMMY + LY). In support of the notion of morphological decomposition there is not only linguistic and psychological evidence from normal language processing (Badecker & Caramazza, 1989; Butterworth, 1983; Henderson, 1989; Taft, 1985), but also findings from impaired performance. We shall see that lexical components can be damaged such that certain morphological processes are disrupted but not others.

Form Class

In a morphologically decomposed lexicon there will be differences in the way that words of different parts of speech (i.e., FORM CLASSES) are represented. These differences are based in part on systematic differences in the morphology of different form classes (Caramazza, 1988). Thus, for example in English, nouns can be inflected with an S in forming the plural (*dog, dogs*), and transformed to adjectives in a derivational process that adds AL, (*nation, national*). Verbs can be inflected with S, ED, ING, and EN (*melts, melted, melting, taken*). Adjectives may take the comparative ER and the superlative EST (*big, bigger; small, smallest*); but function words (*on, of,*

this, that) are usually uninflected. Consequently, form class information must be represented in order to correctly restrict productivity—we will not form a superlative such as *walkest.*

The claim that form class information forms a part of lexical representation is further supported by the fact that brain damage can selectively affect one form class and not another. There have been a number of reports describing selective deficits of form class. For example, Miceli, Silveri, Villa, and Caramazza (1984) described patients who had greater difficulty in naming verbs than nouns, as well as patients who had the reverse order of difficulty. A similar dissociation between verbs and nouns was described by Miceli, Silveri, Nocentini, and Caramazza (1988) for comprehension. These authors also noted that a form class effect in production was not necessarily associated with a similar deficit in word comprehension (for a discussion of similar cases, see Baxter & Warrington, 1985; McCarthy & Warrington, 1985; Zingeser & Berndt, 1988).

Lexical Semantics

Although this chapter does not discuss in detail the difficult and highly debated subject of the internal structure of the lexical semantic system (for a review of some relevant issues, see Jackendoff, 1983; Miller & Johnson-Laird, 1976), certain points, necessary for subsequent discussion of patient data, can be made fairly easily.

The morphological decomposition hypothesis implies that the semantic representation of a morphologically complex item used to address the output components must consist of distinct subsets of features, each specifying the different parts of the lexical item (Miceli & Caramazza, 1988). The semantic representation would include root semantic features, derivational semantic features (where present), and inflectional semantic features. For example, the word *colpevoli* (Italian for *guilty*—plural) might have the semantic representation [(COLPA) (ADJ) (PL)], where COLPA is the semantic component for the root morpheme (COLP—); ADJ is the semantic component for the selection of an adjectival affix (EVOL—); and PL is the semantic component for the selection of the appropriate inflectional plural affix (L). This semantic representation specifies the output form addressed and assembled in either the phonological or orthographic output lexicons.

Besides a componential morphological structure, semantic representations may have a componential structure with regard to the meaning of individual morphemes. In other words, the meaning of an item may be represented in terms of semantic features (e.g., the semantic representation for *dog* may consist of the following features: animal, mammal, domestic, four

legs, barks, etc.). This characteristic of semantic representations allows for category relationships among lexical items. Words that belong to the same semantic category (e.g., *cat* and *dog*) will share more semantic features that words from different categories (e.g., *dog* and *cheese*).

The fact that items that share semantic features are structurally related to one another is reflected in certain empirical findings with normal subjects. For example, it has been found that processing an item—deciding whether or not *dog* constitutes a word—facilitates the subsequent processing of semantically related items—making a decision for *cat* (e.g., see Meyer & Schvaneveldt, 1971). Presumably this facilitation is the result of the fact that dog and cat share certain semantic features and that with the activation of the features of dog certain features of cat will become active; and because a subset of the features of cat has been preactivated, the semantic representation corresponding to cat will take less time to reach a threshold level of activation when the subject is presented with the word *cat.* This time reduction will be reflected in faster processing times with the word—reading, semantic judgments, lexical decision, and so on, will be faster than if *cat* had been preceded by an unrelated word such as *chair.*

Providing additional support for the notion of category relatedness are findings of category-specific impairments. Goodglass, Klein, Carey, and Jones (1966) reported patients who appeared to have disproportionate difficulty with particular semantic categories; Dennis (1976), McKenna and Warrington (1978), and Warrington and McCarthy (1983) reported a selective impairment in the comprehension of body-part names; Warrington and McCarthy (1983) presented evidence of a selective impairment in the comprehension of inanimate object names; Warrington and Shallice (1984) reported a selective impairment in the ability to identify living things and foods, and Hart, Berndt, and Caramazza (1985) described a patient with a selective deficit in the category of fruits and vegetables (see also Basso, Capitani, & Laiacona, 1988; Berndt, 1988; Goodglass & Budin, 1988; A. E. Hillis & Caramazza, 1990; Sartori & Job, 1988; Semenza & Zettin, 1989; Silveri & Gainotti, 1988; Warrington, 1981b; Warrington & McCarthy, 1987, 1988).

Another type of dissociation observed in impaired performance is one between performances on abstract and concrete words. Thus, patients have been described who can read concrete words but have difficulty with abstract words (Marshall & Newcombe, 1966; K. Patterson & Marcel, 1977; Saffran & Marin, 1977; Shallice & Warrington, 1975). Other patients have the reverse pattern, abstract words are defined well whereas concrete often are not. For example, Warrington and Shallice (1984) described a patient who defined *debate* as "discussion between people, open discussions between group." *Ink,* however, was defined as "food, you put on top of

food you are eating, a liquid" (see also Warrington, 1975, 1981a). Although it is unclear how the properties of abstractness and concreteness are represented, this dissociation has been interpreted as indicating that abstractness is a relevant dimension of the organization of semantic information.

Having briefly described the arrangement of the functional components of the lexical system, as well as some aspects of their internal structure, we can evaluate the role that such a theory can play in the interpretation of patients' performance. A patient's impairment is often most strikingly reflected in the types of errors produced in performing different tasks. Therefore, we have organized the following discussion in terms of error types: nonword errors, semantic errors, and morphological errors. It is not our intention to provide an exhaustive survey of error types, but instead to use certain error types to illustrate how, within the context of a theory of lexical processing, we can better understand the origin of the errors and the functioning of the system that underlies them. In this context, we discuss issues of current controversy in the field, such as the grouping and classification of patients, the usefulness of syndromes, and the notion of compensatory strategies.

NONWORD ERRORS

Table 6.2 shows errors made by patients JG (Goodman & Caramazza, 1986a) and ML (Hillis & Caramazza, 1989) in oral and written spelling to dictation. Although ML displayed a more severe impairment than JG, each patient performed with comparable accuracy in the written and oral spelling of words: 65% / 74% for JG and 25% / 36% for ML. Nonword responses, such as those in Table 6.2, constituted the bulk of errors for both patients in the two tasks.

According to our model of lexical processing (Figure 6.1), a lexical deficit exclusively to the ability to spell would result from damage to the OOL.

TABLE 6.2
Examples of Errors and Accuracy Rates for JG and ML in Written and Oral Spelling to Dictation

Task	JG	ML
	senate → cenit	dumb → dub
	debt → dete	priest → rpiest
	urge → erg	lamb → llamb
	severe → savier	fabric → frbric
	mercy → mursy	degree → dgree
Written spelling	74% (241/326)	25% (81/326)
Oral spelling	65% (212/326)	36% (15/42)

The OOL is thought to contain information about the abstract letter sequences that form the basis for written or oral spelling of words. It has been proposed that the representation of the letters is abstract in the sense that letter identities but not specific aspects of the letter form, such as case, font, and name, are represented. Consequently, both written and oral spelling are based on the orthographic information represented in the OOL. The specific form of the output, letter shape for a written response and letter names for an oral response, is computed at a later stage of processing by allographic and letter-name conversion devices, respectively (see Figure 6.3). Data from neurologically impaired patients have confirmed this proposed functional architecture of peripheral spelling processes (Ellis, 1987; Goodman & Caramazza, 1986b; Kinsbourne & Rosenfeld, 1974; Kinsbourne & Warrington, 1965). Thus, for our purposes, we can assume that damage to the OOL should affect both written and oral spelling equally.

If we consider levels of performance, the fact that accuracy in oral and written spelling are comparable is consistent with a deficit to the OOL. However, comparable levels of performance in oral and written naming does not require a deficit to the OOL—such a conclusion would be premature without first ruling out alternative loci suggested by the model of lexical processing. The other major lexical component involved in spelling to dictation is the PIL; thus it is possible that both patients were impaired in the recognition of spoken words and, for this reason, failed to spell them correctly. We can reject the hypothesis of a deficit to the PIL in these patients because they both showed excellent auditory comprehension abilities: JG correctly defined 98% of the words dictated to her, whereas ML scored 100% on the auditory comprehension subtests of the Boston Diagnostic Aphasia Exam (BDAE; Goodglass & Kaplan, 1972). Furthermore, both patients made errors on a written naming task where they were required to write the name of a pictured object, which, therefore, did not involve the PIL.

Up to this point, the performance patterns of the two patients look quite similar, suggesting an impairment to the OOL. Earlier we discussed the fact that we could expect that damage to a lexicon would result in a frequency effect such that performance with low-frequency words would be more impaired than performance with high-frequency words. Table 6.3 presents the performance levels of these patients with high- and low-frequency words. JG showed a significant effect of frequency in both oral ($\chi^2 = 31.94, p < .001$) and written spelling ($\chi^2 = 49.18, p < .001$), whereas ML did not. Thus these two patients differed with respect to what is often considered to be an indicator of a lexical deficit, suggesting that we might want to explore further before localizing the deficit of both patients to the OOL.

Thus far we have simply examined overall *performance levels* on a vari-

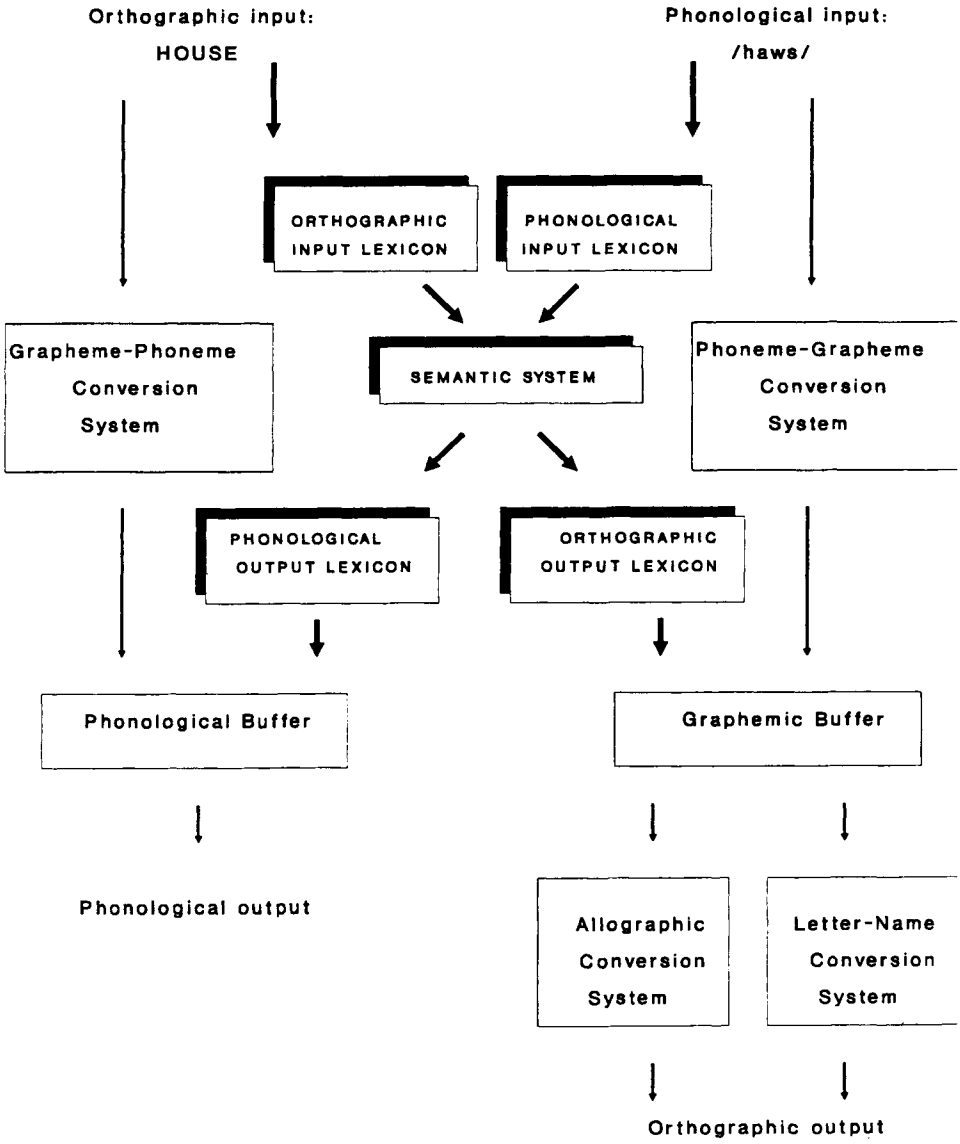

FIGURE 6.3. *The lexical processing system shown in Fig. 6.1, including processing components that allow for the mapping of graphemes to phonemes in reading as well as for the assembly of graphemes from phonemes in writing. In addition, output buffers and conversion mechanisms are depicted.*

TABLE 6.3
Accuracy in Written and Oral Spelling of High- and Low-Frequency Words for JG and ML

Task	JG	ML
Written Spelling		
High Frequency	85% (124/146)	28% (41/146)
Low Frequency	46% (67/146)	26% (38/146)
Oral Spelling		
High Frequency	89% (130/146)	33% (6/18)
Low Frequency	60% (88/146)	39% (7/18)

ety of tasks. However, another potential source of information regarding the nature of a deficit lies in the *form of the errors* that are produced. We will show that a consideration of the form of a patient's errors will often provide much more detailed and specific information regarding the nature of a deficit. We noted that both patients produced nonword responses. Nonetheless, when we consider the errors more closely we notice that for JG, the responses, although incorrect, would sound like the target word if pronounced (e.g., SILENCE → sylence); that is, the responses consisted of plausible although incorrect spellings (for descriptions of patients with similar error patterns, see Baxter & Warrington, 1987; Beauvois & Derouesne, 1981; Sanders & Caramazza, 1990). In fact, 88% of JG's responses could be categorized as "phonologically plausible errors," whereas only 7% of ML's errors were of this type. ML's errors contained substitutions (e.g., HAPPY → fappy), deletions (e.g., JUNK → jnk), transpositions (e.g., TIGER → tgier), and insertions (BOTTLE → bolttlee) of letters that did not result in a plausible phonological rendition of the target word.

It has been suggested that in order to be able to develop a spelling for unfamiliar words, we must have knowledge of sound–letter relationships (e.g., the sound /f/ may be written as F, GH, PH, etc; see Cummings, 1988). This is information that we have acquired through our experience with the written language and is often referred to as knowledge of phoneme–grapheme correspondences. The system, which produces a written form corresponding to a phonological input, is often referred to as the phoneme–grapheme conversion system or PGC system. Thus, if we wished to spell a word whose spelling we did not know, /hɛzIkæst/, we would probably arrive at spellings such as HEZIKAST, HESICAST, HESYCAST, and HESYCHAST. All would be plausible and all could be produced by a system that represents information such as /z/ → S or Z, /I/ → I or Y, and so on. Without having previously encountered the word in its written form and stored

a representation for its spelling in the OOL, we would have no way of knowing that the last alternative is, in fact, the correct one.

The form of JG's errors supports the hypothesis that for those words for which representations in the OOL were damaged, she made use of her knowledge of sound to print correspondences in English to produce a plausible spelling: given a phonological input—the dictated word, for example—she applied phoneme–grapheme rules to arrive at a possible spelling (see Figure 6.3).

In English, words vary in the degree to which their correct spelling would be produced by application of such a system. Thus, if the orthographic representation corresponding to /dæn/ were inaccessible for whatever reason, application of phoneme–grapheme rules would most likely arrive at the correct spelling DAN; however, if the orthographic representation of /jat/ were inaccessible, application of the same rules is most unlikely to result in the correct spelling YACHT. This contrast is often described by saying that English spellings differ in terms of the regularity or irregularity of the relationship between their pronunciation and their spelling. We refer to this as the "probability of correct phoneme–grapheme conversion" (see Hanna, Hanna, Hodges, & Rudorf, 1966, for the PGC probabilities in English). Words such as /dId/ would be said to have a high phoneme–grapheme conversion probability, whereas words like *doubt* (which could be spelled as DOWT, DOUTE, DOUGHT, etc.) would have a low probability of being correctly produced by application of phoneme–grapheme correspondence rules.

Given these assumptions we can make specific predictions regarding JG's performance in spelling tasks; specifically, we would expect that (a) most high-frequency words, regardless of their probability of correct phoneme–grapheme conversion, should be spelled correctly because orthographic representations should not be damaged for these items; and (b) low-frequency items, being less accessible, will be more often spelled through the PGC system and we should therefore expect that the spelling of low-frequency items should differ depending on the phoneme-grapheme conversion probabilities of the items: low probability/low frequency words should be spelled incorrectly more often by the PGC system than high probability/low frequency words. Table 6.4 shows the results obtained by JG. High-frequency words showed no effect of phoneme–grapheme probability ($\chi^2 = .37$, ns), whereas low-frequency words could be distinguished according to grapheme–phoneme conversion probabities ($\chi^2 = 4.42$, $p < .05$).

Another means of determining whether JG had intact knowledge of PGC rules was to ask her to spell unfamiliar or invented nonwords, such as /klejk/, which we could expect to be spelled KLAKE, KLAIK, or

TABLE 6.4
JG's Written and Oral Spelling Performance with Words of High- and Low-Frequency and High and Low Phoneme–Grapheme (PG) Conversion Probabilities

	PG conversion probability	
Frequency	High	Low
High frequency		
Written spelling	100%	90%
Oral spelling	100%	98%
Low frequency		
Written spelling	80%	50%
Oral spelling	93%	65%

KLAICK. On the basis of her performance with familiar words, we would expect that she should produce phonologically plausible renditions of unfamiliar or invented items, just as normal subjects do (Campbell, 1983). In fact, JG was able to produce plausible spellings for 100% of the nonwords she was asked to write, and for 98% of those she was asked to spell orally. The evidence thus converges on an explanation of JG's performance that indicates that the OOL was damaged and that the responses observed when this patient was asked to spell were the result of the functioning of an undamaged PGC system.

The case of ML appears to be different. Good comprehension as well as the fact that both written and oral spelling were equally affected suggested a deficit to a component beyond the semantic system, yet shared by oral and written spelling processes. However, the absence of a frequency effect was an indication that the OOL might not be the locus of impairment in this patient. Phonologically plausible errors were not observed and words of both high and low GPC probability were equally affected: high, 37% correct; low, 30% correct. When asked to spell nonwords, ML performed similarly as she had with words: 38% correct in written spelling and 45% correct in oral spelling of nonwords. In fact, errors with nonwords took the same form as those with words: substitutions, deletions, transpositions, and additions of letters (e.g., mʃræm/ as RUSHRAM; /dansɛpt/ as DNCEPT; /fɔjt/ as FIOT; and /riʃ/ as RRECECH).

It has been assumed that a postlexical memory component or buffer is necessarily involved in written and oral spelling (Caramazza, Miceli, Villa, & Romani, 1987; Wing & Baddeley, 1980). The location of this component in relation to the lexical processing system is shown in Figure 6.3. The

purpose of this memory component, referred to as the graphemic buffer, is to store orthographic representations while allographic conversion and letter-name conversion processes take place. Allographic and letter-name conversion processes take abstract letter identities and assign them a particular form: name, case, and font as needed. These processes are thought to act serially, processing one letter at a time. The graphemic buffer maintains the whole letter string available while the sequential conversion processes take place. Thus, an impairment to the graphemic buffer would be expected to produce comparable error rates in the oral and written spelling of both words and nonwords.

It would appear that in the case of ML, the results described thus far are consistent with damage to the graphemic buffer:

1. Absence of a frequency effect. We would not expect lexical factors such as frequency to be reflected in damage to such a component because it is postlexical; that is, it should not matter whether a word is of high or low frequency in the lexicon—to the graphemic buffer it is simply a string of letters to be temporarily stored.
2. Similar performance in oral and written spelling. Because of its location within the processing system, we would expect oral and written spelling to be affected equally by damage to the graphemic buffer.
3. Similar performance with words and nonwords. Because the buffer stores orthographic information regardless of its origin, word and nonword spelling should similarly affected.
4. Memory components are typically restricted in the number of elements that can be held simultaneously active, and disorders of memory are often characterized by a reduction in the capacity of the component. Damage to the graphemic buffer is thought to result in more errors when longer sequences are stored. If we examine ML's accuracy in written and oral spelling according to the length of the response we find that whereas performance is as good as 61% (60/99) with three- and four-letter words, accuracy falls to 4% (2/50) with seven- and eight-letter words (see Table 6.5).

All of the evidence gathered suggests that ML, although impaired in the processing of words, did not have damage to the orthographic output lexicon (i.e., to the stored information regarding the orthographic representation of words), but rather to a postlexical memory component referred to as the graphemic buffer (for other cases with hypothesized damage to the graphemic buffer, see Caramazza et al., 1987; Miceli, Silveri, & Caramazza, 1987; Posteraro, Zinelli, & Mazzucchi, 1988).

In this section we described the impaired spelling of two patients who made superficially similar errors in both oral and written spelling. As a result of testing predictions motivated by the architecture of the lexical pro-

TABLE 6.5
ML's Spelling Accuracy in Three Tasks According to Stimulus Length

Letter length	Writing to dictation	Written naming (pictures)	Oral spelling
3–4	55% (41/75)	76% (13/17)	86% (6/7)
5	33% (27/123)	45% (10/22)	63% (12/19)
6	20% (17/86)	8% (1/12)	36% (10/28)
7–8	5% (2/42)	N/A	0% (0/8)

cessing system we have nonetheless concluded that one suffered from a lexical deficit to the OOL, whereas the other had damage to the nonlexical component referred to as the graphemic buffer. This example illustrates how apparently similar patterns of impaired performance may stem from damage to different underlying mechanisms.

It has been similarly hypothesized that for reading, as for writing, there is a similar system of rules that maps letters or letter clusters onto their possible pronunciations—typically referred to as a system of grapheme–phoneme correspondences (GPC; Figure 6.3; see Coltheart, 1978, and Venezky, 1970, for a discussion of this topic, but see Humphreys & Evett, 1985, for opposing views). In normal subjects, such a system would be used to produce a pronunciation for words that are not represented in the OIL, words we have never or seldom encountered previously in their written form. Consistent with this hypothesis is the performance of patients such as MP (Bub, Cancelliere, & Kertesz, 1985), who had no trouble reading nonwords but who was significantly impaired in word reading. MP's performance in reading resembled the pattern of impairment described for patient JG in writing, demonstrating significant effects of frequency (high-frequency words, 88% correct; low-frequency, 79%) and regularity (regular, 96% correct; irregular, 41% correct). Furthermore, the majority of reading errors consisted of phonologically plausible *pronunciations* of the written form (e.g., AISLE → /ejzəl/), often referred to as regularization errors. It was concluded that MP's performance pattern was, at least in part, the result of an impairment to the OIL in the context of an intact GPC system. This resulted in phonologically plausible readings of items for which a pronunciation could not be computed normally[1] (see Hillis & Caramazza, 1992, and papers in Pat-

1. A deficit to one of the systems that represents relationships between phonology and orthography does not necessarily result in damage to the other. For example, Beauvois and Derouesne (1979) describe a patient who, like JG, had difficulty writing words but not nonwords. In reading, however, the patient apparently had a deficit to the GPC system as evidenced by a difficulty only in reading nonwords.

terson, Marshall, & Coltheart, 1985, for other cases whose reading performance reflects the use of the GPC system).

We have seen that the patterns of performance we observe subsequent to brain damage are the result of the operation of the damaged components in concert with all available intact components. Thus, damage to the OOL in the presence of an intact PGC system will result in phonologically plausible responses in spelling; whereas damage to the OIL may result in phonologically plausible readings or regularization errors.

Patients who exhibit certain error patterns subsequent to brain damage are often described as using "compensatory strategies" (e.g., see Kolk & Van Grunsven, 1985). Thus JG might be said to produce phonologically plausible spellings as the result of a "strategy" induced by the presence of damaged components. It is not entirely clear what is meant by such a statement. One possibility is precisely what has been suggested in the preceding paragraph. It was argued there that, subsequent to brain damage, a person will produce an output on the basis of whatever damaged and intact components are available. In this account, phonologically plausible spellings simply reflect the functioning of the PGC system available to the person before brain damage, although this system is not used for familiar words because these, in an undamaged system, are represented in the OOL. Brain damage, therefore, simply reflects the output of a system given that certain components have been damaged (see Caramazza, 1984, 1986, for a discussion of the transparency principle). It is this view which allows one to use brain-damaged performance as a window into the cognitive system, and, as a consequence, as a tool that can be used to make inferences about what the system must have been like before brain damage—the work of cognitive neuropsychology.

The other possible interpretation of compensatory strategies is that, subsequent to brain damage, the cognitive system undergoes a transformation such that previously unrelated components establish links to one another and previously nonexisting components are developed. Such a view is, of course, a logical possibility. It is important to realize, however, the implications of this position. It would mean that there would be no theory of the cognitive system that we could use as a basis for interpreting observed patterns of impaired performance. This would be the case because the system would have been transformed, presumably differently for different patients according to individual needs as well as to the form and extent of the damage. We would have no way of knowing the shape of the transformation because data from one patient to another would be irrelevant given that each would be functioning with a differently configured cognitive system. Thus, we would not only no longer have a basis for interpreting patient data, but we would also not be able to use patterns of brain-damaged

performance to provide information concerning the structure of the normal cognitive system.

Although it is possible that neurological damage results in a reorganization of the cognitive system, because of the paralyzing implications of such a possibility, in the absence of positive evidence regarding reorganization, we should be willing to accept such a view only to the extent to which we are unsuccessful in accounting for patterns of brain damage in terms of a theory of the structure of the normal cognitive system.

SEMANTIC ERRORS

Naming typically refers to the act of producing, in written or spoken form, the name that corresponds to an item. Thus, one might pronounce or write the name of an object, a picture, or an item or action one has in mind. In terms of our lexical processing model, object or picture naming would require an object recognition component for input, semantic processing, and the use of the POL or OOL to produce spoken or written output, respectively (see Figure 6.2). Naming an item one has in mind would originate in the semantic system and involve one of the output lexicons.

We begin by presenting data produced by two neurologically impaired patients with comparable error rates in the oral naming of objects presented visually and tactilely. It will soon become apparent that performance on a number of other tasks will need to be considered before we can draw conclusions about the nature of the patients' underlying deficits. Table 6.6 gives examples of some of the actual errors produced by RGB in naming tasks. The responses (e.g., *pineapple* for *banana*) clearly show a semantic similarity to the target and are, therefore, commonly referred to as semantic errors. In fact, as shown in Table 6.6, 70/71 of RGB's errors on these tasks

TABLE 6.6

Examples of Semantic Errors in Oral Naming with Object and Tactile Input for KE and RGB as Well as the Proportion of Semantic Errors/Total Errors

Task	RGB	KE
	lemon → sour	lemon → orange
	clam → octopus	clam → crab
	kangaroo → racoon	racoon → rabbit
	mittens → socks	shirt → sock
	banana → pineapple	peach → banana
	Semantic errors/total errors	
Object naming	98% (63/64)	85% (126/149)
Tactile naming	100% (17/17)	95% (21/22)

can be classified as being semantically related to the stimulus item. Table 6.6 also gives examples of errors produced by patient KE on the same tasks (Hillis, Rapp, Romani, & Caramazza, 1990). These errors also bear a semantic relationship to the target. As was the case with RGB, when KE made an error (see Table 6.6), he responded almost exclusively (86% or 147/171) with a word that was semantically related to the target.

On the basis of these data, one might conclude that the two patients had similar impairments. Consequently, given that earlier in this chapter for RGB the deficit was attributed to the phonological output lexicon, we might be tempted to conclude that KE also suffered from an impairment to the same functional component of the lexical system. However, recall that our model of lexical processing makes a number of predictions regarding performance on other tasks given a hypothesized impairment in the POL. Specifically, we should observe relatively good performance and an absence of semantic errors on all tasks that do not involve the POL. On the basis of the data presented thus far, however, we are not in a position to determine whether KE's performance is consistent with these predictions.

Table 6.7 presents the results obtained by RGB and KE on a number of the critical tasks. RGB, as seen earlier, was relatively unimpaired in tasks not involving the POL. KE, on the other hand, showed comparable semantic error rates on all of the tasks. A possible conclusion is that KE had a number of deficits in addition to a deficit to the POL similar to the one exhibited by RGB. However, if we return to our model of lexical processing we find that the one component of the system that is involved in all tasks of lexical processing is the semantic system. Given its central, mediating role between input and output systems, we would expect that damage to such a system would have comparable impact on all tasks that involve lexical processing regardless of input/output or orthographic/phonological status. This is precisely what we observe in the case of KE—he made semantic errors in all production tasks as well as in comprehension tasks where he was only required to say whether a word and picture matched (e.g., whether the word /lajən/ and the picture of a tiger corresponded to the same item; Table 6.7). These results yield the conclusion that KE, unlike RGB, had a deficit to the semantic system.

Given that the deficit for RGB presumably involved an output lexicon, whereas for KE it did not, we would expect that for RGB, but not for KE, performance should have been affected by the frequency of occurrence of the words that served as stimuli. The data support this prediction. RGB showed a significant frequency effect such that in reading, low-frequency words resulted in significantly fewer correct responses (60% correct) than did high-frequency words (71% correct; $\chi^2 = 4.68$; $p < .03$). KE's perfor-

TABLE 6.7
Semantic Error Rate (Semantic Errors/Total Errors) for KE and RGB on Six Tasks

Task	RGB	KE
Oral reading	98% (59/60)	82% (116/142)
Oral naming: pictures	98% (62/63)	85% (126/149)
Oral naming: tactile	100% (17/17)	95% (21/22)
Written naming: pictures	0% (0/12)	71% (107/150)
Writing to dictation	0% (0/12)	60% (84/139)
Matching		
Auditory word/picture	— (0/0)	96% (77/80)
Written word/picture	— (0/0)	79% (54/68)

mance did not appear to be affected by this parameter; an equal number of low- and high-frequency words were read accurately.

An interesting question is raised by these results: Why should the same type of error, a semantic error, result from damage to different components—the POL and the semantic system? Damage to the semantic component may take different forms. It is believed that in some cases the semantic representations can be damaged in such a way that it is difficult to distinguish between items that are similar to one another. For example, it is probably the case that APPLE [fruit, edible, round, sweet, smooth skinned, red, yellow, green] and PEACH [fruit, edible, round, sweet, fuzzy skinned, yellow] have more shared semantic features than they have features that differentiate one concept from the other. A loss of some of the differentiating features would render the two items almost indistinguishable. The patient should, as a consequence of such damage, not only have difficulty in discriminating between the two in comprehension tasks, but also in producing the correct name in a naming task (Caramazza & Berndt, 1978; Lesser, 1978).

Damage to either of the output lexicons, on the other hand, should not affect a patient's ability to understand the meaning of words, and good comprehension should be observed. In the case of damage to one of the output components, semantic errors are explained by assuming that, in a naming task, the presentation of, for example, a peach necessarily results in the activation of some of the features of semantically related items (e.g., apple). If the semantic representation of peach does not result in the activation of the damaged output form /pitʃ/ in the POL, another, accessible phonological representation that is normally activated by the semantic representation because it shares semantic features with peach (e.g., /æpl/), could be produced instead (Caramazza & Hillis, 1990a; Gordon,

Goodman-Schulman, & Caramazza, 1986; Nolan & Caramazza, 1982; K. Patterson, 1978).

In summary, we have described the performance of two patients with basically very similar errors in picture and tactile naming. By considering the patients' performance in the context of a model of lexical processing, we have shown, however, that these patients have deficits affecting different lexical components. This example illustrates that questions such as the following serve only to create confusion: What is *the* source of semantic errors in aphasic patients? It is apparent that there is no reason to assume that patients who make similar errors have similar deficits, or that damage to different underlying components must necessarily result in different error types.[2]

Semantic errors have also played a prominent role in the discussion of a syndrome observed in reading and referred to as DEEP DYSLEXIA (Marshall & Newcombe, 1966; see also papers in Coltheart, Patterson, & Marshall, 1980). Deep dyslexia is typically described in the following manner (Morton & Patterson, 1980): (a) semantic (e.g., *table* for *chair*), derivational (e.g., *walked* for *walking*), and visual paralexias (e.g., *chair* for *chain*) in reading single words aloud, also omissions; (b) severe if not total impairment in reading nonsense words; (c) part-of-speech effects in word reading, with nouns read better than adjectives, which, in turn, are read better than verbs or function words; and (d) abstractness effects in word reading such that imageable, concrete words are read better than abstract words. These symptoms are considered to represent a category of disorder or a syndrome because a number of patients have been observed in whom these symptoms co-occur; the semantic error is believed to be the central or defining feature of the syndrome.

The fact that a quite specific constellation of behaviors should be observed across a number of patients is commonly taken as an indication that the observed performance pattern results from damage to a single mechanism. Consequently, reports of syndromes typically lead to attempts to determine the underlying mechanism. The example of deep dyslexia allows us to explore the notion that the observation of symptom co-occurrence necessarily reflects damage to a single functional component.

2. We have not, of course, described a number of other patterns of impaired performance that can be observed in naming tasks. Clearly, however, if the theory of lexical processing is correct, these too should be able to be explained as the result of damage to one or more of the underlying components. For example, a commonly observed behavior occurs when a patient is apparently unable to produce any response when asked to provide the name of an item (often referred to as anomia). Within this model, such responses can be considered to occur when the POL is damaged to such a large extent that neither the correct response nor any semantically similar responses become active enough to serve as the basis of a spoken response.

We can turn to our lexical processing model to determine the location of a functional deficit that would result in only the constellation of symptoms that define deep dyslexia. An impairment to nonword reading suggests a deficit to GPC mechanisms, but such damage does not predict the observed pattern of performance with words. Although the production of semantic, derivational, and visual paralexias, as well as word class and abstractness/concreteness effects, might be consistent with a semantic deficit, many of these patients show good comprehension for many of the words that result in semantic errors—and certainly the nonword difficulties cannot be explained by a semantic deficit. There is no single lexical component that when damaged would produce this performance pattern. In fact, no one has yet been successful in accounting for the syndrome in terms of a single underlying deficit.[3]

In addition, if one claims that this constellation of symptoms can result *only* from damage to a single component, then the expectation is that the occurrence of one of the symptoms should be predictive of the other symptoms. This certainly does not hold for deep dyslexia. For each of the symptoms of deep dyslexia, patients have been described who exhibit one or more of the symptoms but no others. For example, of the symptoms of deep dyslexia, RGB (Caramazza & Hillis, in press) and KE (Hillis et al., 1990) exhibited only semantic errors and difficulty in nonword reading; patients LB (Caramazza, Miceli, Silveri, & Laudanna, 1986) and WB (Funnell, 1983) had difficulty only with nonwords. These findings are consistent with the conclusion, at least within the lexical processing framework we have described, that deep dyslexia can only be understood as the result of multiple deficits (Nolan & Caramazza, 1982; Shallice & Warrington, 1975).

We have seen, therefore, that the co-occurrence of symptoms does not necessarily implicate a common deficit to the functional architecture of a cognitive system. Nonetheless, the question remains: Why should this coincidence of symptoms be repeatedly observed? There are two possible answers. (a) The symptoms may reflect damage to a number of different mechanisms that are anatomically adjacent in the brain. In this view, damage to a particular brain region, unless very selective, may be expected to affect a number of functional components. (b) The second possibility is that the theory we are using as the basis for the interpretation of patient performance is wrong. This is entirely possible, of course, but if a new theory

3. At a neuroanatomical level an explanation has been proposed that is referred to as the Right Hemisphere Hypothesis. This suggestion, however, has been widely criticized (for a review of the RHH, see Coltheart et al., 1980; K. Patterson & Besner, 1984; Saffran, Bogyo, Schwartz, & Marin, 1980; Zaidel & Schweiger, 1984; Rabinowicz & Moscovitch, 1984).

is developed that predicts that this particular constellation of symptoms should result from damage to a single underlying mechanism, the theory must also be able to account for all of the other patterns of observed impairment that are accounted for by the current theory. This has not yet been done for the case of deep dyslexia.

Just as the co-occurrence or association of symptoms need not be an indication of a common functional deficit, the dissociation of performance on different tasks does not necessarily indicate that different components are required to perform the various tasks. An example of a dissociation leading to the claim of multiple components has resulted from the observation of what has been termed modality-specific aphasia. Patients have been described who demonstrate an inability to name objects presented in one modality, in the face of correct naming of the items in other modalities. Thus, for example, Lhermitte and Beauvois (1973) described the patient JF who made numerous errors (primarily semantic) in naming objects and pictures whose function he was able to mime, but made many fewer errors in naming objects or pictures upon presentation of their characteristic sound, their definition, or their tactile presentation (for other relevant cases, see Beauvois, 1982; Beauvois, Saillant, Meininger, & Lhermitte, 1978; Denes & Semenza, 1975; A. Hillis, 1988; Riddoch & Humphreys, 1987; Silveri & Gainotti, 1988). This pattern of performance involves a possible paradox: Adequate miming of the function of visually presented objects, as well as good naming to definition, sound, or tactile input, appears to require an intact semantic system, whereas the presence of semantic errors in object and picture naming suggests a damaged semantic system. One means of reconciling this discrepancy is to suggest that there are multiple semantic systems, and that, in the case of JF, the connection between the system of "visual" semantics and the system of "verbal semantics" required for correct naming was damaged (see Shallice, 1987, for discussion). This would result in poor naming in spite of adequate comprehension (as demonstrated by miming) of visually presented items, as well as in good naming and comprehension in all other modalities.

The claim of modality-specific semantic systems, therefore, raises the possibility that the specific functional architecture with which we are working, and which specifies a single amodal semantic system, might be incorrect. Specifically, it has been suggested that in contrast to the single semantic system represented in Figure 6.2, there are numerous semantic systems differentiated according to modality (Beauvois et al., 1978; Lhermitte & Beauvois, 1973; Shallice, 1987, 1988; Warrington & Shallice, 1984). Thus, there would be a semantic system for visual information that is addressed when a person is presented with an object or picture, a verbal semantic system to represent the meaning of words, perhaps an auditory sys-

tem to represent the meaning of sounds, and so forth (for a discussion of the different things that might be meant by the term modality specific semantics, see Caramazza, Hillis, Rapp, & Romani, 1990b; Hillis et al., 1990; Riddoch, Humphreys, Coltheart, & Funnel, 1988).

However, Caramazza et al. (1990b) and Hillis et al. (1990) have argued that the data regarding modality-specific aphasia do not require such conclusions, because they can naturally be accounted for within a theory that posits a single central semantic component. This is done in the case of JF by showing how damage to those processes that address the semantic system from an object recognition component (Figure 6.2) could result in only the observed performance pattern. It has been argued that such damage could often result in the activation of semantic representations that would be inadequate for naming although sufficient for correct miming. It was further pointed out by these authors that proponents of modality-specific semantics have failed to articulate the content of the various semantic systems: What is represented in visual semantics? How is it similar to or different from the information represented in the other semantic systems? For example, how is the meaning of DOG different in a system of visual versus verbal semantics?

It is not enough, therefore, to observe a dissociation in order to conclude that the dissociation must reflect a distinction among underlying functional components; just as in the case of deep dyslexia, it was not enough to observe an association in order to conclude that a common component must be implicated. A common component can be inferred if, within some theory, there is a component whose function is such that, when damaged, the observed association should occur. Likewise, a distinction among components will be motivated only if, within some theory, a pattern of performance cannot be accounted for without postulating additional components. Thus, although the functional architecture of the lexical processing system proposed in this chapter may very well be incorrect, it has not been shown to be incorrect with these examples.

MORPHOLOGICAL ERRORS

We have seen, in previous sections, that similarity of performance levels and error types does not guarantee similarity of underlying impairment. Nonetheless, it is common practice to group patients by error type, assign a name to this grouping, and assume that patients categorized in this manner form a homogenous group that will be similar in all other relevant respects. A common example of such a practice is the case of the classification of AGRAMMATISM (Berndt & Caramazza, 1980; Kean, 1985). Patients are typically classified as agrammatic according to the following characteristics of speech production: omission and/or substitution of func-

tion words and inflections and reduced phrase length. (Some would include as part of the classification criteria the symptom of "asyntactic" comprehension; see Zurif, Gardner, & Brownell, 1989; but, see also Berndt, 1987; Caramazza & Badecker, 1989, for a critical evaluation of this position.) Patients categorized according to these criteria are then treated as a group (e.g., common rehabilitation programs are applied, test results are averaged across patients, etc.). Note that these practices are necessarily based on the assumption that patients similarly classified are fundamentally homogeneous with regard to the nature of their underlying deficit; otherwise, why average across their performance and apply common treatment programs?

However, even a very simplistic consideration of what might be involved in producing correct sentences might distinguish between mechanisms that determine an appropriate syntactic structure for the sentence to be uttered and mechanisms that select and represent the individual words to be used. A deficit to either of these levels of processing would be expected to result in an impairment in sentence production. We examine this possibility by considering the performance of two patients who, according to the preceding criteria, are agrammatic. They will prove, however, to have fundamentally different underlying deficits.

Table 6.8 contains samples of spontaneous speech of both patients, with translations provided for FS (Miceli & Caramazza, 1988), an Italian patient. Both patients omitted function words; FS in particular made numerous inflectional substitution errors (e.g., *leggeva* [he was reading] → *leggere* [to read]). If a lexical deficit involving an output component were the source of the observed omissions and substitutions, we would expect errors in other tasks involving that lexical component.

The performance patterns of the two patients can be directly compared on their ability to repeat single words: prefixed, suffixed, and function words (see Table 6.9). ML (Caramazza & Hillis, 1989a) made no errors on this task, whereas FS was similarly impaired across all word types. In addition, ML did not make errors of the sort observed in her spontaneous speech in any other task involving single word production: reading, repetition, or writing. This indicated that ML did not have a lexical deficit affecting function words and morphological affixes. In fact, the conclusion was reached that ML's deficit in sentence production could be attributed to an impairment at the level of the sentence planning mechanisms that specify the morphemes and function words to be selected from the output lexicons for production.

For FS, on the other hand, an analysis of the error types indicated a close resemblance between the errors observed in sentence production and those observed in repetition of single words. Consequently, in the case of

TABLE 6.8
Spontaneous Speech Samples and Error Rates for ML and FS

ML

Cleaning . . . Annie Thompson . . . Boston . . . got up four dollars . . .
taken for little children . . . eaten . . . not eaten three days . . .
touched by the story . . . made up purse her . . . for her

couple having picnic . . . boy kite flying . . . playing in the water . . .
a sailboat, flagpole, dog . . . the boat is down water . . . dog watch boy

FS

Then returns my house — Then I listen to the television
Poi ritorna la mia casa — *Poi io ascolto il televisione*

or then make lunch because, dear doctor, I live alone!
o poi fare il pranzo perche', caro dottore, vive solo!

then I telephone, receives, make because the days long
Poi telefono, riceve, fare, perche' il giornate lungo

	ML	FS
Omission of function words	62% (108/173)	22% (54/242)
Function word substitution	2% (4/173)	20% (48/242)
Inflection/derivation errors	15% (5/33)	27% (75/275)

FS, the role of a lexical deficit in sentence production could not be ruled out. We see once again that patients with apparently similar performance patterns may differ in terms of their underlying impairment: FS with a lexical deficit affecting sentence production, and ML with apparently no deficit at the level of single words.

A more detailed analysis of the errors produced by FS in repetition of single words revealed aspects of the internal structure of the POL. It was noticed that the majority of the errors were morphological (84.5%). Most of the morphologically based errors consisted of correct repetition of the stem of the stimulus word and the substitution of the affix (e.g., *vestire* [to wear] → *vestivi* [you were wearing]). Only 15.5% of the errors were phone-

TABLE 6.9
Accuracy of Repetition Performance for ML and FS with Prefixed, Suffixed, and Function Words

	ML	FS
Function words	100%	31%
Prefixed words	100%	48%
Suffixed words	100%	33%

mic paraphasias (e.g., *pagata* [paid] → *pagara* [nonword]). This distribution of errors suggests that although the patient had some impairment affecting the selection and/or the production of individual sounds (as evidenced by the phonemic paraphasias), such a deficit cannot account for the massive presence of morphological errors. It would appear, instead, that a deficit specifically affecting morphological structure played a major role in this patient's impairment.

Additionally, a striking feature of these morphologically based errors is that they were essentially all (615/637 or 96.7%) inflectional errors. This result suggested a selective deficit to mechanisms in the lexicon dedicated to inflectional processing. Comparable proportions of errors were made on derived and inflected words: 33.4% accuracy for each type in a specially designed list of derived and inflected words matched for length and frequency. Nonetheless, *errors* for both inflected and derived words consisted predominantly of inflectional errors. For example, the inflected word *leggeva* (he was reading) → *leggere* (to read) and the derived word *regionale* (regional, singular) → *regionali* (regional, plural).

Within the model of lexical processing proposed here, the deficit suffered by FS can best be described as an impairment to those processes that select specific affixes within the inflectional component of the lexicon. That is, the stems or root forms and derivational affixes are correctly selected, whereas inflectional affixes are often misselected. Such a deficit should give rise to the frequent inflectional substitutions observed in the course of sentence production and repetition.

This example illustrates the difficulties involved developing legitimate categories of impairment (see Badecker & Caramazza, 1985; Caramazza, 1984, 1986; Ellis, 1987; McCloskey & Caramazza, 1988, for detailed discussions of problems of classification and categorization, but see also Caplan, 1986; Shallice, 1988; Zurif et al., 1989). The classification of a patient is based on the assumption that similar performance on a particular task or tasks—sentence production in this case—is the result of a similar deficit or deficits. Clearly then, classification is only warranted (a) to the extent to which one can motivate the assumption of similarity of underlying deficits, or (b) to the extent to which it would not matter that patients similarly classified might differ in this fundamental respect.

Important in a consideration of (a) is the nature of neurological impairment. The extent and location of impairment will differ from patient to patient. Because of this, different cognitive functions may be affected in patients who otherwise share certain deficits. Thus, although two patients may have damage to the same underlying component, one of the two patients may have damage to other components as well. Thus, similar performance on tasks 1 to 5—suggesting an impairment to mechanism X—

does not guarantee similar performance on tasks 6 to 10. If performance on tasks 1 to 5 is the classification or grouping criterion, then very heterogeneous groups may result. Within the common practices of research or treatment, group heterogeneity is extremely problematic. One cannot expect a positive outcome if patients grouped according to the criterion of agrammatism, for example, receive a common treatment. Similarly, research attempts to further our understanding of the cognitive system and its impairments will yield incorrect conclusions if such patients are grouped and conclusions are drawn based on average group results. (For data and discussion of the heterogeneity of performance in agrammatic production see Miceli, Silveri, Romani, & Caramazza, 1989, and Saffran, Berndt, & Schwartz, 1989.)

Although there may be purposes for which the heterogeneity of the group with respect to underlying dysfunction is unimportant, it is not obvious what these might be.

In this section, by examining two cases of apparently "agrammatic" aphasia, we have (a) illustrated the difficulties involved in classifying and grouping patients; and (b) found evidence in support of the notion that certain sentence production deficits can be understood, at least in part, as deficits to the morphological aspects of lexical processing, whereas others are best understood as impairments to processes specific to sentence planning.

Conclusion

With these examples, we hoped to show how patterns of impaired performance cannot be understood simply by consideration of performance levels, error types, or the observation of dissociations or associations of symptoms. These overt manifestations of damage to the functional components of processing can be interpreted only within a theoretical framework that provides a basis for their interpretation. A detailed description of the possible underlying mechanisms and their functions is clearly necessary.

The level of detail at which theories need to be specified will become apparent when we evaluate the issue of whether or not impaired performance can be considered to result from damage to a representation itself, resulting in loss of the representation, or, alternatively, from damage to those mechanisms that access the representation, making the representation difficult to recover. Shallice (1987) discusses this issue and proposes a number of criteria which, he argues, can be used to distinguish deficits of storage from deficits of access. One criterion mentioned is consistency.

Shallice argues that loss of a representation should result in the consistent inability to identify a given item across test sessions, whereas an access difficulty would result in inconsistent performance across repeated testing. We would argue that this issue, like all others raised in this chapter, cannot be decided without reference to a specific theory of lexical processing at the level of interest—in this case, a description of the structure of representations and access mechanisms is required.

We have shown earlier that without making reference to a theory of the possible processing components and their interrelationships, it could not be determined whether a particular pattern of performance, for example, the production of semantic errors, has only a single possible source. Likewise, it will be impossible without making reference to a description of mechanisms of access and storage to determine whether a particular pattern, in this case inconsistent performance, can result only from damage to an access mechanism, whereas consistent performance results only from damage to a stored representation.

For example, one might imagine that the mechanism that accesses the POL from the semantic system works in the following manner. A semantic representation consisting of a set of features, such as <animal, mammal, domestic, four legged, hairy, barks, canine>, serves as input to the POL. Phonological representations in the POL are activated by the semantic input in proportion to the degree to which they share semantic features in their access specification. In this example, the phonological units /dag/, /bigl/, /kæt/, /lajən/ and so forth, are all activated to some degree, and the representation that receives the most activation, /dag/, may reach threshold for output and subsequent production. As discussed in an earlier section of this chapter, the ease (measured by accuracy or speed) with which a lexical representation reaches threshold is a function of at least two parameters: (a) the degree of fit between the input and the access code for a representation, and (b) the activation threshold of the representation (as indexed by frequency of usage), so that in the case where two or more phonological representations are equally activated by a semantic input, the lexical entry with the lowest threshold (most frequent) will be the one to be produced as a response (Caramazza & Hillis, 1990a).

Let us now consider whether the consistency criterion proposed by Shallice distinguishes between access and storage deficits for this type of model of lexical access and representation. What are the expectations of damage to the access procedure? It depends on the assumptions we are willing to make about the consequences of damage to the hypothesized access procedure. Thus, for example, we might imagine that *one* consequence of brain damage is that semantic representations would activate phonological representations to a lesser extent than normal. In that case, the

phonological representation for the correct response would still receive the most activation, but not necessarily enough to reach threshold. And, if there is a semantically related lexical representation with a lower threshold of activation, it could be the one to reach threshold and to be produced *consistently* as a response. In such a situation, one would observe consistently incorrect responses to the target.[4]

Consider now the case of damage to the stored semantic representations themselves. One type of damage could take the form of the "loss" of some of the features that comprise the meaning of words. One consequence of this situation is that the semantic representation computed for an object would now be underspecified in such a way that it activates equally a number of phonological representations in the POL, including the correct one. For example, considering only the semantic category of animal terms, if damage were to result in the loss of all features except <animal, domestic, and hairy>, the residual information would activate equally *cat, dog, Pekinese,* and so forth. Assuming that the semantic information computed under the hypothesized conditions of damage is too impoverished to activate any one phonological representation to threshold, a response may be selected at random from the set of phonological representations that have the highest levels of activation, leading to inconsistent errors and error responses and, by chance, to correct responses as well.

Thus it is possible to articulate plausible models of lexical processing, which, when damaged at the level of access procedures or stored representations, lead to expectations about the consistency of performance that are the opposite to those derived by Shallice. Obviously, then, it is not possible to say, independently of a specific theory of lexical processing, that one particular pattern of consistency in performance signals a deficit to access mechanisms and another pattern of consistency of performance signals a deficit to stored representations. In other words, it is not possible to establish general criteria for determining when representations and access mechanisms are impaired without a description of the structure and function of the relevant mechanisms. More importantly for present purposes, we see that the kinds of expectations that we may derive from a model depend crucially on increasingly detailed claims about the processing structure of the components that comprise a particular functional architecture.

We hope that we have shown how a theory is an enormously powerful and useful tool for the interpretation of human behavior—normal or impaired. Furthermore, we hope that it has become apparent that it is not the

4. In the particular instantiation considered here, whether or not an item is consistently misnamed and whether or not a consistent error response is produced depends entirely on the distribution of thresholds of semantically related cohorts.

case that the need for theory can be dispensed with or left unused in our tool chest. All interpretation is based on some theory we have of cognitive structure; the theories simply differ in terms of the degree to which we are aware of them and use them, and in terms of the level of detail at which they are described. If this is the case, then the progress we make in understanding language and cognitive disorders will depend to a large extent on our willingness and ability to develop theories that are both explicit and detailed.

Acknowledgments

The work on this chapter was supported by National Institutes of Health Grants NS22201 and 23836 to The Johns Hopkins University. We thank Lisa Benzing, Argye Hillis, and Michael McCloskey for their helpful comments.

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7

Sentence Processing in Aphasia

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Almost 20 years have passed since the publication of the first edition of *Acquired Aphasia.* Those years have witnessed sustained interest in research directed at aphasic impairments that involve the comprehension and production of sentences. This research has been carried out from a number of different perspectives and has focused on a variety of different types of impairments to sentence processing. This updated version of "Sentence Processing in Aphasia" summarizes some of the earlier trends and developments and incorporates recent findings.

When the first edition of *Acquired Aphasia* was published, there was considerable optimism that studies of aphasia could provide important information about how sentence processing is normally accomplished. In a chapter of that volume entitled "Syntactic Aspects of Aphasia," Berndt and Caramazza reviewed the hypothesis that a form of aphasia existed (AGRAMMATISM) with a set of symptoms that reflected a central syntactic impairment affecting performance in all tasks that require syntactic processing, that is, in the production and comprehension of written or spoken sentences. Had this thesis been upheld, it would have meant that a single set of syntactic representations is exploited in comprehension and production, and that this set of representations is selectively vulnerable to brain damage. Moreover, such selective impairment of syntactic capacities in agrammatism would have provided an extraordinary opportunity for the investigation of other aspects of language (phonology, semantics, pragmatics) in relative isolation from syntax.

This strong version of the "syntactic deficit hypothesis" was not supported by subsequent investigations of individual cases. These studies, reviewed in detail in the second edition of *Acquired Aphasia,* uncovered mul-

tiple dissociations among the set of symptoms that had been argued to arise from a single, syntactic deficit. Three types of findings were reviewed in the second edition that questioned the appropriateness of the focus on "agrammatism," which had dominated research on aphasic sentence processing up to that time. Subsequent research has supported the view that exclusive focus on "agrammatism" provides only a narrow window into the many forms that sentence processing impairments can take.

One of the primary reasons that agrammatism has been viewed as a coherent, homogeneous entity is the existence of a contrasting grammatical disturbance labeled "paragrammatism" (see Goodglass, 1993, chaps. 5 and 6, for definitions and samples of speech characteristics). In many ways, prototypical paragrammatism was argued to constitute the mirror image of agrammatism: paragrammatic sentences are produced fluently (sometimes hyperfluently) and contain many grammatical morphemes (inflections and free-standing function words), though these may be used inappropriately. Agrammatic speakers, in contrast, labor to produce halting and fragmented sentences that tend to contain content words without the support of grammatical morphemes. As reviewed in the previous edition, careful study of individual patients blurred the distinction between agrammatism and paragrammatism by demonstrating a number of similarities in the kinds of difficulties both types of patients demonstrate when producing grammatical morphemes. Contemporary work has shown that when differences in fluency are ignored (these differences are, after all, not necessary to the syntactic arguments), the sentence production patterns of the two types of patients show considerable overlap (e.g., Bird & Franklin, 1995–1996). It has even been proposed that the differences between the two patterns of sentence production reflect different strategic responses to what is essentially the same underlying deficit (Kolk & Heeschen, 1992). There is reason to believe, therefore, that comprehensive research on sentence processing in aphasia will need to encompass production patterns in addition to the "agrammatic" pattern.

Another reason that agrammatism was the focus of much early work on sentence processing was that there appeared to be overlap between the sentence production pattern of agrammatism and a type of comprehension impairment demonstrated by agrammatic patients. This comprehension pattern is one for which special tests are designed that require the patient to understand the syntactic structure of the sentence in addition to the meaning of the words it contains. For example, the meanings of active and passive voice sentences with two nouns that can play the role of "agent" of the action ("the boy is kissing the girl"; "the boy is kissed by the girl") can be distinguished only if the morphology and word order signaling sentence voice are appropriately interpreted. Because morphology and word

order are two elements that are argued to be disrupted in agrammatic *production* (cf. Saffran, Schwartz, & Marin, 1980), the fact that such patients were shown to have difficulty with these special comprehension tests (i.e., to choose between a picture of a boy kissing or a girl kissing in the preceding example) was interpreted as evidence that a single impairment was responsible for both types of symptoms.

As detailed in this chapter in the second edition, patients were described who demonstrated the agrammatic pattern of omission of grammatical morphemes in production who also demonstrated normal comprehension. This finding indicates that the co-occurrence of these two symptoms is not a necessary one. A recent meta-analysis of all published studies of agrammatic patients' comprehension of active and passive sentences showed that normal performance with these sentence types is relatively common (Berndt, Mitchum, & Haendiges, 1996). The analysis was done on all studies of agrammatic comprehension published between 1980 and 1993 using a sentence–picture matching task such as the one described here. A total of 64 data sets (i.e., separate patient testings) from 15 published studies found that about one-third of the patients performed the task with above-chance accuracy; many performed at ceiling levels. It is clear from these results that the omission of grammatical morphemes in spontaneous speech (i.e., the agrammatic pattern) is not necessarily accompanied by an active–passive sentence comprehension impairment. The reverse is also true: many nonagrammatic patients have been described who demonstrate a comprehension pattern that is indistinguishable from the "agrammatic" pattern (e.g., Berndt, Haendiges, Mitchum, & Sandson, 1997; Saffran, Schwartz, & Linebarger, 1998). Clearly, study of this sentence comprehension pattern and its underlying causes need not be limited to patients with agrammatic speech.

A third problem that mitigates against exclusive focus on agrammatism is that the characteristics that were often assumed to define the agrammatic pattern do not provide an accurate description of the sentences actually produced by most patients who are given that classification. In addition to the many dissociations reviewed in the previous edition, recent work has highlighted dissociations between grammatical morpheme omission and other sentence structural abnormalities (Nadeau & Gonzalez-Rothi, 1992), between bound and free-standing grammatical morphemes (Bird & Franklin, 1995–1996; Saffran, Berndt, & Schwartz, 1989), and among specific elements within the classes of free-standing and bound grammatical morphemes (Friedmann & Grodzinsky, 1997). In light of this complex set of patterns, it is unlikely that a single account of agrammatism can accommodate all possibilities (but see discussion of symptom variability in Kolk & Heeschen, 1992, discussed later). In fact, the variety of possible ways in

which "agrammatism" can be manifested produced a spirited debate about the validity of agrammatism as a unitary concept (reviewed in the previous edition). Although interest in agrammatism is still high, especially among linguists (see special issues of *Brain & Language, vol. 50,* nos. 1, 2, and 3, 1995), there is widespread concern about the issues of variability and heterogeneity that have been raised (see, e.g., Caplan, 1995). Consequently, even in studies focused exclusively on agrammatism, there has been a discernible increase in attempts to provide a more complete description of patients' deficits than is implied by their designation as agrammatic. Most importantly, such information now frequently includes demonstrating that the patient actually exhibits the particular symptom of interest (e.g., a specific pattern of comprehension impairment) rather than relying on an assumption that the symptom must be there because the patient's speech is "agrammatic."

In addition to this continuing evolution in thinking about the types of patients who should be studied in sentence processing research in aphasia, two other general trends can be detected. In both cases, the topics of interest have been accompanied by, and are perhaps driven by, new research methodologies that are suited to their study.

Variability (and Malleability) of Patients' Symptoms

Symptom variability across similar patients, as described earlier, may appear to be one of those unfortunate facts that only complicates the life of the researcher. Although there is little doubt that variability across patients complicates attempts to provide general, testable hypotheses about the nature of sentence processing impairments, the variability itself also constitutes a factor that must be accommodated in any account of sentence processing impairments. That is, it is ultimately of great interest to ask why sentence-relevant symptoms can be manifested in such a variety of ways in different patients, and in different ways in the same patient if task demands change (Cupples & Inglis, 1993). Sentence production can show markedly different characteristics depending on whether the patient is speaking spontaneously, describing a picture, using a provided word in a sentence, or performing a more constrained task such as sentence completion (Berndt, Haendiges et al., 1997; Hofstede & Kolk, 1994; Kolk & Heeschen, 1992).

Kolk and colleagues have developed a model of sentence processing in aphasia that readily accommodates (even predicts) variability of symptoms across patients and within patients across tasks (Hofstede & Kolk, 1994; Kolk & Heeschen, 1992). Within this framework, several possible

processing deficits are hypothesized that could undermine patients' attempts to comprehend and/or to produce sentences. With regard to sentence production, temporal limitations in the normal dynamic flow of information are argued to produce asynchronies in the coordination of lexical retrieval and syntactic elaboration. In response to these limitations, patients are hypothesized to limit the complexity of the messages that they intend to convey (Kolk, 1995). This adaptive strategy is argued to account for the "structural simplification" that is characteristic of the sentences produced by many types of aphasic patients and for the telegraphic character of agrammatic speech (Hofstede & Kolk, 1994).

For present purposes, the important aspect of Kolk's model involves patients' methods of adapting to these limitations. Faced with the likelihood of failing to articulate a well-formed sentence, for example, patients will adopt a number of positive and negative adaptations: they might choose simple sentence forms with predictable structures and/or they might omit inessential items. In fact, they might adopt any of the characteristics of normal ellipsis used in conversational speech, such as using the infinitival form of the verb. Kolk and Heeschen (1992) studied the conversational and elicited speech of Dutch- and German-speaking Broca's and Wernicke's aphasics and found that the Broca patients' spontaneous speech contained significantly more elliptical elements than the speech of the Wernicke patients. Furthermore, the use of elliptical features declined in a sentence elicitation task, and the Broca patients' sentences became similar to those of the Wernicke patients in that they contained more substitutions and fewer omissions.

In an attempt to replicate these results with English-speaking patients, Hesketh and Bishop (1996) studied the productions of 14 agrammatic speakers, 11 nonagrammatic Broca's aphasics, 5 fluent aphasics, and 10 control subjects using somewhat different procedures and response measures from Kolk and Heeschen (1992). On tasks that should leave less room for adaptive behavior (sentence elicitation from an auditory model, written sentence ordering, and a cloze procedure), the Broca patients produced very few paragrammatic responses (in contrast to the results of Kolk and Heeschen) and continued to omit bound morphemes. Kolk and Heeschen (1996) have argued that differences in procedures undermined the possibility of a replication. Nonetheless, Hesketh and Bishop's results suggest that alternation between omitting and substituting among grammatical morphemes is not a pattern that is easily induced among aphasic patients. It is not yet clear under what conditions symptoms of adaptation might be elicited and precisely what factors are involved. The possibility also exists that languages differ in the number and utility of elliptical speech mechanisms, which could result in true cross-language differences.

Another aspect of Kolk's model that is still unclear is the extent to which

patients' adaptations, when they do occur, are under conscious control. This issue was addressed directly by Bastiaanse (1995), who used adaptation theory to explain a patient's apparent spontaneous shift during narrative speech from an almost normal pattern (with mild syntactic anomaly) to a grossly agrammatic pattern characterized by quite severe morphological as well as syntactic aberrations. When questioned about her variable speech pattern, the patient demonstrated clear knowledge that her production was aberrant; furthermore, she explicitly denied adopting any purposeful pattern of leaving out words on some occasions. In contrast to this apparent evidence that adaptation is not under conscious control, Kolk and Hofstede (1994) elicited marked changes in the form of a patient's productions by increasing the formality of the testing situation and by simply asking the patient to speak in complete sentences. Thus, although at least some patients can consciously control the general form that their impaired productions take, this control does not appear to be universal.

The apparent malleability of sentence processing symptoms has been exploited in a number of recent studies seeking to effect change in patients' sentence processing symptoms. The "targeted treatment study," discussed at the end of this chapter, may be regarded as a new methodological approach to the investigation of aphasic disorders (see Mitchum & Berndt, 1995, for arguments), which relies on some malleability of patients' symptoms.

Integrating Lexical, Semantic, and Syntactic Information in Sentence Processing

The other general trend that has emerged from recent work on sentence processing concerns the increased importance assumed by the issue of how various types of information are integrated over time during sentence processing. This emphasis on information coordination appears to have a number of contributing sources. The first involves proposed changes to the normal psycholinguistic models that have largely guided research on aphasic sentence processing. Strictly serial models of production, in which information feeds from level to level in a single direction, have been challenged by data that argue for more widespread interaction among processing components (e.g., Dell, 1986). By the same token, in contrast to models of sentence comprehension in which syntactic parsing precedes any semantic interpretation (Frazier, 1990), newer studies have demonstrated very early effects of semantic information on the extraction of a syntactic representation from the auditory signal (e.g., Tabossi, Spivey-Knowlton, McRae, & Tanenhaus, 1994).

Another factor that has contributed to an interest in the time-course of information availability is the development of methodologies that have only recently been applied to analyses of aphasia. Computational models of language have been designed to allow systematic variation in the dynamic flow of different sorts of information through networks that simulate language processing components. The emergence of computational models as a research tool provides the opportunity for testing hypotheses about pathologically induced disruption of the normal time-course of information availability (and maintenance) during sentence processing (Cornell, 1995; Haarmann & Kolk, 1991a). A study by Dell and colleagues simulating aphasic naming errors illustrates the potential usefulness of this methodology (Dell, Schwartz, Martin, Saffran, & Gagnon, 1997). By using an interactive activation model with a number of parameters set to fit normal error patterns, these investigators simulated error data from 21 fluent aphasic patients' naming responses by successively "lesioning" the model's connection weights, decay rates, or both. Once the individual patient data were fit to a "lesioned" model, predictions were made and confirmed for individual patients concerning a number of aspects of performance, including recovery patterns. Although this model has not yet been directly applied to *sentence* production in aphasia, its application to naming errors suggests that the control it affords over the temporal dynamics of a number of separate processing parameters will prove useful in investigating perturbations of processing in aphasic sentence production.

Another methodological technique that has been increasingly applied to the investigation of aphasic deficits is the direct study of sentence processing as it unfolds over time (Friederici & Kilborn, 1989; Zurif, Swinney, Prather, Solomon, & Bushell (1993). These "on-line" studies have been applied primarily to issues of sentence comprehension; they test hypotheses proposing disruption of the ability to integrate different types of information in real time. Some of these studies are reviewed later.

Quite aside from these methodological innovations, the issue of how lexical, semantic, and syntactic information is integrated has emerged as an important issue because of evidence that many patients experience selective difficulty processing words in different grammatical categories (e.g., nouns vs. verbs; Berndt, Mitchum, Haendiges, & Sandson, 1997; Caramazza & Hillis, 1991). A failure to gain access to words within a specific grammatical class could cause major disruption of information integration. The challenge taken up in recent research is to elucidate the relationship between such category-specific impairments and symptoms involving sentence processing. That is, depending on the model of normal sentence processing that is adopted, selective difficulty retrieving words from particular grammatical classes might be viewed either as a *cause* or

an *effect* of sentence processing failure. It should be clear that solving this "chicken and egg" question could have important implications for developing treatments to improve the comprehension and production of sentences.

Sentence Production: Conceptions of Normal Production

The problem that must be explained by models of normal sentence production is formidable: How does the human speaker constrain what may be a complex, multidimensional conceptual "message" into a linear string of correctly chosen words that conveys to a listener the basic information about who does what to whom, as well as other information such as when, in what manner, and why? For English speakers, the linguistic devices that must be considered in structuring an utterance to convey such information include the order in which words are produced and their coordination with a relatively small number of bound and free-standing grammatical morphemes (e.g., verb inflections, auxiliary verbs, determiners, etc.). As noted earlier, one critical question concerns how lexical information is coordinated with these different structural devices during the act of speaking, an issue that critically depends on the order in which different types of information becomes available.

The models of normal sentence production that have dominated attempts to explain sentences produced by aphasic patients were built primarily on a database of slips-of-the-tongue produced by normal speakers (Dell, 1986; Garrett, 1980; Levelt, 1989). Regularities in the occurrence of particular types of errors suggest that there are distinguishable levels in the structural formulation of a sentence. The primary distinction among these models concerns the flow of information between levels. Serial models of sentence production assume that processing at each level is influenced only by information represented directly above it; that is, information flows in only one direction (Bock & Levelt, 1994; Garrett, 1980; Levelt, 1989). Interactive models, in contrast, assume that information transmission through the system can be bidirectional, allowing for a wide range of possible mutual effects among levels (Dell, 1986). The issue of whether sentence production should be viewed as a strictly sequential or more interactive process has been addressed in a number of experiments with normal subjects, but the issue is still debated.

Despite this important difference in how these classes of models view information flow during sentence production, there are major similarities in the types of information postulated to be involved. Generally, all of these accounts assume that conceptual and discourse processes external to the

sentence production system generate a "message" that needs to be rendered into a sentence. The message activates meaning-based lexical representations (often called LEMMAS) as well as elements of meaning-based sentential representations such as thematic roles (agent, theme, etc.); information at this level is ultimately related to form-based lexical representations (LEXEMES) which are coordinated into the appropriate slots of a syntactic frame that includes the necessary grammatical morphology. These models thus postulate separate lexical representations for meaning and for form, and they assume that both types of information must be activated at different points during production. The serial models argue that meaning is activated first and drives the activation of lexical forms; interactive models postulate that semantic and phonological activation can be at least partially simultaneous, allowing interacting effects on word retrieval.

The basic framework of the production model to be used here draws primarily from Bock and Levelt's (1994) serial model. This model evolved from earlier work (Garrett, 1980; Levelt, 1989) and includes an explicit assumption that variations in the order in which information is delivered from one component to the next can affect the order in which elements appear in speech. This assumption is clearly relevant to an analysis of aphasic sentences, as it has been argued that lexical accessibility may be degraded in aphasia in ways that have predictable effects on sentence production.

The most interesting aspect of the model for present purposes includes two processing levels involved in GRAMMATICAL ENCODING, that is, the processes that are necessary to transform the "message" into a morpho–phono–syntactic representation that can drive phonetic encoding. The first of these levels (labeled functional processing) guides the coordination problem: based on conceptual information in the intended message, clause-size units of semantically specified lexical items (lemmas) are assigned roles in a predicate/argument-type structure. At this level only the semantic–syntactic identity of the words to be spoken is made available (not their pronunciations), and selected lemmas are marked in some fashion as to the roles they will play in the ultimate sentence (e.g., a noun might be marked as agent of the action or as logical subject of the predicate). This abstract, unordered representation of marked lexical information serves as input to the next, positional processing, level. Here, phrase-size units of ordered words are created through the insertion of phonologically specified lexical elements into a syntactic frame made up of bound and free-standing grammatical elements. This representation, which also includes some information about phrasal stress assignment, provides a basis for the elaboration of phonetic form and the creation of the next (phonetic) level of representation.

In this model, much that will determine the ultimate form a sentence

will take occurs prior to the level of "functional integration": words become differentially active because of previously occurring sentences, other conversational or environmental influences, or from experimental semantic priming. Some of these words will be verbs that carry with them inherent information about functional relations, that is, about the mappings between thematic roles and grammatical roles. Different forms of the same verb (e.g., active and passive forms) will become available, and these different forms have different "base" strengths, with the active form more likely to prevail. Functional integration involves linking together these activated elements according to the functional relations specified by the verb, beginning with the assignment of a word to the role of grammatical subject.

Using the Model to Understand Aphasic Symptoms

As detailed in this chapter in the second edition of *Acquired Aphasia,* several attempts have been made to attribute aphasic impairments of sentence processing to problems arising during creation of specific representational levels of the model given here, or of one of its precursors. Recent research has attempted to link specific aspects of disordered performance to elements of the model.

Symptoms That Involve Grammatical Morphemes

A critical question regarding aphasic impairments is whether or not the entire spectrum of difficulty with bound and free grammatical morphemes that is found in aphasia should be viewed as emanating from the same level of disturbance within the production model. Garrett (1992) has raised a number of ways in which selection of different grammatical morphemes could arise from different levels of the model, allowing the possibility that grammatical morphology could appear to be quite heterogeneous in its propensity for disruption. In earlier versions of the model (e.g., Garrett, 1980), the structural frame constructed through the operation of positional processes was hypothesized to be constituted of bound and free grammatical markers, and slots to be filled for content words. The speech error corpora on which the model was based provided little reason to distinguish among different types of grammatical morphemes. More recently, LaPointe and Dell (1989) have argued that only bound morphemes are associated with structural planning frames; other elements of grammatical morphology may be conditioned by other parts of the system. If principled

distinctions along these lines can be supported, they might accommodate different vulnerabilities of bound and free-standing grammatical morphemes, as has been demonstrated in aphasic production (Saffran et al., 1989).

Levelt (1989) has drawn further distinctions among the free-standing grammatical morphemes. For example, some prepositions may be viewed as semantically driven (and thus available relatively early in production), whereas other prepositions (e.g., verbal particles) may be specified only as part of the lexeme entry of another word and thus retrieved later. The latter elements are presumably not driven by message-to-functional-to-positional processes, but are "indirectly" elected through the syntactic construction of phrases during positional processing. A distinction between the production of "contentful" and "syntactic" prepositions has been noted in data from agrammatic patients (Friederici, 1982).

A similar distinction may be possible with regard to bound morphemes, although this is less clear. Butterworth (1994) has argued that under some conditions, subject–verb agreement may be conditioned from higher-level, conceptual information rather than strictly determined by syntactic factors at the positional level. Butterworth and colleagues (Vigliocco, Butterworth, Semenza, & Fossella, 1994) described two aphasic patients who were similar in many aspects of their sentence processing including the frequency with which they produced subject–verb agreement errors. Nonetheless, these patients differed markedly in their ability to exploit higher-level influences on subject–verb agreement that presumably arise at the conceptual (message) level. These results are interpreted as evidence that, although the processes governing subject–verb agreement operate primarily over positional level processes, they are influenced by conceptual information that may be selectively affected by neural insult.

It would appear, therefore, that the sentence production model allows distinctions to be drawn within the class of grammatical morphemes that predict differential vulnerability of grammatical morphemes when a disruption occurs at a specific level; whether such distinctions will be able to explain all of the dissociations that have been uncovered remains to be seen.

Symptoms That Involve Grammatical Class Differences

Most aphasic patients suffer some degree of lexical retrieval impairment—deficits that are believed to be largely independent of sentence structural problems. Bock and Levelt (1994) have identified several points in the sentence production process at which the factors influencing selection of a lexical item can affect the form that a syntactic structure will take.

These influences are relatively subtle, operating within a limited timeframe in the speech of fluent speakers; it is not clear what might happen to syntactic structures in conditions of serious (and chronic) lexical inaccessibility. One possibility (frequently described in the aphasia literature but true for normal speakers as well) is that a more accessible word will be substituted for a less accessible word (e.g., *thing* substituted for *astrolobe*). However, if word retrieval deficits are more pervasive, or if retrieval latencies are simply slowed in general, the important lexical influences that have been shown to affect the construction of a syntactic frame may not be available when they are needed.

One hypothesis about how lexical retrieval impairment might be manifested in sentence production was originally proposed by Saffran and colleagues (1980), who noted that many agrammatic patients have considerable difficulty producing main verbs. Because verbs determine the arguments that will be filled by sentence nouns, a problem realizing verbs for production could be an important component of failure to encode functional relations. The verb is the repository of information about the form that a sentence will ultimately take—information that is essential in determining the order in which nouns will be produced, as well as in specifying the grammatical morphemes (auxiliary and inflection) that will constitute the form of the verb.

Group studies have indicated that poor verb retrieval is a characteristic of agrammatic aphasics even in naming tasks, whereas other types of patients (anomic aphasics) are typically *better* able to produce verbs than nouns (e.g., Zingeser & Berndt, 1990). This double dissociation is important, as the syntactic and conceptual complexity of verbs might be argued to make them particularly vulnerable to the effects of brain damage (see Zingeser & Berndt, 1988, for arguments.)

Although these group studies have provided some basis to expect that verb retrieval impairments and sentence production impairments may be related, studies of individual patients have highlighted the complexity of any relationship that might obtain. First, selective verb retrieval impairments are not by any means found only among patients with agrammatic speech (Berndt, Mitchum et al., 1997; Breedin & Martin, 1996; Caramazza & Hillis, 1991). Second, not all patients with agrammatic speech have inordinate difficulty producing verbs (Berndt, Mitchum et al., 1997). Finally, the verb retrieval problems appear to arise from different sorts of impairments in different patients.

Breedin and Martin (1996) addressed this last point by studying verb production and comprehension in four patients who had more difficulty naming pictures of actions than of objects. These authors demonstrated that the four patients had problems with quite distinct aspects of verb pro-

cessing, including difficulty with elements of meaning, with assignment of thematic roles, and with knowledge of the verbs' subcategorization requirements. These results suggest that difficulty producing verbs to name action pictures may arise from a number of distinct disruptions to the information that is typically carried by verbs. In this study, however, all of the patterns uncovered appeared to implicate an impairment at the level of the model at which functional information is computed about meaning, thematic mapping, and syntactic consequences, that is, at the level of lemma retrieval.

Other findings suggest that selective verb impairments may occur later in the process of sentence formulation, as phonological information is activated. Caramazza and Hillis (1991) described two patients who showed difficulty producing verbs compared with nouns across a variety of tasks. Both patients showed normal comprehension of verb meanings; they differed in that one patient had difficulty with verbs only when speaking, and the other had difficulty only when writing. On the basis of these findings, Caramazza and Hillis argued that the verb-specific production impairment occurred at the level of lexeme production, where form-specific phonological or orthographic information is represented to support production in the two modalities of output. Unfortunately, little information was provided about the effect of such impairment of the patients' sentence production in the relevant modalities, but a lexeme-level impairment might be expected to have relatively minor effects on sentence production.

Berndt, Haendiges et al. (1997) have argued that if verb production is impaired at the level of lemma retrieval, then widespread disruption to sentence production should be expected. Since the lemma-level representation provides information about the argument structures of verbs, and about thematic mapping relations, failure to activate this representation should interfere, not only with *verb* production in sentences, but also with realization of obligatory noun arguments. In contrast, if selective verb impairment arises at the lexeme level, as argued by Caramazza and Hillis (1991), then the consequences for sentence production should be limited to a failure of verb insertion.

In an attempt to explore the relationship between verb production impairments and sentence production, Berndt, Haendiges et al. (1997) studied the sentence production of five patients who showed selective verb (relative to noun) impairments in picture naming. The sentence production capacities of two of the patients were substantially improved by giving them a verb to use: these patients formulated more adequate sentences using target verbs than using target nouns, and the realization of noun arguments in picture-description tasks improved if spoken verbs were given to them. For these patients, it was hypothesized that the verb retrieval im-

pairment emanated from a failure to activate semantically and syntactically specified lemma representations. When these were provided for them, sentence production improved in a variety of ways.

Three other verb-impaired patients were not affected by these manipulations. Two of these three appeared to have difficulty constructing a syntactic frame into which verbs could be inserted. When given a verb, they often used it as if it were a noun. The third patient showed a relatively clear lexeme-level impairment, producing phonological errors in sentence production but showing few other symptoms of disruption despite difficulty producing verbs. Again, the message appears to be that a variety of functional impairments can result in similar symptoms. In this case, verb retrieval may fail at a number of different points within the model. If retrieval fails early in the process of sentence formulation (at the hypothesized point of lemma retrieval), this failure may have detrimental consequences on several aspects of sentence production.

This being said, it remains true that, in general, patients who have difficulty producing verbs have difficulty producing sentences. Berndt, Haendiges et al. (1997) also analyzed the narrative production abilities of 10 patients with different patterns of grammatical class retrieval impairments. Noun/verb ratios generated in an object–action naming test were compared across the patient group to a number of morphological and structural indices from the quantitative analysis system of Saffran et al. (1989). The noun/verb ratio was found to correlate strongly, not with morphological omissions in narrative speech, but with structural simplification of sentences. This finding supports and expands the dissociation noted earlier between verb retrieval impairments and agrammatism; although the morphological omissions characteristic of agrammatic production are not strongly related to verb retrieval problems, there remains some indication of a relationship between verb production and sentence elaboration.

One striking finding that emerged from this analysis of the narrative speech samples was that the verb-impaired group succeeded in producing a minimal "sentence" through reliance on the use of "light verb" constructions, that is, constructions that use high-frequency, relatively empty verbs (to be, to have, to make) that essentially pass off the propositional burden of the sentence to adjectives or noun complements. Similar results were obtained when patients were asked to construct a sentence using noun target words. When they succeeded in producing a sentence, the patients did so largely by producing light verb constructions. This strategy could emerge for several reasons. These constructions are very simple, many of them involving the use of a copula and predicate adjective ("Cinderella is pretty"). Many "light" verbs have irregular forms and thus do

not require the addition of an inflection to mark, for example, third person singular. Light verbs are very high in frequency, and thus may be more easily accessible. Finally, in keeping with the discussion in the previous section, it is possible that these relatively "contentless" verbs emerge, not through conceptually and semantically driven functional processing but as part of the creation of the structural frame at the positional level. Thus, an impairment that affects functional level processing (lemma selection) may not prevent production of these types of verbs.

The use of light verb constructions when verb retrieval fails is clearly an adaptive strategy that is not feasible in some elicitation conditions. For example, picture description tasks do not elicit light verb constructions. Breedin, Saffran, and Schwartz (in press) have recently shown that under some elicitation conditions patients with relative verb retrieval impairment produce verbs that are *more* semantically complex and specific than other possible options. In a delayed repetition–sentence completion task, six of eight verb-impaired patients opted to use more contentful verbs when a light verb construction was possible (e.g., "bake a cake" rather than "make a cake"). These authors also failed to find evidence among these patients of reliance on light verb constructions in narrative production, contrary to the results of Berndt and colleagues. Although it cannot be ruled out that there are essential differences between the underlying deficits of the patients tested in these two studies, there are methodological differences that might have contributed to these conflicting results. For example, Breedin et al. analyzed *all* verbs produced in narrative production, whereas the findings of Berndt et al. were based only on verbs that appeared in sentences. Agrammatic patients frequently produce words in isolation (e.g., *dance*) that may be interpreted as a verb using the scoring criteria of the quantitative production system. These fragments are not likely to involve light verbs (i.e., *make* is unlikely to appear in isolation). Even so, it seems clear that when verb retrieval is difficult, patients may opt to produce a sentence using a light verb construction, but only under some circumstances that have not as yet been clearly specified.

Sentence Comprehension: A Framework for Normal Comprehension

The mismatch between the apparent simplicity of the experience of speaking and the actual complexity of the processes that make it possible is, if anything, even more striking for sentence comprehension. In most conditions, normal speakers are not aware of much "processing" between the sensory experience of hearing someone speak and the interpretation of

that speech. It feels as if sound is mapped directly onto meaning, and that this mapping happens continuously even while the speech is ongoing. For those concerned with aphasic disorders, however, it is clear that much can go wrong between hearing someone speak and understanding what was said. The goal here is to sketch out some of the operations that need to be performed on a spoken sentence in order to arrive at a correct interpretation of its meaning, and then to account for some patterns of aphasic sentence comprehension disorders in light of that model.

It might be possible, for the sake of reducing the complexity of this review, simply to "reverse" the production model described earlier and continue to refer to the same levels of representation. To some extent this is possible, as sentence production was not described at a level of detail that involved processes or representations that could not also apply to comprehension. Nonetheless, there are some important differences that must be emphasized between the tasks of producing and understanding sentences. The first involves the TIME-COURSE within which processing must be completed: most importantly, it is not under the control of the listener. Sentence comprehension requires the interpretation of a fleeting, sequential auditory stimulus, with (presumably) the necessity of backtracking over a memory representation of that input if elements are missed or misinterpreted. There is considerable evidence that auditory comprehension normally involves immediate interpretation of semantic and syntactic elements as the sentence is being processed (Marslen-Wilson & Tyler, 1980). Thus, disruption in the temporal aspects of processing, or a limitation of memory capacity, might be expected to have important effects on patients' ability to understand sentences.

In addition, the LINEARIZATION problem is quite different for production (where the linear sequence is the goal and everything must work toward it) and comprehension (where the linear sequence is the starting point, and might be abandoned as soon as it is no longer needed). Little attention has been given to aphasic deficits that implicate processing characteristics such as these that are specific to comprehension; in fact, as with production, the actual processes that are carried out to generate structural representations are largely undescribed. For our purposes, then, it seems best to emphasize the comparability of operations in comprehension and production, and to use a framework that encourages this comparison.

We assume, then, that auditory comprehension of sentences begins with analysis of a complex and ongoing acoustic event into linguistic (phonetic and lexical) elements. The details of acoustic–phonetic conversion are themselves controversial and will not be considered here. This exclusion is not to be taken as an indication that these early aspects of processing have no bearing on sentence comprehension, but only that research on this lev-

el of the system has not focused specifically on sentence-level processes. Thus, our framework begins after the acoustic waveform has been phonetically interpreted (see Blumstein, this volume). From this information, the listener needs to construct a structural representation that (though linguistic formulations differ) at least characterizes the phrasal organization of the sentence into hierarchically interpretable constituents (subject NP, object NP, etc.). In some ways this representation must be similar to the output of positional processing in the production model: Lexical elements are ordered into syntactic frames made up of appropriate grammatical morphemes. Arriving at this type of representation must be quite different for comprehension and production, however. In comprehension, the representation must be parsed from the spoken input as the sentence continues to be spoken. Moreover, unlike the situation for production, in comprehension there is a single opportunity to extract the information needed to build this syntactic representation.

Despite these differences, the levels of representation postulated for comprehension and production are grossly similar. The grammatically interpretable constituent structure extracted from the speech signal in comprehension corresponds to the output of positional processing in the production model. The semantic and thematic interpretation that must be attained in sentence comprehension is similar in content to the information made available through functional processing during sentence production. It was noted earlier that recent revisions of the production model have focused (among other things) on modifying its serial processing requirements to allow an influence from lower levels onto higher levels. This is also true for models of sentence comprehension: Some models propose that the parsing of the speech signal into syntactic constituents must precede semantic interpretation (e.g., Frazier, 1990), whereas others argue that semantic information can exert an influence on sentence parsing (see Carlson & Tanenhaus, 1988, for review). Thus, as discussed earlier with regard to production, one of the important issues to be resolved concerns the nature and time-course of the mechanisms that connect the syntactic (positional) structure and semantic (functional) representations.

Sentence Comprehension Impairments in Aphasia

The type of comprehension disorder of most interest is one that emerges in patients who demonstrate relatively good comprehension of single words, and thus their comprehension problem appears to be specific to sentences. Patients with ASYNTACTIC comprehension fail in sentence–pic-

ture matching tasks in which (a) the sentence to be understood cannot be interpreted simply by mapping knowledge of content word meaning onto knowledge of the real world; and (b) the distractor items depict plausible interpretations that might be given to the sentence if the patient failed to comprehend the structural information in the sentence.

The syntactic complexity of the sentence may be a predictor of performance in some patients, but it is not the essential element. For example, the sentence "the boy that the bee stung cried and ran home" is a complex sentence, but it is one in which the content word meanings alone yield a highly predictable interpretation. When paired with distractors showing, for example, a girl victim, a dog biting, or a child laughing, such a sentence could be easily associated with the correct picture if the patient understood the meanings of the nouns and verbs. In contrast, the sentence "the girl kisses the boy" is less complex, but when paired with a distractor showing a boy kissing a girl would be impossible to interpret based on word meanings alone. In addition, the listener would need to "know" that the subject of "kisses" maps onto the thematic role of agent of the action. It is this recovery of the functional (thematic) roles of the nouns in semantically reversible sentences that has been most at issue in studies of sentence comprehension in aphasia.

A variety of explanations have been given for the pattern of comprehension impairment described; some have focused on the syntactic parsing operations that yield interpretable constituent structures, or on the integrity of syntactic representations themselves. Others have emphasized the difficulty in mapping between constituent structures and thematic information. Still others have postulated a global reduction of processing resources as the responsible underlying deficit. Each of these types of explanation for asyntactic comprehension has a number of variants.

Hypothesized Impairments of Morphosyntactic Processing

Many of the earliest explanations for asyntactic comprehension emphasized the emergence of this pattern among patients with agrammatic production; these accounts focused on the morphological impairments believed to be central to both symptoms (see this chapter in the second edition of this volume for review). A number of possible reasons were given as to why patients should have difficulty with grammatical morphemes in comprehension (e.g., Bradley, Garrett, & Zurif, 1980), all of which had difficulty accommodating the multiple-symptom dissociations that have been reviewed. Nonetheless, a similar account of asyntactic comprehension has been offered by Pulvermuller (1995), who argued that compre-

hension is undermined by selective difficulty in the perception of grammatical morphemes. The impairment is conceived as of probabilistic, with different severities of abnormal "perception probability" resulting in somewhat different performance profiles on different occasions of testing. Although this aspect of the hypothesis allows some room for heterogeneity among patients, a relatively strict hierarchy of difficulty across sentence types is predicted for different severity levels. Although Pulvermuller (1995) argues that this hierarchy is exactly that found among agrammatic aphasic patients, it is not clear that this is the case. In a recent test of the predicted hierarchy, Berndt, Mitchum, and Wayland (1997) found that the comprehension of five Broca's aphasics tested did not conform to the predicted hierarchy across active, passive, and relative clause sentences.

Hypothesized Impairments in the Representation of "Empty Elements"

It is interesting to note that Pulvermuller's hierarchy of difficulty across sentence types is similar to the hierarchy predicted to occur (for very different reasons) among the same types of patients in the original formulation of Grodzinsky's Trace Deletion Hypothesis (Grodzinsky, 1984). As reviewed in more detail in the second edition of *Acquired Aphasia,* this hypothesis held that the "agrammatic condition" is one in which some types of information are unspecified at the level of syntactic structure. Grammatical elements that enter into particular types of structural relations, as well as the "empty elements" ("trace" and "PRO") that mark lexical positions when words are moved from their original positions, are left unspecified. Thus, constructions with moved elements (passive voice and sentences with object-relative clauses) should be particularly difficult. In a recent evaluation of this view, the predicted ordering of sentence types was not found for a group of five Broca's aphasics, four of whom performed equivalently with actives and passives, and two of whom performed equivalently with subject- and object-relative clause sentences (Berndt, Mitchum, & Wayland, 1997). In fact, the predictions of the Trace Deletion Hypothesis (and of Pulvermuller, 1995) regarding the particular vulnerability of passive compared to active voice sentences are supported by performance patterns for only about one-third of patients with agrammatic speech (Berndt et al., 1996).

The Trace Deletion Hypothesis has undergone revision that restricts its scope on the basis of new data about comprehension patterns (Grodzinsky, 1995). Moreover, the basic tenets of Grodzinsky's theory have been adopted by other linguists, who have offered modifications stemming in part from somewhat different views of the motivating linguistic theory (Cor-

nell, Fromkin, & Mauner, 1993; Hickok, Zurif, & Canseco-Gonzalez, 1993; Mauner, Fromkin, & Cornell, 1993). All of these accounts are similar in their postulation of an impairment that affects agrammatic patients' representation of syntactic structure, specifically their ability to represent an antecedent trace of constituents that have been moved from their original (deep structure) positions.

A number of experimental investigations have also been carried out in the spirit of the Trace Deletion Hypothesis; these studies attempt to provide experimental evidence that agrammatic patients fail to represent the hypothesized empty elements. Zurif and colleagues have conducted a number of "on-line" comprehension studies using a paradigm (cross-modal lexical decision) in which subjects must simultaneously listen to a spoken sentence (and show evidence of comprehension) while preparing to make a lexical decision to a written word presented at some point during presentation of the spoken sentence. On some trials, the written word presented for lexical decision occurs at the point in the sentence (the gap) that is filled by the hypothesized empty element. Research with normal listeners has indicated that lexical decision latencies for "word" responses to targets presented at the gap in the sentence decrease if the target is semantically related to the moved element (the "filler"). Such indications of priming at the gap site have been interpreted as an indication that listeners reactivate the filler on-line at the gap site (e.g., Tanenhaus, Carlson, & Seidenberg, 1985).

Zurif and colleagues have presented data that Wernicke's aphasics, but not Broca's aphasics, show evidence of reactivation at the gap site for both subject-relative clause sentences (Zurif et al., 1993) and object-relative clause sentences (Swinney, Zurif, Prather, & Love, 1996). These results are interpreted as an indication that Broca's aphasics experience a processing deficit that affects their ability to build syntactic representations in real time. Blumstein and colleagues (1998) have recently reported a failure to replicate this result using an auditory–auditory paradigm that successfully elicits normal filler-gap priming. In contrast to the results reported by Zurif and colleagues, eight Broca's aphasics showed the normal result of priming at the gap, whereas six Wernicke's aphasics did not.

Blumstein and colleagues raise a number of possible reasons that results from the two series of studies might have differed. Primary among them is the requirement in the Zurif studies that patients simultaneously attend to stimuli presented in two modalities. Because Zurif and colleagues base the (theoretically strong) interpretation of their results with Broca's aphasics on failure to reject the null hypothesis, the possibility that the task demands undermined the performance of the Broca patients should not be dismissed lightly. Unequivocal data demonstrating that the patients could

read the lexical decision targets, and that they showed priming to the prime–target pairs presented outside the sentence context, are required before the null result in sentence context can be interpreted. Another possibility that cannot be ruled out is that there are important differences among the patients tested in these two series of studies that contributed to the conflicting results. Minimal information is presented about patients' off-line sentence comprehension in any of these studies; thus, it is difficult to know what the relation might be between gap-filling and sentence comprehension.

Evidence against a General Syntactic Parsing Problem in Asyntactic Comprehension

A substantial body of evidence has been reported that undermines all of the arguments just reviewed suggesting that asyntactic comprehension arises from difficulty computing syntactic structures. One paradigm that has been used is one in which subjects are asked to judge the syntactic well-formedness of sentences; they are not asked to interpret their meanings. In an important study, Linebarger, Schwartz, and Saffran (1983) demonstrated that a group of four agrammatic aphasics who failed on sentence–picture matching tasks with semantically reversible sentences was nonetheless quite good at judging the syntactic acceptability of sentences. The subjects showed an appreciation for a range of syntactic facts, including knowledge about the subcategorization requirements of specific lexical items (i.e., restrictions on the kinds of complements that can apply to verbs) and an ability to interpret discontinuous elements in sentence structures.

Perhaps most importantly, this study demonstrates remarkable sensitivity to the structure-marking role of a range of grammatical function words. Because these elements are often omitted from agrammatic speech, and because they are important elements indicating syntactic structure, it has been hypothesized (as noted earlier) that the agrammatic deficit reflects a primary impairment in processing this category of words (e.g., Pulvermuller, 1995). A number of studies have now demonstrated a large gap between the limited ability of individual patients to understand semantically reversible sentences and their well-retained ability to perform grammaticality judgments (Berndt, Salasoo, Mitchum, & Blumstein, 1988; Linebarger, 1990; Shankweiler, Crain, Gorrell, & Tuller, 1989).

Another task that has been used extensively in studies of aphasic comprehension is word monitoring, in which subjects listen to a sentence and press a reaction time key when a particular target word is recognized. This task has been widely employed in studies of normal sentence comprehension, specifically to address questions regarding the time-course of activa-

tion of specific types of information during processing (Marslen-Wilson & Tyler, 1980). Subjects monitor for a target word that is not itself at issue but occurs in the sentence immediately following some important structural manipulation. Often this manipulation involves an ungrammatical or semantically anomalous element. In general, normal response times are slowed by the occurrence of irregularities in the sentences, often to different degrees for different types of violations.

In one type of manipulation, the focus was on the semantic and syntactic restrictions on elements that can follow specific verbs. Across a number of studies with different patients, Tyler (1985, 1988, 1992) found clear evidence of sensitivity to syntactic structure among nonfluent patients. In one study, listeners monitored for target words immediately following the verb, and the pragmatic, semantic, and subcategorization relations between the verb and the target noun were manipulated. Four of five nonfluent patients showed normal sensitivity to all aspects of verb–argument relations, that is, latency to detect the target was slowed for all three violation types. In another series of studies, patients and control subjects monitored for targets embedded in semantically anomalous, scrambled, and normal prose. Like normal subjects, the patients showed the normal word position effects when monitoring in normal prose—reaction times were faster when targets appeared later in sentences. This result is interpreted as evidence that the patients are able to construct a syntactic representation "on-line" that integrates lexical–semantic information normally.

Another study employing the word monitoring paradigm uncovered remarkable sparing both of grammatical class information (nouns vs verbs) and of the structure-marking role of function words (determiners, modals) that signal grammatical class (Wayland, Berndt, & Sandson, 1996). Four patients with the asyntactic comprehension pattern showed the same word-monitoring performance pattern as normal controls; that is, processing speed was slowed when subjects encountered a syntactic mismatch even when that mismatch was semantically consistent with the context. Thus it appears that patients who fail to comprehend semantically reversible sentences continue to provide evidence of substantial ability to perform normal syntactic processing.

Temporal Perturbations and the Computation of Syntactic Structure

An noted earlier, considerable interest has emerged in the idea that sentence-processing disorders in aphasia might arise, not from a complete inability to perform some operation, but from a disruption of the normal time-course over which that operation must be executed in order to be in-

tegrated with the output of other operations. Some accounts have argued that Broca's aphasics (or agrammatic aphasics) experience a slowing of activation of structural information (Friederici & Kilborn, 1989; Haarmann & Kolk, 1991b). A related, alternative account is that information decays prematurely, also leading to an "asynchrony" between pieces of information that need to be coordinated in the course of various processing operations. Haarmann and Kolk (1991a) implemented a computational model of aphasic comprehension (SYNCHRON) in which manipulations of the activation and decay parameters successfully simulated aspects of asyntactic comprehension.

Several studies have attempted to resolve the slowed activation or fast decay accounts by testing patients in a semantic priming paradigm that manipulates the time available between the onset of the prime and the onset of the target. Friederici and Kilborn (1989) hypothesized that the temporal problems experienced by Broca's aphasics interfered with the normal representation of grammatical morphemes in the computed surface structure; thus, this hypothesis is a variant of the "grammatical morpheme" deficit approach reviewed earlier. Using a cross-modal lexical decision task with five Broca's aphasics, these authors found some evidence both for fast decay and for slowed activation in different aspects of the data.

Haarmann and Kolk (1991b) used some different methodology in conducting a similar study and found most consistent evidence for slow activation. In a later study investigating sensitivity to subject–verb agreement, Haarmann and Kolk (1994) found evidence not for slowed activation but for fast decay: The authors reconcile these results by arguing that Broca's aphasics may show either slow syntactic activation or fast syntactic decay, but not both at the same time.

Tyler, Ostrin, Cooke, and Moss (1995) have raised a number of objections to the methods used and to the interpretations given to the results in these studies. In addition, they point out that there was apparent heterogeneity across the patients tested in whether or not they showed these temporal effects, again questioning the feasibility of providing any general account of processing disruption that will hold for all members of a clinically defined group. It must be emphasized that the experimental paradigms used in these studies (especially cross-modal lexical decision) are very complicated; they require patients to perform two different tasks simultaneously. Furthermore, even the strongest of findings involve quite subtle results, differences across conditions measured in milliseconds against a backdrop of considerable baseline reaction time variability. As emphasized by Tyler and co-workers (1995), there are a number of seemingly small methodological decisions in the design of these tasks that can have im-

portant consequences for the outcome. Thus, we exercise caution in interpreting the results of these studies. At this point, there appears to be evidence that at least some patients experience a breakdown of the ability to coordinate access to distinct sorts of information over time, and that this deficit could contribute to their sentence comprehension disorder. However, there remain substantial questions concerning the proportion of patients who demonstrate such an impairment and the conditions in which it emerges.

Resource Limitations, Capacity Constraints, and Short-Term Memory

A number of proposals have been made that comprehension fails in aphasic patients because of cognitive limitations that are either generalized in their effects on performance or that limit language processing in relatively specific ways. Kolk's "time-based" approach to comprehension impairment (1995) is sometimes couched in terms of a resource limitation (Haarmann & Kolk, 1991b; Kolk & Weijts, 1996), which as noted earlier may interfere with the normal time-course of information availability. The primary effect of this hypothesized resource limitation is that the syntactic structure of the sentence is not computed normally; the superficial symptoms that emerge in any individual patient, as reviewed earlier with regard to sentence production, will reflect the types of adaptations that the patient makes to the lack of availability of a complete structural representation delivered in a timely manner.

Other discussions of resource constraints have postulated that these limitations exercise their effects somewhat differently. For example, Blackwell and Bates (1995) have argued that the resource limitation in agrammatic aphasia makes specific sets of grammatical morphemes particularly vulnerable. Within the framework of the Competition Model (Bates & MacWhinney, 1989), the vulnerability of different aspects of grammatical morphology is predictable from their "cue validity" (their relative importance in the language as cues to sentence interpretation) and their "cost" (their relative level of detectability). Blackwell and Bates induced errors in normal subjects' judgments of sentence grammaticality by requiring that those judgments be made while holding in memory digit lists of various lengths. Under even relatively low load conditions (two digits), normal subjects found it more difficult to detect errors of agreement than errors of omission, which was in turn more difficult than detecting word transpositions. This hierarchy was not linked directly to aphasic performance patterns in grammaticality judgment tasks; rather, the point was made that the

vulnerability of grammatical morphology under conditions in which the system is stressed does not necessarily indicate an essential disorder of syntactic processing.

A somewhat similar approach was taken by Miyake, Carpenter, and Just (1994), who induced aphasic comprehension profiles in normal subjects by presenting written sentences word by word at a very fast rate. The hierarchy of difficulty across sentence types for normal subjects in this condition of processing overload was similar to the reported hierarchy of difficulty for the same sentence types in aphasic patients (e.g., Caplan & Hildebrandt, 1988). Using the framework of the Capacity Theory of Comprehension (Just & Carpenter, 1992), Miyake and colleagues (1994) interpreted these findings as reflecting the effects of reducing a general-purpose working memory capacity—a capacity that supports not just language processing, but cognitive processing across a range of tasks. The implication is that brain damage and experimentally induced processing overload result in a similar degradation of working memory capacity and thus undermine sentence comprehension in the same way. This interpretation was formalized and tested further with a computational model that allows control over the temporal synchrony of lexical, semantic, and syntactic aspects of sentence processing (Haarmaan, Just, & Carpenter, 1997). A number of symptoms that have been noted in aphasic comprehension patterns were successfully simulated (e.g., declining performance with added length, poorer performance with passive and object-relative clause sentences than with active and subject-relative sentences). The reduction of resources in the model results in slowed processing and only partial comprehension, represented as a less complete and less activated meaning representation. Resource reduction within this account affects processing at a number of different levels during sentence comprehension. That is, it does not exert its effects selectively on grammatical morphology (as in Blackwell & Bates, 1995) or on syntactic structure more generally (as suggested by Caplan & Hildebrandt, 1988).

Caplan and colleagues (Caplan & Hildebrandt, 1988; Caplan & Waters, 1995) have also invoked the concept of reduction of processing resources to explain aphasic sentence comprehension patterns, but this account differs substantially from the Capacity Theory of Comprehension. In Caplan's account, capacity reduction is argued to affect a specific set of resources dedicated to syntactic processing; the resources needed for other cognitive activities, such as problem solving, are not affected by the resource limitation. Caplan and Waters (1995) offer a number of critiques of the analyses of aphasic comprehension offered by Miyake et al. (1994), including the relatively large gap in the severity of the "induced" comprehension failure of normal subjects (around 80% correct) and that reported

for aphasic patients (around 50%) on some sentence types. Another rather compelling critique of the global capacity limitation view is the fact that patients with Alzheimer's Disease, who demonstrate clear working-memory limitations when tested in a variety of ways (including the working-memory test favored by Miyake et al.), fail to show complexity effects in sentence comprehension. Caplan and colleagues argue that the resources that are limited in aphasic patients and that undermine comprehension are resources that are specifically dedicated to the processing of the syntax of sentences.

The working memory capacity that is postulated in the Capacity Theory of Comprehension differs in many respects from other formulations of the nature of working memory, including the model of working memory that is the most widely cited. These different views of working memory, and the various ways in which working-memory limitations have been implicated in aphasic sentence comprehension, are highlighted in a critique of Miyake et al. that argues for selective impairment of specific language-processing components in aphasia (R. C. Martin, 1995). This critique also challenges the view of working-memory capacity that is represented by the Capacity Theory.

R. C. Martin and colleagues (Martin, 1993; Martin & Feher, 1990; Martin & Romani, 1994; Martin, Shelton, & Yaffee, 1994) have investigated the relationship between working memory and sentence processing from a number of different perspectives. These studies amount to a major challenge to the global capacity account as well as to the prevailing view of working memory (Baddeley, 1986), which postulates that auditory–verbal short-term memory consists of a phonological buffer and an articulatory rehearsal system. A number of patients have been described who demonstrate a fairly selective deficit to phonological short-term memory, and arguments have been presented that a reduction of short-term memory should invariably result in sentence comprehension problems (e.g., Vallar & Baddeley, 1984). In contrast to this view, Martin and colleagues demonstrated quite clearly that short-term memory impairment (as measured by reduction of phonological span) is not invariably related to sentence comprehension impairment (see R. C. Martin, 1993, for review; see also Caplan & Waters, 1990).

Furthermore, the research of Martin and others has led to the suggestion that the conception of short-term memory should be elaborated to include storage capacities associated with different components of language processing (see also Saffran, 1990, for arguments). Working primarily from a detailed study of individual patients, R. C. Martin and colleagues (1994) demonstrated that memory impairments for different types of information (e.g., phonological, semantic) are associated with comprehension impair-

ments for the same types of information. A similar conclusion was reached in a study investigating the interrelationships between the memory and language processing abilities of 15 aphasic patients (N. Martin & Saffran, 1997). Performance across a range of language tasks assessing relative impairments of phonological versus lexical–semantic processing was correlated with indices of relative reliance on phonological and semantic information in span tasks. When patients demonstrated relative impairments in phonological processing in language tasks, there was evidence that they relied primarily on lexical–semantic information in span tasks. In contrast, patients who showed semantic impairments as a primary aphasic deficit showed evidence of reliance on phonological information in span tasks. These and other data are used to support a new conception of short-term memory that blurs the traditional separation of memory and language processing and highlights the complexity of the relationship between storage and processing in sentence comprehension. Clearly, this emerging view of distinct storage capacities for the distinct outputs of separate language components stands in sharp contrast to Capacity Theory, which postulates a single working-memory resource, not just for all aspects of language processing, but for all aspects of cognitive processing.

Linking Syntactic Structure and Sentence Interpretation: Thematic Role Assignment

Most of what has been reviewed here concerning sentence comprehension in aphasia focuses on patients' ability to extract (or compute) syntactic structure from the speech signal. Although many and diverse hypotheses have been offered that place the source of comprehension failure in an inability to compute the correct syntactic representation, other data were reviewed that indicate considerable retention of syntactic processing abilities—a level of retention suggesting that a syntactic processing deficit is not solely responsible for comprehension failure. In response to the earliest demonstrations of the extent to which the syntactic parsing abilities of asyntactic comprehenders are intact (Linebarger et al., 1983), Saffran and colleagues offered a new account of patients' comprehension failure (Saffran & Schwartz, 1988). This hypothesis argued that the essential nature of patients' comprehension was not "asyntactic" or "agrammatic," as considerable syntactic integrity was demonstrated in the grammaticality judgment task. Rather, the impairment was hypothesized to involve the processes that map between syntactic constituents and their thematic roles. In an experimental exploration of this hypothesis, reviewed at length in the previous edition of *Acquired Aphasia*, Schwartz, Linebarger, Saffran, and

Pate (1987) used an anomaly detection task to investigate the relationship between elements of syntactic structure (e.g., active v. passive) and patients' ability to detect implausible thematic relationships. The primary finding was that syntactic structure played an important role in whether or not patients could successfully "map" between sentence constituents (e.g., subject, direct object) and thematic roles (e.g., agent, theme). Structures with a "transparent" mapping between the order of elements in both deep and surface structures (e.g., active voice sentences) can be interpreted even when mapping processes are impaired; structures that are not transparent in this sense are much more difficult for patients to interpret.

In a new study using the anomaly judgment task, Saffran, Schwartz, and Linebarger (1998) manipulated the semantic relations between verbs and their noun arguments to explore the extent to which normal subjects and asyntactic comprehenders are influenced by semantic information as sentences are being heard. This study was motivated by work with normal subjects demonstrating clear effects of semantic information on listeners' initial assignment of thematic roles (e.g., Caplan, Hildebrandt, & Waters, 1994; Tabossi et al., 1994).

Different types of implausibility were compared in this study. In the "verb constrained" condition, the implausibility arises because of a selectional restriction mismatch between the verb and one of its arguments. If the thematic roles of the nouns were reversed, the plausibility of the sentence would change. The mismatch might involve the sentence agent ("the cheese ate the mouse") or the sentence patient–theme ("the child frightened the movie"). In the proposition-based condition, the implausibility arises not from any semantic constraint on individual verb–noun thematic assignments, but from the relationships expressed by the proposition as a whole, for example, "the car demolished the tornado". In this example, both tornadoes and cars can demolish things, but only one of them can demolish the other. Control sentences were also included in which the assignment of thematic roles to arguments did not affect plausibility ("the cook sliced the water"). Four syntactic structures (active, passive, subject cleft, and object cleft) were used in each condition.

Although the plausible members of the pair in the two experimental conditions were judged equally plausible in a separate rating study, normal subjects were faster to respond to plausible verb-constrained sentences than to proposition-based sentences. Importantly, response times were affected by the position of the "constrained" argument in the verb-constrained sentences; subjects responded faster when the constrained noun occurred early in the sentence, resulting in faster responses to agent-constrained actives and patient-constrained passives, and slower responses to patient-constrained actives and agent-constrained passives.

These results are interpreted as evidence that lexical–semantic information facilitates thematic assignment, and that it does so "on-line" as the sentence is being processed.

The same task was presented to seven aphasic patients who had difficulty understanding semantically reversible sentences (i.e., "asyntactic" comprehenders). As in the Schwartz et al. (1987) study reviewed earlier, the patients failed more often when sentences were either passive voice or other structures with nontransparent mappings. The most interesting finding was that the aphasic patients performed much worse with the implausible verb-constrained sentences (46% error rate) than with the implausible proposition-based items (23% error rate). This result is interpreted to mean that when strong semantic constraints are present in the sentence, patients tend to rely on them at the expense of syntactic structure. However, the relatively good performance shown by the patients in detecting implausible sentences in the proposition-based sentences suggests that the same patients were able to use syntactic structure if they were not misled by semantics.

Saffran and Schwartz (1994) have discussed these results and compared them to findings of a quite different sort obtained from patients with severe semantic impairment. Several case reports have now documented the striking preservation of the ability to use syntactic information to assign thematic roles in sentences among patients who have lost basic lexical–semantic information about sentence nouns (Marshall, Chiat, Robson, & Pring, 1995–1996; Schwartz, Marin, & Saffran, 1979). The contrast between the performance of such patients (who rely on syntactic structure when semantics is weak) and the asyntactic comprehenders (who rely on semantics when syntax is weak) provides compelling evidence that these components of language can be selectively affected in different types of brain damage. The contrast also indicates that patients will attempt to interpret sentences using whatever resources they have available to them.

Discussion: Treatment of Sentence Processing Deficits

This review has attempted to use conceptions about how sentence processing is normally performed to guide investigation of the nature of sentence processing impairments in aphasia. It should be clear that reliance on the normal model is useful if one's interest is in understanding either how the language system is organized or how components of language are represented in the brain. But the question can be raised as to whether this emphasis on normal processing has any practical value in the clinical setting.

That is, if we succeeded in attributing a specific symptom (e.g., asyntactic comprehension) to a single hypothesized impairment within the model (e.g., to a failure to access thematic role information in verbs), would this knowledge be of any value to the patient?

There are a number of ways in which the model-driven approach has proven useful in clinical applications. In one series of studies investigating the language processing abilities of severely aphasic patients, Weinrich and colleagues have uncovered considerable residual processing abilities among global aphasics who are trained to use a visual communication system (C-VIC). In terms of the normal model, C-VIC can be viewed as removing several of the requirements of positional processing (morphological and phonological operations) from language tasks. Nonetheless, use of C-VIC continues to require knowledge of thematic and grammatical roles, as well as information about how the two can be "mapped" onto one another. These studies have demonstrated that severely aphasic patients retain substantial ability to understand and produce reversible locative sentences (Weinrich et al., 1993), and to assign thematic roles in sentences with verbs (Weinrich, McCall, & Weber, 1995).

These studies have also identified important limitations in generalization of skills across levels of the sentence-processing model. For example, patients who (after training) were able to construct C-VIC sentences to describe pictures continued to have great difficulty constructing multiple sentences to describe a more complex video enactment (Weinrich, Shelton, McCall, & Cox, 1997). This result is interpreted within the model of sentence production outlined here as providing information about the myriad factors that constitute the patient's extraction of a "message" from videotaped scenes (or from fast-moving real life). Several other studies using C-VIC have demonstrated substantial improvements in patients' production of spoken language after training (Weinrich, McCall, Weber, Thomas, & Thornburg, 1995; Weinrich, Shelton, Cox, & McCall, 1997), presumably reflecting improved functional and positional level processing that resulted from experience using C-VIC. The conceptions of normal language processing that have guided this work have provided a coherent framework within which these results can be interpreted.

Another approach to demonstrating the clinical relevance of model-driven interpretation of aphasic symptoms involves the development of new approaches to treatment. The idea is that the design of treatment for a specific symptom can be motivated by an understanding of the nature of the impairment, and the treatment can be tailored to fit the deficit (Mitchum & Berndt, 1995). Treatment is thus directed at improving processing at the locus of the disruption; if processing can be improved, the next step is to look for the effects of the change on other language func-

tions. Although the goals of this approach clearly include designing new treatments that will ultimately prove useful in the clinic, the most immediate point is to attempt to validate the interpretation of the patient's impairment in terms of the normal processing model.

The studies carried out from this perspective have highlighted the complexity of the approach. Much interest was generated some years ago when Byng (1988) reported a successful model-driven treatment with an agrammatic patient. A series of diagnostic tests suggested that the functional locus of the patient's deficits (both to sentence comprehension and production) involved the set of procedures for "mapping" between thematic and grammatical roles. Even though the patient was shown to have specific difficulty with verbs, therapy was directed at practice in mapping between thematic and grammatical roles from locative prepositions in reversible contexts. Not only did this intervention reestablish the patient's comprehension of reversible locative sentences, but it was also followed by an improvement in his comprehension of reversible active and passive sentences. The "mapping" process that was reinstated in this patient was apparently very abstract, presumably granting to him not only an appreciation of the necessity of word order in the expression of relationships, but also giving him the important information that those word orders are dictated by a single sentence element (i.e., the preposition or the verb).

Several attempts have been made to address similar "mapping" deficits in sentence comprehension, and results of these studies have uncovered a number of interesting problems. Efforts to replicate Byng's study have been relatively unsuccessful, apparently because the patients involved suffered from additional impairments that were not addressed in therapy (Byng, Nickels, & Black, 1994; Nickels, Byng, & Black, 1991). For example, in addition to the hypothesized mapping deficit, some patients may have difficulty with comprehension tasks because of problems understanding the events depicted in test materials (Marshall, Pring, & Chiat, 1993).

An innovative approach to the issue of treatment replication, and to the problem of symptom variability among candidate patients, was proposed by Schwartz, Fink, and Saffran (1995). These authors argued for a series of preplanned interventions that could address separate symptoms in different treatment modules. For example, if the patient has an impairment of event perception in addition to a mapping deficit, the event perception impairment can be addressed before mapping treatment is initiated (Marshall et al., 1993).

Schwartz and colleagues have carried out a programmatic series of treatment studies directed at thematic mapping in sentence comprehension, and these studies have demonstrated varying degrees of success among the broad range of patients who have participated (Schwartz, Saf-

fran, Fink, Myers, & Martin, 1994). A different protocol for the treatment of asyntactic comprehension was adopted by Mitchum, Haendiges, and Berndt (1995), who used systematic error feedback in sentence–picture matching to induce patients to learn the relationship between structure and meaning in active and passive sentences. This approach has been used with three patients, all of whom eventually learned to understand these sentence types. However, the three patients demonstrated very different patterns of generalization to new sentence materials, which implicated additional, untreated deficits (see Berndt & Mitchum, 1998; Haendiges, Berndt, & Mitchum, 1996). It appears from these studies that chronic aphasic patients can learn to "map" between grammatical and thematic roles. What still remains to be determined is how best to identify the patients who will respond to this approach, and why.

Other studies in a similar vein have focused on sentence production. Several of these have used the framework of the sentence production model reviewed here, and have attempted to determine the relationship between problems producing verbs and problems producing sentences. As noted, there is a correlation between these two symptoms, but the direction of the relationship has not been determined. In testing the hypothesis that verb retrieval impairments *cause* sentence production impairments, several studies have sought to improve patients' access to verbs in production tasks and have tracked the effects of this improvement on sentence production. Results have not been encouraging.

Mitchum and Berndt (1994) succeeded in training a patient with sentence production impairments to produce a core set of verbs by using repeated picture naming. Despite the patient's facile ability to produce the target verbs after treatment, sentence production using the same verbs did not improve. A similar outcome was recently reported by Reichman-Novak and Rochon (1997). However, continued study of the same patients demonstrated the efficacy of a different approach. Rather than focusing on functional processing to enhance verb production, another study was done that motivated functional-to-positional processing in the production of grammatical morphemes. This treatment focused on training the patient to produce the elements of verb morphology responsible for marking verb tense, and it resulted in much improved sentence production. The availability of the morphological elements of the sentence frame that was induced by the treatment resulted in improved production of verbs in sentences across a range of untrained tasks (see also Mitchum, Haendiges, & Berndt, 1993; Weinrich, Shelton, Cox, & McCall, 1997).

The explicit treatment of elements of the sentence frame thus appears to result in a more positive outcome than does treatment of verb retrieval. However, it is possible that the verb treatments failed to target the correct

aspect of verb representation. As noted in the review of the issue of verb impairments, some patients experience quite selective impairment of specific aspects of verb representation. Marshall, Chiat, and Pring (1997) reported a successful treatment of verb retrieval that focused on thematic relations and mapping requirements for specific verb type. Although the treatment resulted in marked improvement of sentence production using those types of verbs, there was no generalization to verbs having a different predicate–argument structure. This study highlights the importance of using a model to guide treatment in attempting to interpret generalization to untreated items. In this study, the selection of the verbs to be treated was explicitly motivated by theoretical issues involving thematic mapping; thus the lack of generalization to untrained materials, so often found in aphasia treatment studies, had a ready explanation.

This emphasis on theoretically driven studies of treatment generalization is clearly illustrated in the work of Thompson and colleagues, who are motivated by a linguistic theory of syntactic representation (see Thompson & Shapiro, 1995, for review). These studies organize sentences of different types into categories based on their hypothesized linguistic representations. The expectation is that generalization across sentence types will be limited by the underlying similarity of treated and untreated constructions. A complex and interesting pattern of generalization has been reported that frequently, but not always, conforms to the predictions of the model. These treatment studies may thus motivate changes in the way that the processing requirements of different but similar structures are conceived.

These studies allow a number of interesting conclusions in addition to the obvious but important one that long-standing sentence processing disorders can yield to treatment under the right conditions. The roughly described levels of the system that were the focus of these interventions are still underspecified in the models. It is possible that detailed studies such as these—studies that even more closely control the information made available to the patient—might contribute in important ways to specifying what these operations involve. For example, it is conceivable that some of the treatment techniques that focused on thematic mapping in comprehension contributed to an improved structural representation, and that this improvement was responsible for the posttreatment gains. Future studies might employ some of the on-line methods described here to assess the integrity of this structural level of representation before and after treatment in order to assess the effect of "mapping" therapy on that level.

This review has summarized some of the extensive research that has been conducted on the breakdown of sentence processing in aphasia. Although much has been learned in recent years about the various ways in

which sentence processing can be disrupted, it should be clear from this review that many critical questions remain unanswered. Active research will continue to be directed at this important aspect of acquired aphasia.

Acknowledgment

The preparation of this chapter was supported by National Institutes of Health Grant R01-DC00262 to the University of Maryland School of Medicine.

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8

Explanations for the Concept of Apraxia of Speech

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The term APRAXIA OF SPEECH has had an extremely variable history. It has been used to describe different types of behaviors, in different types of patients, and under different stimulus conditions. The term has been used, often with qualifying modifiers, to describe syndromes or parts of syndromes. The different conceptualizations of apraxia in general have engendered inconsistencies for the definition of apraxia of speech.

I propose that under certain interpretations there are additional forms of apraxia of speech that differ from the frontal lobe and insular speech apraxias. It is a sine qua non that certain phonological functions operate in ways that do not acknowledge the physical descriptions that characterize the sensory–motor practic functions of the nervous system (Goldsmith, 1995). Much of the impasse and tension in the discussions of apraxia of speech is due to the gulf between the explanatory vocabularies of the mental versus the physical sciences (J. Jackson, 1931b). Phonological functions involve selecting and sequencing of phoneme-like units that speakers BELIEVE themselves to be uttering. In actuality, they are uttering sounds, or PHONES, which may then be considered as being produced by nervous firings giving rise to complex and synchronous muscular contractions that result in acoustic impingements on the air. The description of phonological processing should not imply that phonemes "are sounds" or that they are articulated. Phonemes ARE NOT SOUNDS. They are "psychologically real" abstract categories for which no invariant factor, articulatory or acoustic, has yet been found—nor is ever likely to be found. Furthermore, the subphonemic errors made by certain patients typically involve dis-

turbed limb–praxic function for the vocal tract "limbs" and the motor commands for outputting speech sounds (phones) and prosody—the phonemes and their sequential order having been somehow readied at higher levels.[1] The result is an effortful, groping speech output, usually seen when the left frontal region is damaged in, around, and/or underneath the foot of the third convolution, encompassing pars opercularis (Broadman Area 44) and/or pars triangularis (Broadman Area 45).

A close analysis of the historical development of apraxia and of its many current characterizations (Rothi & Heilman, 1997; Rothi & Heilman, 1996) explains why the apraxias in general permit alternate explanations: one implicating disconnection lesions (lesions that disengage centers) and another implicating cortical lesions of the centers themselves. This state of affairs has unwittingly resulted in confusion over the issue of apraxia in general and its derived notion of "apraxia of speech," "rekindled" by Darley (Sasanuma, 1989, p. xii) but originally identified by Liepmann (Rosenbek, 1993, p. 444).

Historical Background

Liepmann's Precursors

Before the work of John Hughlings Jackson (1931c) and Hugo Liepmann (1900; also see Eling, 1994; Kimura, 1980; Rothi & Heilman, 1996),

1. The wording here is dangerously close to the fallacy that John Hughlings Jackson (1878/1931b) warned against. He believed that a "psychical state" (an "idea of a word" or simply "a word") cannot produce an articulatory movement, which is clearly a physical phenomenon. Rather, it was "discharge of the cells and fibers of the anatomical substratum of a word [which] produces the articulatory movement" (p. 156). Jackson stated, "In our studies of diseases of the nervous system we must be on our guard against the fallacy . . . that somehow or another an idea produces a movement" (p. 156). Phonemes, like words, are abstractions and like the "psychical" elements of Jackson do not "fine away" into physical articulatory production. This dualism of Jackson is suggestive and should be carefully considered when dealing with phonemes and neurological theories of speech production; that is, the problems are precisely what Jackson was defining last century (Buckingham, 1986b; Engelhardt, 1975). Phonemes are mentalistic constructs of the mind; they have psychological, not physical, reality. A phoneme is an abstract unit, which by its ontological nature cannot be uttered. What are actually produced are phones (sounds). Phones that pattern together, that are phonetically similar, and that most often are found in complementary distribution are referred to as the ALLOPHONES of a phoneme. The Greek morpheme *allo-* means closely related, being one of a group whose members together constitute a structural unit. Phones group together as allophones, whose members together constitute a structural unit—the phoneme. Fromkin and Rodman (1993) quote the early twentieth century linguist, Edward Sapir: "In the physical world the naive speaker and hearer actualize and are sensitive to sounds, but what they feel themselves to be pronouncing and hearing are 'phonemes' " (p. 172).

who are usually credited with developing the notion of apraxia (Brown, 1988b; Harrington, 1985, 1987), Paul Broca (1861/1960) considered two alternative explanations for the speech problem of his patient, Leborgne (Tan) (Clarke, 1980; Lebrun, 1989; Schiller, 1992; von Bonin, 1960). The first, which he eventually adopted, was that the patient had lost his "faculty of articulate speech."[2] This was an intellectual faculty and consisted of the "memory for the procedure one has to follow in order to articulate the words" (p. 54), an idea attributed to Bouillaud. Broca (1861/1960) suggested that this modular faculty may be "a kind of memory." He writes, "This memory is not in relation with any other memory or the rest of the intelligence" (p. 53). It was not a general memory, but one specifically slotted to remember how to coordinate articulatory movement. There is an intriguing similarity with Liepmann's notion of "movement formulas." These were kinetic memories; they were procedural but were closer to the kinetic memories of Liepmann than to Liepmann's so-called "movement formulae."

Broca (1861/1960) also considered the possibility that aphemia might "be a kind of locomotor ataxia, limited to the articulation of sounds" (p. 54). According to Broca, this option would imply that the disorder would not be the loss of an intellectual memorial faculty, "which belongs to the thinking part of the brain" (p. 54), but rather "it would only be a special case of the general coordination of actions, a faculty which depends on the motor centers of the central nervous system" (p. 54). At the time of Broca's paper, the "Bell-Magendie" sensorimotor dichotomy had not reached the cerebral cortex (Young, 1990). Consequently, it was difficult to conceive of a CORTICAL motor (nonintellectual) system, and therefore damage to the cortex should only disrupt "intellectual faculties"; motor organization and function were not thought to be intellectual. Broca (1861/1960) states quite succinctly, "Everyone knows that the cerebral convolutions are not motor organs" (p. 70). Subsequent to Broca's presentation, the stage was set for arguments as to whether Broca's aphasia involves language or speech, although in 1861, the discussion was whether it was an intellectual impairment of a modular form of memory for speech–movement procedures or a nonintellectual problem with locomotion, not unrelated to our present-day quandaries about teasing apart certain forms of speech apraxia from the language proper.

2. The preponderance of the word FACULTY at the time Broca was writing was due to the development of the so-called faculty psychology of the eighteenth-century Scottish philosophers Thomas Reid and Dugald Stewart. Franz Gall drew heavily from the Scottish school when developing his theories of phrenological faculties. These theories subsequently influenced Bouillaud, who passed them on to Auburtin and Broca. Thirty-five years before Broca, Bouillaud (1825) had argued (in support of Gall) that the "faculty of articulate language" is mediated in the anterior lobes of the brain (Young, 1990).

Benton and Joynt (1960) documented historical observations of speech-specific lingual incoordination secondary to brain damage and found that it is a long-observed phenomenon. The same observation was made by Auburtin and Bouillaud (Stookey, 1963), both of whom were precursors of Broca (Buckingham, 1981). In addition to noting simply that patients could swallow, masticate, and so forth, but could not utter words normally, Baillarger (Alajouanine, 1960) and Auburtin (1861) observed that patients could quite fluently produce speech automatisms. Still, a third observation of the Broca's aphasic was made. Broca wrote, "that they can immediately WHEN BEING ASKED bring their tongue up, down, right, etc. But, however precise these movements may appear to us they are infinitely less so than the excessively delicate movements which the language demands" (p. 54, emphasis added). From the description provided by Broca, we have apraxia of speech without oral–facial apraxia (at least no involvement of the tongue).

A fourth description added a "nonprotrusion" of the tongue upon VERBAL request. J. Jackson (1866/1931a) wrote,

> In some cases of defect of speech the patient seems to have lost much of his power to do anything he is told to, even with those muscles that are not paralyzed. Thus, a patient will be unable to put out his tongue when we ask him, although he will use it well in semi-involuntary actions, e.g. eating and swallowing. (p. 131)

He added, "He will not make the particular grimace he is told to, even when we make one for him to imitate" (p. 121). Twelve years later, Jackson (1878/1931d) again wrote, "It will have been noticed by every medical man that some patients who have loss or defect of speech do not put out the tongue when they are asked" (p. 153). Jackson, unlike Broca, was describing an apraxia of speech *with* oral–facial apraxia. Jackson was the first to stress that the disorder was one of volitional movement; he believed that encoding a propositional message was highly volitional, as was the protrusion of the tongue on auditory–verbal command.

An intriguing ambiguity developed at this point in the history of the study of apraxia. It created a lingering tension that remains with us today in the cognitive neuropsychology of movement systems. The patient who, to the verbal command, was unable to grimace or to protrude the tongue for Jackson, might have had a lesion disconnecting or disengaging both the area for comprehending language and the area for outputting motor commands. The patient would not necessarily have any lesions in the motor zones of the frontal lobes. On the other hand, the lesion could be present in the frontal cortical motor zones. In this case, disconnection would not serve as the crucial element of explanation, but rather the explanation

would rest at the motor level. The disconnection hypothesis strictly involves LANGUAGE as a stimulus condition; the other does not. Therefore, the mechanisms would be quite distinct and in general the abnormal behaviors would be tightly linked to the nature and modality of the stimulus conditions. The non-disconnection hypothesis emphasizes a direct and primary breakdown in motor planning and sequencing, whereas the connectionist argument explains the apraxias on the basis of disconnection of the areas in which the command is comprehended from those areas where the command is carried out. The origins of *both* accounts of apraxia are found with Liepmann.[3]

Hugo Liepmann

Hugo Liepmann developed and applied both explanations—disconnection *and* center lesion. His "limb-kinetic" apraxia was caused by lesions in the frontal motor association areas, and his "ideational" (now claimed to be more of a "conceptual" disorder in Ochipa, Rothi, & Heilman, 1992) apraxia stemmed from damage to the sensory association zones in the posterior temporoparietal regions (Brown, 1972, 1988a; Head, 1926; Rothi & Heilman, 1996). Liepmann felt that motor aphasia was a particular form of limb-kinetic apraxia of the "glossolabiopharyngeal" apparatus; subsequently, he added the larynx to this string of anatomical structures (Head, 1926, p. 99). According to Leipmann, a limb-kinetic apraxia was characterized by a "loss of kinaesthetic memories of a definite part of the body" (Head, 1926, p. 97). The patient had "lost the power to execute certain combinations of acquired movements," and "delicate movements" were impossible (Head, 1926, p. 98). Accordingly, Liepmann, was extending the notion of "limb" to the speech articulators. It is for this reason that he is often credited with being the first to formulate the notion of APRAXIA OF SPEECH (Rosenbek, 1993, p. 444).

Liepmann's system contained three levels: the "movement formulae," the "innervatory patterns," and the "kinetic memories" (Rothi & Heilman, 1996). Kinetic memories are similar to Bouillaud's memories for the procedures of articulation. The movement formulas for Liepmann were space–time coordinates, essentially sensory and most commonly visual, except, of course, for speech (Square, Roy, & Martin, 1997, p. 6). Liepmann

3. The anatomical connectionism of Meynert, Wernicke, and Liepmann that was rekindled by Geschwind (Buckingham, 1984) has little if anything to do with the so-called "connectionist" arguments of parallel distributed processing or of the interactive activation models so popular today in the cognitive sciences (Caplan, 1994, p. 1033, fn. 5). The only link that I can see between the two is their shared affinity with the basic tenets of the centuries-old association psychology (Buckingham, 1984, 1999).

felt that the movement formulas were mediated somewhere at the interface of the left temporal and parietal lobes (see Sirigu et al., 1996, for some recent neuroscientific evidence that Liepmann was not far off the mark). His innervatory patterns allowed the transcoding of the formulas into the positioning of the limbs "according to directional ideas" (Rothi & Heilman, 1996, p. 116). Actual movements laid down "kinetic memories," which according to Liepmann were properties of sensory–motor cortex and required high frequency of use in the formation of familiar movement associations.

Liepmann drew up a typology of praxic errors: perseveration of whole movements or of parts of movements, movement substitution, spatial disorganization, movement delay or protraction, and movement incompleteness or distortions. Each of these has a counterpart for speech movements. Anatomically, there was a kind of continuum for Liepmann. The further posterior a lesion went, the more likely that error types would reflect misselection of the movement memory, whereas more anterior lesions would bring about "amorphic movements," which were understood as mutilations of the correctly selected movement plans.

Liepmann's Links to Modernity

Norman Geschwind (1965) never embraced the classical grouping of the apraxias that Liepmann advanced. The categories of limb-kinetic, ideomotor, and ideational, for Geschwind (1965, 1974), were not very useful and frequently proved confusing. The limb-kinetic disorder, and therefore Liepmann's limb-kinetic speech apraxia of the glossolabiophrayngeal apparatus, was "not defined clearly enough to separate it from mild pyramidal disturbance" (Geschwind, 1974, p. 201). The modern-day, neo-Geschwinians still feel this way, and therefore have been predictably reluctant to label any aphemic-like disorder as an apraxia (e.g., Schiff, Alexander, Naeser, & Galaburda, 1983). Many French aphasiologists prefer to use Pierre Marie's term ANARTHRIA, but they do not appear to be overly preoccupied with others' use of "apraxia" to describe it (Lecours, Lhermitte, & Bryans, 1983, p. 296).

Nowadays, however, much of the zeal and furor with which the anatomical connectionist accounts were proposed has subsided. There is growing evidence from the neurosciences that a strict, anatomical connectionist paradigm is seriously flawed and in need of radical rethinking and resynthesis (e.g., Deacon, 1989). There are, nevertheless, several investigators who cast their findings in more or less connectionist formulations (e.g., Gazzaniga, 1989; Heilman & Rothi, 1993; Rothi, Ochipa, & Heilman, 1991; and many, but not all, papers in Rothi & Heilman, 1997), and it often turns out that these investigators were influenced greatly by the late Norman

Geschwind. The recent death of Geschwind has no doubt taken the wind out of the sails of cerebral connectionism, as he was a vigorous, productive, and articulate defender and docent of this model of brain and behavior (Schachter & Devinsky, 1997).

When Liepmann discussed limb-kinetic verbal apraxia, he did not refer to the stimulus-specific language command, but rather invoked the Jacksonian notion of volition as the key explanatory device (Paillard, 1982). That is, the patient could be said to be apraxic in his or her speech behavior even though no one had provided a verbal command to speak. Accordingly, that type of apraxic behavior would not be fastened to a verbal command stimulus at testing. Note, too, that the upper limb arm and hands function in transitive movements and intransitive movements, whereas the speech limbs operate intransitively—they move through time toward targets in vocal tract space, but they do not manipulate objects when so doing. Articulation is by its very nature intransitive.

Apraxia of speech does not appear to be restricted in any diagnostic manner to certain specific stimulus conditions. Nowhere that I am aware of has it been recorded that patients with apraxia of speech are apraxic when repeating but not, say, when naming objects, reading aloud, or speaking spontaneously. Although the original study by Johns and Darley (1970) elicited much of the data by asking patients to repeat, it is doubtful that these patients spoke normally under any other conditions except for automatisms and for those short stretches of "islands of error free production."

Finally, Liepmann (1900), aside from a supramarginal gyrus ideokinetic apraxia (the famous case of the *Regierungsrat*), described a "sympathetic" ideokinetic apraxia of the left limbs arising either from callosal disconnections in the anterior regions or from left prefrontal lesions. Both of these anatomically disengage the dominant left hemisphere language zones from the right hemisphere motor areas that move the left hand (Rothi & Heilman, 1996).

Before leaving the earlier studies of apraxia, it is instructive to look at how Head's discussion of the issue extended the dual interpretation of apraxia. When writing about Liepmann and apraxia in his chapter entitled "Chaos," Head (1926) begins the section with the clear implication that apraxic behavior is secondary to a linguistic command, such as "told to protrude his tongue," "show his teeth to command," and "execute such movements to order" (p. 93). In Head's treatment, the label APRAXIA is directly linked with the language stimulus condition as an essential component, and accordingly it would be subject to a connectionist explanation. However, by page 95 in his discussion, we find out that apraxia of speech is a disorder with the "higher mechanics of verbal formulation" (p. 95). Because formulation is involved, it is now possible to consider the stimulus

condition as self-induced on the part of the speaker. The "will" is invoked at this point. Once volitional intentionality comes to the foreground, that very willful act may serve as the stimulus condition. Consequently, the disconnection explanation would no longer be necessary. As we shall see, this is exactly what happened with developments in apraxia of speech and the concomitant nonconnectionistic explanations in the experimental work (e.g., Katz, 1988a; Rosenbek, McNeil, & Aronson, 1984; Square-Storer, 1989; Tuller & Story, 1987, 1988; Ziegler & von Cramon, 1985, 1986).

Ample historical precedent exists for two quite distinct explanations for movement disorders, which are both referred to as apraxia. One explanation leads to the acceptance of an apraxia of speech; the other does not. As a consequence, one's position on what constitutes an apraxia in general largely determines whether it even makes sense to talk about an apraxia of speech. Furthermore, there appear to be two ways to read the word *apraxia*. This Greek word is composed of two morphemes: *a*, meaning "without" and *praxis*, meaning skilled movement or action. The word itself is vague, as a person can be "without skilled movement" in two ways. The first way is to be without movement because the movement patterns, formulas, programs, or whatever cannot be driven either from the language command system or from some higher intentional level that would otherwise drive them. The two are disengaged. In this case, praxis is fine; it simply cannot be accessed. The result is still "without skilled movement"—*a + praxia*. The disengaged movements are often described as "bizarre" and do not appear to be driven by the patient's faulty motor system as much as by the patient's inability to engage the movement system. It would make no sense to study the dynamics of the bizarre, disconnected movements with an eye toward understanding any elementary movement disorder; there simply *is no* disorder. Another way to be "without skilled movement" would be brought about by direct compromise or disruption of those movement patterns, formulas, programs, or representations themselves. In that case, the praxis is *not* fine. These two readings of *apraxia* are directly related to the distinction between disconnection lesions and center lesions.

Current Stands

The Mayo School

One contemporary position on apraxia of speech is often referred to as the "Mayo School" position. The influence of F. L. Darley (Sasanuma, 1989), Arnold Aronson, and J. R. Brown is well known. Numerous researchers have been extending their views (Duffy, 1995; Johns and LaPointe, 1976; Mlcoch & Noll, 1980; Rosenbek, 1984; Rosenbek, McNeil, &

Aronson, 1984; Square et al., 1997; Square-Storer, 1989; Wertz, LaPointe & Rosenbek, 1984). Their claim is generally that this syndrome is often found among the symptom complex of Broca's aphasia, but at times it is dissociated from the latter and can be seen in a more or less "pure" form. Dronkers (1996), through a comparison of computerized lesion overlapping in patients with and without apraxia of speech, found that 100% of those who had apraxia of speech had lesions that included the precentral gyrus of the left hemisphere insula, which would not have surprised Freud (1891/1953, p. 12). It is not surprising that several cortical regions are involved in articulatory anomalies that are neither dysarthric nor purely phonological. Recently, more attention is being paid to the role of the parietal lobe in speech apraxias (Buckingham, 1983; Buckingham & Yule, 1987; Kimura & Watson, 1989; Square et al., 1997). At times it has been claimed that an apraxia of speech is not a language problem at all, which is the logical extension of separating apraxia from aphasia. A purely motor view would adopt Broca's postulated (but ultimately not chosen) explanation that the "aphemic" disorder is primarily motoric, not a higher level problem of memory or intelligence. The Mayo School in general would agree with Liepmann that what is involved is a type of limb-kinetic apraxia of the glossolabiopharyngeal musculature. In addition, the Mayo position separates apraxia of speech from oral–facial apraxia, although like most others (e.g., DeRenzi, Pieczuro, & Vignolo, 1966), the proponents are well aware that the two most often occur together.

Another proposal may be regarded as the result of a reaction to the early positions of the Mayo School that every nondysarthric articulation problem is an apraxia of speech. This second proposal makes a clear distinction between frontal articulatory disorders and posterior articulatory disorders. The work reported in Canter (1973), Trost and Canter (1974), and Burns and Canter (1977) showed that the nature of the articulation parameters in anterior versus posterior patients is readily distinguishable. An apraxic, nonfluent character is seen in the frontal lobe subjects, whereas the temporoparietal lesion patients show a fluent and well articulated phonemic disorder in which, for instance, all allophones of substituted phonemes have their predicted phonetic shapes. This second current proposal brings to bear not only neuroanatomical questions, but also LINGUISTIC questions. Controversy now surrounds determining precisely what is and what is not phonemic, especially with the resurgence of observations of speech apraxias following left parietal lesions (Square et al., 1997).

Neurologists and Neuropsychologists

Luria (1973) is reminiscent of Liepmann in his views of apraxia. He equated Broca's aphasia with a kinetic apraxia of speech, which he referred

to as an efferent motor aphasia. To this he added a kinesthetic apraxia of speech, which he took to be a sort of "afferent motor aphasia." He correlated the phonological paraphasias of sensory aphasics with an ideational apraxia. It turns out, however, that Luria acknowledged only center-lesion explanations for apraxic behavior. Luria (1973) thus described the speech problems secondary to lesions in the inferior postrolandic zones of the parietal lobe:

> If a lesion of the secondary (kinaesthetic) zones of the post-central region affects the lower zones of this region of the left (dominant) hemisphere, i.e., the region of secondary organization of kinaesthetic sensation in the face, lips and tongue, the kinaesthetic apraxia may manifest itself in a special manner in the organization of movements of the speech apparatus, leading to the distinctive disorder of speech which has been called afferent motor aphasia. (p. 174)

Luria, as Liepmann, viewed any voluntary movement as mediated by a "complex functional system" involving ideation, kinesthetic afferentiation, and kinetic organization. Each of these organizational levels has its anatomical substrate, and one or another type of apraxia will arise secondary to lesions in the respective areas. Movement parameters for speech are disturbed in various ways depending on the localization of damage along the posterior to frontal axis. The articulatory aspect, therefore, of the different types of aphasia will be characterized as one or another type of "apraxia of speech," from ideational to kinetic.[4] The ideational level of apraxic behavior is much closer to the level at which we characterize the more abstract aspects of the phonological sound systems of human language, and if there is an apraxia here, logic would lead to the oxymoron of "apraxia of language" (Buckingham, 1983).

Jason Brown (1972), not unlike Luria, postulated "interference at comparable stages in microgenesis of speech and movement" (p. 198). Brown wrote that "disorders of movement, like those of speech, are disturbed in a posterior–anterior fashion" (p. 195). Worded differently, "the movement complex passes from a conceptual to a motoric form, undergoing a progressive differentiation comparable to that which occurs in the speech system" (p. 195). Subsequently, Brown (1975a, 1975b, 1977, 1988a) refined his notion of the microgenesis of action (Hanlon, 1991). For Brown (1975a), "the term microgenesis has been proposed for the continuous formative activity which underlies cognition" (p. 26). For instance, he wrote (1977),

4. One could certainly argue that "ideation-to-kinetic" feedforward microgenesis schemes come dangerously close to Jackson's (1878/1931b) edict against the direct mind–brain leap taken in models that allow psychic states to fine away into physical states. This parallels the problem alluded to in footnote 1 of going from phoneme to articulation (Kelso & Tuller, 1981, p. 227), and is in general part of the extremely complex issue of mental causation (see F. Jackson, 1996).

"Both facial and limb apraxia can occur with frontal and temporoparietal lesions. In this respect, they are comparable to phonemic paraphasia, which also occurs with anterior and posterior pathology" (p. 72). At the phonemic level, the anterior versus posterior differences appeared to be quantitative, not qualitative, just as Poeck and Kerschensteiner (1975) found for oral apraxia. Similarly, De Renzi et al. (1966) wrote, "Oral apraxia characterizes patients whose speech productions may be very different from one another" (p. 68).

Brown (1975a) acknowledged center lesion apraxias rather than disconnecting lesion apraxias, but for him the centers of traditional neuropsychology were best conceived as LEVELS by means of which cognition is carried one stage further. White fiber pathways, for Brown, "do not serve to associate ideas, perceptions of movements, written words to spoken words, etc., but rather link up temporally transformations occurring at different points in the microgenetic sequence" (1975a, p. 29). Brown proposed his dynamic microgenesis model as an alternative to disconnection theory. His discussion of conduction aphasia (1975b) is instructive. Speech paraphasia and movement apraxia represent disturbances at comparable levels in the actualization of speech and movement. Because speech is a form of movement, the two collapse into one, and thus, for Brown, conduction aphasia is an "ideomotor apraxia of speech." Brown (1972, chap. 10) also draws an important analogy between ideational apraxia and posterior fluent paraphasic speech in Wernicke's aphasia. He writes:

> We may almost speak of ideational apraxia as a 'fluent' apraxia, contrasting it with 'non-fluent' apraxias of anterior origin. In ideational apraxia there is an abundance of partial movements, each normal in itself, and the overall movement sequence, though disorganized, has an ease and an effortless quality as is in the speech of posterior aphasia. (p. 170)

Turning away from Brown's microgenetic model, we now focus specifically on apraxias secondary to supramarginal gyrus lesions (Brown, 1972; Denny-Brown, 1958; Geschwind, 1965; Mateer & Kimura, 1977). Predictably, those who acknowledge center-lesion explanations have considered supramarginal gyrus apraxia of speech. This gyrus is a crucial zone for the discussion of apraxia in general AND of apraxia of speech. Although it is a cortical area of the parietal lobe strategically located among the language regions of the perisylvian rim, it is also anatomically quite near the arcuate fasciculus fibers, which are subjacent to it and which travel through the opercular zones (Galaburda, 1982). The plenum of the inferior parietal lobule is also quite near this cortical region, its motoric physiology having been laid out clearly by Galaburda (1982). That some form of praxic malfunction should follow lesions of this zone is therefore not surprising. Some have preferred to call the repetition disturbance of conduction apha-

sia an "ideokinetic apraxia for the formation of sounds" (Kleist, 1916). Recall that center-lesion theories of apraxia are predicated upon the assumption that memories (or representations) for movements are stored in those centers. As mentioned earlier, the age-old notions of memories for movements are now often referred to as kinetic memories. Computer-age metaphors would include the phrase "movement programs."

Denny-Brown (1958) is also representative of those neurologists who emphasize posterior center-lesion theories of apraxia of speech despite the fact that his student, Geschwind, developed connectionist accounts. He claimed that

> Although the complex movements of lips, tongue and larynx show disturbances that are apractic in nature, these occur in isolation from other types of body apraxia, as if the praxis of speech had developed independently in the dominant insula and parietal operculum, more removed from the parietal–occipital region concerned in other types of ideation apraxia. Thus apraxia of the tongue is a special and particular variant of motor apraxia, associated most commonly with executive aphasia, and usually dissociated from apraxia of facial expression. (pp. 12–13)

Denny-Brown made no mention here of a relevant stimulus (the linguistic command). Again, disregarding relevant stimuli is an earmark of center-lesion theories of apraxia. It should be pointed out, however, that by 1965, Denny-Brown had shifted his focus to frontal lesions (Broca's aphasia) and consequent speech disturbances. He described (Denny-Brown, 1965) a Broca's aphasic with lesions in the left third and part of the second frontal convolution. The patient (a) had difficulty initiating words, (b) had occasional substitution and anticipation of segments and syllables, (c) had a "slowing of rhythm," and (d) showed great variability. Denny-Brown concluded, "This particular difficulty is associated with varying degrees of apraxia of the tongue, lips, face, and respiratory control" (p. 462). He still acknowledges center-lesion apraxias, but now the region involved for apraxia of speech is in the dominant frontal lobe instead of in the insular and parietal opercular zones described in the 1958 paper.

In a series of articles (Kimura, 1976, 1977a; Kimura & Archibald, 1974; Mateer & Kimura, 1977), Kimura suggested that "lesions of the left hemisphere impair the performance of complex motor sequences, regardless of whether the sequences are meaningful or not" (Kimura & Archibald, 1974, p. 346). On this view, speech disturbances and apraxia are simply distinct manifestations of a breakdown in the control of the action of motoric sequencing. Kimura believes the actual sequential control system is in the parietal lobe (Kimura, 1976; Kimura & Watson, 1989). Kimura (1980) has no doubt been much influenced by Liepmann's early notions. Her theory is that the movements need only be complexly coordinated, not necessar-

ily meaningful or symbolic. The reasoning has been extended to her hypothesis that "the left hemisphere is particularly well adapted, not for symbolic function per se, but for the execution of some categories of motor activity which happen to lend themselves readily to communication" (Kimura, 1976, p. 154).[5] Kimura subsequently characterized apraxia as "a failure to achieve target motor responses when more than one is required rather than just an improper ordering of those targets" (Mateer & Kimura, 1977, p. 274). Similarly, Kimura (1977b) stated that her analysis of those apraxic errors following left posterior lesions reveals that the principal difficulty rests, not with ordering the movements, but rather with selecting and/or executing new postures, whether of brachial or oral musculature (Kimura, 1982; Kimura & Watson, 1989).

Geschwind's disconnection model (Geschwind, 1974; Schachter & Devinsky, 1997) is stimulus specific—the apraxic behavior is secondary to a verbal–linguistic command only. Geschwind ran into difficulties, however, in accounting for those patients who are apraxic to imitation as well, since, as he often claimed (1975), "We are led to expect that the patient[s] will respond correctly to nonverbal stimuli, which can reach the motor regions without going through the speech areas" (pp. 190–191). Geschwind (1975) also wrote, "Clearly some factor other than disconnection between language and motor areas seems to be necessary to account for these findings" (p. 191). This other factor invoked by Geschwind (1975) is that of cerebral dominance for limb (arm, hand) movement that usually is, but may not always be, in the same hemisphere for speech (Heilman, 1979; Heilman, Coyle, Gonyea, & Geschwind, 1973; Heilman & Rothi, 1993; Rothi & Heilman, 1997). The addition of motor dominance to Geschwind's disconnectionism does not necessarily compromise his model, as the dominant hemisphere can be said to "lead" or "drive" the nondominant hemisphere in many instances. Heilman et al. (1973) and Rubens, Geschwind, Mahowald, and Mastri (1977) proposed models that incorporate dominance theory AND disconnectionism. The earlier sine qua non explanatory nature of the verbal language input is accordingly weakened. Heilman and colleagues have more recently shifted away from an exclusive disconnectionist account to include a "representational hypothesis" as well (Heilman, Rothi, & Watson, 1997). For them:

> Right-handed apraxic patients with left parietal lesions cannot correctly gesture to command, imitate, or use actual objects and tools because they have either

5. Kimura's position is that human language articulation is a parasitic function scaffolded evolutionarily over primordial neural architectures of early humans, architectures that were initially involved with the control and timing of sequential movement (Calvin, 1983; Code, 1998; Corballis, 1991; Deacon, 1989).

> destroyed the movement representations stored in the left inferior parietal lobule or disconnected them from premotor or motor areas. (p. 176)

Neo-Geschwinians still eschew mention of SPEECH apraxia. Note that in this quotation, the writers include gesturing to command and imitation or use of actual objects or tools. There is *no* mention of speaking.

Center Lesion Theories versus Disconnection Lesion Theories: An Evaluation

Johns and LaPointe's (1976) theoretical assumption is that there is an apraxia of speech, and it is a type of limb-kinetic apraxia for the speech musculature—essentially Liepmann's view of that type of apraxia. Their literature survey and in-depth discussion of terminological confusion focuses almost entirely on limb-kinetic, center-lesion apraxia of speech and the plethora of nomenclature it has engendered throughout the history of aphasia. Their article is an extension of Darley (1967, 1968) and of John's doctoral dissertation research. It is cast in the framework of center-lesion theory. At different times over the past 100 years or more, this frontal lobe apraxia of speech has been referred to as "aphemia, Broca's aphasia, motor aphasia, anarthria, verbal aphasia, phonetic disintegration of speech, apraxia, apraxic dysarthria, cortical dysarthria, and oral verbal apraxia" (Johns & LaPointe, 1976, p. 163).

In 1965, at a National Institutes of Health conference on brain mechanisms underlying speech and language (Millikan & Darley, 1967), Darley was "asked to reflect as a speech pathologist on the impact of this conference"; he responded that the term *motor aphasia* (or the Broca type) would be "better labeled an apraxia, to be exact, an oral verbal apraxia" (Millikan & Darley, 1967, p. 236). He also lamented that "our terminology needs disciplining" and that "perhaps we need a kind of International Standards Organization for aphasia terminology and classification" (p. 237). Darley said that "to do this kind of critical review surely requires unusual courage and scintillating scholarship, and who is good enough and brave enough to do it?" (p. 237). Benton's (Millikan & Darley, 1967) reaction to Darley's comments is instructive: "I could not agree more with Dr. Darley about the sad state of terminology and classification in the field of aphasia. I doubt there would be any disagreement among us. However, I shudder at the thought that these questions will be decided by an international commission, either by an elite or by a democratic process; really we would be carrying democracy too far" (p. 239). Three years after this conference, Darley presented a paper to the American Speech and Hearing Association (1968) entitled, "Apraxia of Speech: 107 Years of Terminological Confusion" (Rosenbek, 1993, p. 445). An examination of the numerous descriptions of limb-kinet-

ic apraxia of speech, under all of the diverse labels, nevertheless reveals that reasonably good clinical agreement has always existed. There is never much argument with the ACTUAL descriptions of the articulatory output.

There are, however, a few places in Johns and LaPointe's (1976, pp. 186, 191) review where they mention disconnection phenomena and refer to another center-lesion type of apraxia of speech. When discussing the DeRenzi et al. (1966) findings concerning the possible (but not often obtaining) separation of limb-kinetic apraxia of speech from oral–facial apraxia, they compared the two possible anatomical explanations suggested in that paper. One is that oral nonverbal movements and oral verbal (speech) movements could be mediated by two distinct frontal cortical association areas. The second consideration of DeRenzi et al. (1966), however, as Johns and LaPointe (1976) wrote, is "that apraxia from commands can result from lesions to pathways connecting frontal and temporal lobes; and apraxia from imitation can result from lesions disrupting occipital and frontal fasciculi" (p. 186). Later in their review, they noted that Jay Rosenbek, one of the most influential clinicians and researchers of the Mayo tradition "revised the concept of apraxia of speech to include sensory-perceptual influences rather than the traditional view of it as strictly a motor, or output, speech disorder" (1976, p. 191–2). What Rosenbek essentially proposed was that Luria's "afferent apraxia" could be an added component to limb-kinetic apraxia.

Johns and LaPointe (1976) did not include discussion of Kimura's work on posterior apraxia of speech or of the whole issue of disconnection explanation in apraxia. Kimura (e.g., 1982) denied that frontal lesions result in anything more than weakness, slowness, and incoordination of movement. For her, complex praxis for movement is a dominant POSTERIOR function of the parietal lobe. She is in agreement with Geschwind (1965), who wrote that "'limb-kinetic' apraxia has not been defined clearly enough to separate it from mild pyramidal disturbance" (p. 617). Kimura (e.g., 1982) and Geschwind (e.g., 1974) challenged the Mayo position that aphemia is an apraxia, but early on neither Darley nor others of the Mayo school paid them much heed. For example, there is no reference to Geschwind's work on apraxia in Johns and Darley (1970), a seminal paper on the Mayo position. This, in turn, probably explains why there is so little discussion of disconnection mechanisms in the follow-up publication by Johns and LaPointe (1976). The recent work of Square et al. (1997), however, evaluates both Kimura's and Geschwind's contributions.

The testing for limb apraxia does not easily translate into tests for (articulatory) limb apraxia (apraxia of speech). Commands such as "show me how you would talk," "imitate what I do" (experimenter talks), and so on, sound droll. As pointed out earlier, all articulatory limb movements are intransitive. Unlike the hands, the articulators do not use objects when they go about their skilled movements. With what objects do the articulatory

limbs move better? In this sense, speech is ALWAYS a pantomime of sorts, with exclusive intransitive movement. Articulate speech is largely out of visual range and is composed of coordinated, synchronized, and sequential movements of the articulating limbs through time and space for the achievement of gestural–acoustic targets, all of which is significantly unlike the movement of the upper limbs (also see Square et al., 1997, pp. 5–6). When one runs through a list of "praxis tests" of the neo-Geschwinians (e.g., Heilman et al., 1997), one rarely if ever finds a test that would make sense to administer for speech.

The theories of Kimura and Geschwind, although founded upon different theoretical bases, have challenged the claim that aphemia, anarthria, phonetic disintegration, or whatever, are articulatory manifestations of an APRAXIA. Kimura believes that only left parietal lobe lesions can cause apraxia of speech, and the insouciant neo-Geschwinians claim that they "do not know what an apraxia of speech is" (K. Heilman, personal communication, October 30, 1996). Schiff et al. (1983) also typify the Boston School's position. After a detailed study of clinical–anatomic correlations of what they prefer to call *aphemia*, they write:

> Some authors favor the concept and term "apraxia of speech." These investigators . . . have provided an operational definition of apraxia, but there is an overall tendency to use the term without further clarification, as the descriptive one for any and all supramodal motor disturbances. (p. 726)

They go on to stipulate the basic neo-Geschwinian diagnostic for an apraxia: "We favor reserving it for learned motor actions that are impaired in some settings but not in others" (p. 726).

In any event, to avoid ambiguity, when characterizing the nonphonemic and nondysarthric speech disruption secondary to brain damage, one must specify the account being used (center-lesion vs. disconnection), the location and extent of the lesion, the stimulus-testing conditions that do and do *not* evoke the speech movement disorder, and, most importantly, the phonetic anomalies that are present. What are these phonetic characteristics?

General Characteristics of Limb-Kinetic Apraxia of Speech

Patients with a limb-kinetic apraxia of speech have a set of typically observed articulatory anomalies (Duffy, 1995; Rosenbek, 1993; Square et al., 1997). In attempting to articulate, these patients often show a groping behavior, which indicates that they have the underlying phonological schemata in mind. Although there are difficulties with initiating the artic-

ulated sequence, successful articulatory preposturing quite often reveals the vocal tract configuration for the initial segments of the word to be uttered. Limb-kinetic apraxics' disturbances often are exacerbated on elicitation, which relates to the issue of volitional level on confrontation testing.

A typical catalog of the phonetic aberrations recorded in patients said to have apraxia of speech would include

1. Awkward phonetic modulations of vowels and consonants (distortions);
2. Syllable stress errors and equalization of stress, which in turn affects any phonological process that is sensitive to stress, such as vowel reduction and flapping;
3. Initiating articulation and delayed verbal response times;
4. Smooth transition from one sound to the next;
5. Abnormal elongation of fricative and sonorant consonants;
6. Abnormal stopgap durations;
7. Abnormally lengthened vowels, syllables, and CV units;
8. Amplitude and fundamental frequency abnormalities;
9. Asynchronies and uncoupling of upper and lower articulatory systems that affect proper voice-onset time (VOT) parameters and oro/nasal productions;
10. Overly long intersegmental durations;
11. Difficulty adjusting speech rate.

These abnormalities are caused by a variety of physiological dysfunctions, where distorted, variable, and dyscoordinated movements are the rule.

There are some interpretive difficulties with the linguistic and nonlinguistic diagnostic characteristics of limb-kinetic apraxia of speech. PROGRAMMING, INCOORDINATION, and VARIABILITY are three terms that have caused some confusion and in general have not differentiated syndromes very well. INITIATION, SELECTION, and SEQUENCE have not been sharply enough defined, and the term PHONEME has caused confusion because of its abstract nature. Some researchers have cast aspersions on the phonemic–phonetic distinction (Rosenbek, 1993), claiming that the distinction is "simplistic" and that "making it requires the highly subjective interpretation of the data" (Rosenbek, 1993, p. 448). Nevertheless, as long as the "target" must be specified, the phoneme will be around for some time—even cloaked as the "gestureme," or as some kind of "abstract gestural structure" (Mattingly & Studdert-Kennedy, 1991). It is the "—eme" that keeps the notion an abstract one (Pike, 1954). The "—etic" and "—emic" distinction is disregarded at one's own risk.

If an apraxia of speech involves disorders in the "programming" of motor speech, we need to know what the units of speech are that are involved in the programming and precisely where we are in the encoding process.

One should not lose sight of the fact that PROGRAM is a type of memory metaphor, which may be used as a descriptor at any level of production, and that the term itself is simply the computational version of kinetic memory, movement formulas, and the like.

It is also important to distinguish between a lower level "incoordination" of the musculature used for speech and a higher level "incoordination" of the set of synchronous nerve impulses that eventually impinge on different muscle groups for properly timed and interlocked articulatory events. Rare cases of isolated apraxias of phonation have been reported, which are not muscular, but rather are neural (see Hoole, Schroter-Morasch, & Zeigler, 1997; Marshall, Gandour, & Windsor, 1988). There may even be an apraxia of swallowing (Daniels & Foundas, 1996; Tuch & Nielsen, 1941).

VARIABILITY OF BEHAVIOR is another overly general descriptor. It may be used to characterize, albeit it different ways, practically any aphasic or apraxic. Variability has recently been used as a metric to distinguish apraxia of speech from conduction aphasia. The claim is that phonological disruptions do not engender the degree of acoustic variability in the speech signal as do the motor disruptions in apraxia of speech (e.g., Seddoh et al., 1996). Presumably, this term came into use originally to differentiate apraxia from dysarthria, the latter being less variable in terms of articulatory success. The variability in apraxia is explained differentially. The connectionist attributes the variability to the different testing and stimulus conditions under which a patient is requested to do something. The patient often is able to enact a movement pattern otherwise performed apraxically to verbal request. Limb-kinetic speech apraxias also vary along the lines of the occasional "islands of error-free productions" that are thought to be more automatic, less propositional, and therefore less volitional.

Canter, Burns, and Trost (1975) suggested a useful distinction for getting around the preceding problems with the vague term SEQUENCING. They differentiated between "transitionalizing" from articulatory unit to articulatory unit and the "sequential ordering" of phonemes. Consequently, the essential problem of posterior patients is with a higher level sequential "ordering," whereas the problem of the speech apraxic is with sequential "flow."

Experimental Studies

Voice-Onset Time

A major phonetic–acoustic cue for perception of the linguistic distinctive feature [+/−voice] for syllable initial oral stop consonants in English

is the time interval between the release of the consonantal closure (that follows on the heels of the stopgap, whose duration can also be measured) and the onset of vocal fold vibration (cords adducted) at the larynx; this is the VOICE-ONSET TIME (VOT). Various neuroanatomical regions have been implicated in the control of properly synchronized articulatory gestures that produce the required VOT parameters for a given language. The regions often involved include the anterior language zones, the cerebellum, and the basal ganglia (Ackermann & Hertrich, 1997). Many experts feel that different sorts of pathomechanisms could engender similar VOT asynchronies, and others have found different values and ranges for distinct types of brain disorders (Blumstein, Cooper, Goodglass, Statlender, & Gottlieb, 1980). Generally speaking, the closer the voice onset occurs to the time of release, the more likely the consonant will be filtered through the phonological system as [+voice] for a language such as English. The parameters are language dependent, and there will always be a quantal (Stevens, 1989) range of values of onset times where variation within the range causes no perceptual differences for hearers.

A study by Blumstein, Cooper, Zurif, and Caramazza (1977) comparing anterior subjects with posterior subjects on voice-onset timing parameters showed a "lack of synergy" for the former group. Although at times the anterior subjects will produce phonemic substitutions, this group produces asynergic phonetic substitutions that in several instances turn out to be unexpected, incorrect ALLOPHONIC productions for what presumably are correctly selected phonemes. In the case of /t/, for instance, the anterior patient is likely to substitute the allophone of /t/ that normally occurs after /s/, as in [stap], in syllable initial position.

Sands, Freeman, and Harris (1978) followed the improvement over a 10-year period of a patient with a limb-kinetic apraxia of speech. They found that, at the end stage, errors of place and manner of articulation as well as deletion errors were greatly reduced. What persisted were essentially errors in voicing, and thus it was concluded that the remaining apraxic disturbance was one of temporal coordination of the abductory and adductory laryngeal processes with upper articulatory gestures.

Freeman, Sands, and Harris (1978) followed up with a more detailed study of VOT in apraxia and found, not surprisingly, that it differed markedly from productions in non–brain-damaged normals. They noted that apraxic articulations did not include voicing lead for voiced stops and that lag times for voiced stops were longer than normal. On the other hand, the lag times for voiceless stops were shorter than normal. All of these apraxic changes resulted in more closely compact VOT ranges around the point of stop release. Consequently, there was a clear overlapping of [+voice] and [−voice] perceptual categories for the three sets of homorganic oral stops. Apraxics produced little or no prevoicing leads or long

lags. Finally, the authors noted that the apraxics' VOT range constriction to the short lag area (+20 to +30 msec) mirrors VOT ranges in young children, but they offered no explanation for this.

Kewley-Port and Preston (1974), as discussed in Cooper (1977), offered an account of VOT acquisition in children in terms of complexity of neural control. In the first place, vocal fold vibration is brought about by three factors: (a) folds must be adducted; (b) they must be relatively relaxed; and (c) a sufficient drop in pressure across the larynx must be maintained in order to permit an accelerated airflow. The neural control for these three factors is different for the three canonical VOT types: prevoicing, short lag, and long lag. The short-lag VOT is neuromuscularly less complex, because, unlike with prevoicing and long lag, factor (c) is mechanically produced. For short-lag VOTs, the speaker need only adduct the folds prior to release of stop closure and keep them relatively lax. Upon release of closure, a sufficient drop in transglottal pressure takes place automatically due to the equalization of mouth pressure. This enables initiation of glottal vibration shortly after the release. For prevoicing or long-delay VOT, the maintenance or delay of cross-glottal pressure drop requires more complexly timed and controlled neural commands. Eventually, the child comes to master the more complex neuromuscular coordination. It appears that the apraxic has lost this neuromuscular coordination (Wambaugh, West, & Doyle, 1997).

Foreign Language Accent Syndrome—Apraxia?

Incorrect allophonic production may, in some cases, sound like a foreign accent to some listeners. In fact, one characterization of a foreign accent is precisely that nonnative speakers often produce some inappropriate phonetic variant of the target phoneme for the language they are speaking (Ardila, Rosselli, & Ardila, 1988; Blumstein, Alexander, Ryalls, Katz, & Dworetzky, 1987). Whitaker (1975) described a patient with left frontal damage whose speech output gave the partial impression of being Spanish in origin, whereas Ardila et al.'s (1988) patient was a Spanish speaker whose speech gave the impression of being English in origin. Whitaker's patient had characteristics of Broca's aphasia with apraxia of speech. This patient was from central Michigan, had not been outside the area, and had never studied a foreign language. Whitaker (1975) wrote, "There were striking problems with aspiration; initial voiceless stops were often unaspirated" (p. 27). One would suspect that the patient was having difficulties with temporal coordination, although no acoustic measurements were taken. Other articulatory characteristics of this syndrome were clearly not phonemic in nature and gave the impression of being ataxic. This type of subphonemic foreign accent output secondary to frontal rolandic

area lesions has been described by several others as a cortical dysarthria (Whitaker, 1975, pp. 23–25). Moen (1996) described Monrad-Krohn's (1947) first recorded case of the syndrome, which was characterized more by disturbances of prosody (pitch accent and intonation) than by segmental alterations. Nonetheless, from the extant descriptions of this syndrome, it is safe to say that there is an apraxic component to it.

Dysarthria versus Apraxia

Phonetic distortions are less variable in the upper and lower motor neuron dysarthria groups than in the cortical dysarthria group, and, unlike the motor neuron dysarthrias, the limb-kinetic apraxic patients quite often produce phonemic errors with proper allophonic realization. Blumstein et al. (1980) and Itoh, Sasanuma, Hirose, Yoshioka, and Ushijima (1980) demonstrated that dysarthric patients' productions for voiceless oral stop consonants were distributed over a wider VOT range than limb-kinetic apraxics. Broca's VOT values for these consonants rarely surpassed +150 msec. The predominant voiceless productions for the dysarthrics were abnormally long, which is why dysarthric speech at times seems overly aspirated. Furthermore, unlike the Broca productions, the dysarthric productions showed no phonetic overlap between voiced and voiceless categories. This would explain why hearers rarely attribute phonemic errors to dysarthrics. Itoh et al. (1980) clearly demonstrated that the pattern and velocity of dysarthric articulations are distinct from those of limb-kinetic apraxia of speech.

Velo-Pharyngeal Control

Another type of substitutive phonetic error can be seen in apraxia of speech. It represents a phonetic problem, but unlike the [p^h] → [p] in English, where the incorrectly substituted phone may be assigned to the same phonemic unit to which the target phone belongs, the error *must* be assigned to a different phonemic unit from that of the target, even though the phonetic error may not imply a phonemic selection error. Itoh, Sasanuma, and Ushijima (1979) studied velar movements in the speech of a limb-kinetic type of patient by fiber-optic techniques. Due to asynchronous velar movement, a phonetic change of [n] → [d] (i.e., improper lowering of the velum) was demonstrated. The phonetic error, however, is assigned to a phonemic unit distinct from that of the target phone. The phoneme /n/ does not have [d] as a possible allophonic alternation in Japanese; consequently, the phonetic error [d] must be assigned to /d/. However, as the authors pointed out, the fiber-optic measurements showed that, despite

variation in the slope of velar lowering, the pattern of anticipatory lowering was constantly preserved. The authors ruled out a phonemic selection error: "the observed variation of the pattern of velar movements and the resultant phonetic change do not stem from a selection or retrieval error of a target phoneme in the process of speech production, namely, an error of phonological processing" (p. 235). Obviously, then, the error is primarily [n] → [d], not /n/ to /d/, although the target phone and the error phone must be assigned to /n/ and /d/, respectively.

Temporal Asynchronies

Itoh et al. (1980) again measured temporal asynchronies among articulators in apraxia; this time, however, they used a different technique. By placing radioactive pellets on the lower lip, the lower incisor (for tracking jaw movement), the dorsum of the tongue, and the nasal surface of the velum, a computer-controlled x-ray microbeam system could track the simultaneous movements of these structures during articulation. This procedure further demonstrated the variability and temporal disorganization in the speech of limb-kinetic apraxics.

"Pure" Apraxia of Speech

It is certainly true that the patient with limb-kinetic apraxia of speech may or may not have a concomitant oral–facial apraxia AND may or may not have a concomitant aphasia. That is, some sort of "pure" apraxia of speech may be seen. Square et al. (1997), like many, seem to define "pure" apraxia of speech as being dissociable from an aphasia, although they most frequently co-occur. However, for a true "pure" apraxia of speech to be conceptually coherent, this kind of motor disorder with speech should be separable from any other motor involvement as well as from aphasia.

Lecours and Lhermitte (1976) demonstrated that an apraxic (for them, "anarthric") phonetic disintegration can exist in isolation from aphasia and from dysarthria. Their patient had cortico-subcortical softening of the inferior half of the left precentral gyrus, with Broca's area nevertheless intact. Although Lecours and Lhermitte called this "pure" form of phonetic disintegration "a relatively infrequent form of aphasia" (p. 109), the patient had no other aphasic symptomatology. Although some of the errors in this case were at the phonemic level, the vast majority were subphonemic.

Acoustic–Perceptual Studies of Apraxia of Speech

More recently, there has been a spate of acoustic–perceptual studies of the speech apraxias (e.g., McNeil, Liss, Tseng, & Kent, 1990; Seddoh et al.,

1996), most demonstrating a phonetic—not phonemic—disruption with the syndrome. Some (e.g., McNeil et al., 1990) have highlighted similarities between apraxia and other syndromes such as conduction aphasia, whereas others (e.g., Seddoh et al., 1996) have highlighted differences between apraxia of speech and conduction aphasia. There is still much work to be done on the true nature of the segmental speech output in conduction aphasia. In one of the earliest acoustic studies, Kent and Rosenbek (1983) published an in-depth study of the acoustic patterns produced by apraxic speakers. Like most studies of this sort, the exemplars were elicited under different stimulus–response parameters. In the Kent and Rosenbek investigation, the patients (a) repeated monosyllabic words, polysyllabic words, phrases, and short sentences; (b) produced conversational speech; (c) described pictures; and (d) read paragraphs aloud. The major conclusions from this study were the following: (a) slow speaking rates with prolongations of transitions, vocalic steady states, and intersyllabic pauses; (b) a tendency to equalize stress on vowels, which resulted in reduced vocalic centralizing and enhanced maintenance of full vowel quality; (c) slow and inaccurate vocalic and consonantal articulatory movements toward spatial targets; (d) frequent asynchrony between laryngeal and supralaryngeal mechanisms; (e) frequent initiation difficulties; and (f) overly complex sound sequences that were often produced as a result of the articulatory struggle itself. The authors also observed that these patients produced occasional errors of segmental selection, intrusion, metathesis, and elision. If the segments the authors were describing were phonemic in nature, than these errors would, of course, be indistinguishable from the phonemic paraphasias in Wernicke's aphasia or from the phonemic paraphasias of conduction aphasics (Buckingham, 1986a, 1992a, 1992b).

Anticipatory Coarticulation

There has been an increase in the examination of anticipatory coarticulation in apraxia of speech. Ziegler and von Cramon (1985), using a gated speech stimuli paradigm, assessed the temporal extent of anticipatory vocalic speech gestures within the spectral bursts of the prevocalic onset consonants. The language they studied was German, and the vowels examined made it possible to separate out the feature [+round] from the [front] versus [back] parameter, as German has front rounded and unrounded vowel phonemes. The normal speaker productions of the oral stop spectra showed correct upcoming vowel identification by hearers significantly ($p < .001$) above chance as soon as plosion was perceivable for the initial oral stops. For the apraxic spectral productions, hearers were not consistent until the actual vowel appeared. Thus, the rounding for the front

rounded vowel was not apparent with any regularity in the burst. Hearers revealed high rates of confusions between the high front-unrounded and the high back-rounded vowels. Lack of anticipatory lip rounding for the back vowel could have caused these confusions. Other apraxic productions showed some anticipatory coarticulation, but only later on in the spectral burst. These results, then, suggested to the authors that in apraxia of speech anticipatory coarticulation is not totally absent but DELAYED.

In a follow-up study, Ziegler and von Cramon (1986) more closely examined the acoustic waveform of -V1CV2- exemplars produced by the normals and by the apraxic subject in the 1985 study. They examined the formant frequencies and the linear prediction reflection coefficients in the burst spectra that preceded the vowels in question. For the normal productions, vocalic formant information of V2 appeared in outline form as early as V1, which preceded the intervocalic consonant. Anticipatory vocalic information for the apraxic exemplars, however, did not uniformally appear until much later in the waveform. Thus, the 1986 acoustic study provided strong support for the apraxic "delay" in anticipatory coarticulation found in the 1985 perceptual study.

Tuller and Story (1987) were in essential agreement with the delay account of anticipatory coarticulation of Ziegler and von Cramon (1985, 1986), but at the same time they also stressed that not all nonfluent patients show a delay and that delay in general is a relative phenomenon depending on factors such as unit length of items being uttered, the specific vowel in question, and the choice of measurement points. Also, it is certainly reasonable that listeners would use a complex combination of spectral cues for extracting vocalic anticipatory coarticulatory information in consonantal portions of the waveform, and that these cues would not necessarily be restricted to what was focused on in these studies: formant frequencies (for vowel quality) and characteristic spectral prominence (for rounding) (Katz, 1987). Furthermore, Tuller and Story (1987) pointed out that time-locked points established for the appearance of anticipatory coarticulation information in the waveform are based on collapsed figures over multiple speakers; there is also substantial temporal variation across speakers. Therefore, because "no current time-based theory of anticipatory coarticulation suggests that the absolute temporal extent of anticipation is the same across speakers" (Tuller & Story, 1987, p. 256), it is difficult to know precisely how much of the observed delay in the speech of apraxics is truly abnormal and how much is simply indicative of the variation found in normal speakers.

The interesting point in all of this is that Tuller and Story's (1987) results agree both with Ziegler and von Cramon AND with Katz (1987, 1988a; Katz, Machetanz, Orth, & Shonle, 1990), who essentially found no significant differences. Katz compared normal subjects with both anterior and posterior

aphasics and found that hearers could perceive vocalic information in the speech of all groups at roughly the same early points. As would be expected, however, Ziegler's and Katz's studies used different stimuli. Ziegler's German language work used trisyllabic forms that were embedded in carrier phrases, all of which were uttered by the apraxics. Katz, on the other hand, worked only with English syllables and, unlike Ziegler's study where subjects REPEATED, Katz's subjects READ ALOUD monosyllabic words and nonwords. These design differences alone would make direct comparisons between the two studies difficult.

Katz's acoustic analysis demonstrated that the apraxic subjects as well as the fluent and normal subjects had "robust" coarticulatory anticipation of the vowels in [ki] and [ku]. Coarticulatory vowel information was equally apparent in the [s] and [t] of [su] and [tu] across subjects for both [round] and [place]. With the CCV stimuli, all groups produced early acoustic correlates of anticipatory coarticulation of the vowel in the [s] spectrum, the information traveling across (through) the intervening stop. The 10 listeners in Katz's study performed above chance in recognizing the upcoming vowel from the spectral information in the early portions of the consonantal waveforms of all speakers. Although, as a group, the apraxic exemplars were identified above chance, these exemplars received lower identification scores than did those produced by the fluent and normal speakers. In addition, it is interesting to observe that three of the five apraxic speakers were able motorically to produce the CCV stimuli. One actually showed *greater* than normal degrees of spectral shift in the fricative portions of the [stV] and the [skV]. Another revealed normal shifts for [stV] but not for [skV]. The third showed normal anticipatory shifts for both CCV types.

Tuller and Story (1987, p. 257) examined the differences in Ziegler's and Katz's stimuli and suggested that the early appearance of vocalic anticipatory information in the consonant spectra in Katz's study is likely due to the use of monosyllabic STRESSED forms. These isolated, stressed monosyllabic forms would naturally allow for more extensive articulatory PREPOSTURING for both CV and CCV forms, with consonants that would likely produce spectra with greater anticipatory vocalic features. Although Ziegler did not use fricatives, his stimuli were embedded within larger ranges of phonological material, and therefore, as Tuller and Story suggested, the articulations in the Ziegler study may very well have been subject to greater temporal and spatial constraints, inhibiting exaggerated preposturing.

Carryover versus Anticipatory Coarticulation

In a later study, Tuller and Story (1988) looked at normal, fluent, and nonfluent (apraxic) aphasic subjects on "carryover" (left-to-right) coartic-

ulation. The fluent and nonfluent groups performed somewhat worse than normals for carryover phenomena, but were essentially similar among themselves. However, for anticipatory coarticulation, the two pathological groups were clearly different. Only the apraxics demonstrated a delay in coarticulatory shifts in prevocalic fricatives. Although this study did not use the lengthy stimuli of Ziegler, bisyllabic word pairs were used. Length of stimuli, therefore, seems to be a crucial factor determining whether an apraxic speaker will exhibit anticipatory coarticulation. It is also probably advisable to place the articulatory material to be analyzed within a larger span of material, rather than place it simply in an isolated stressed monosyllable or in initial positions of larger stretches. Exaggerated preposturing by patients with apraxia of speech seems to be more likely at the initiation of words or phrases.

Phonemic False Evaluation in Nonfluent Anterior Patients

The phenomenon of "phonemic false evaluation" (e.g., Buckingham & Yule, 1987) has obscured many earlier studies of apraxia of speech and in many cases has made them difficult to interpret. In those early studies of segmental production in apraxia of speech, many of the presumed phonemic substitutions were in fact only category substitutions in the minds of those who were listening to the apraxics. More refined technological instrumentation finally demonstrated that many of what were initially felt to be phonemic substitutions were in actuality phonetic asynchronies that shifted acoustic cues in one way or another such that hearers encoded phonemic category shifts in their own minds. The problem was that those hearers subsequently credited what they themselves coded as phonemic substitutions to the speakers. Indeed, much confusion naturally ensued (Miller, 1995). In the first edition of the present chapter, this point was discussed against the background of the early studies (e.g., Blumstein et al., 1977) that revealed the actual motoric underpinnings of certain of these falsely evaluated phonemic substitutions.

Vowel Lowering in Nonfluent Aphasia: Phonemic or Phonetic?

Since the first edition of *Acquired Aphasia,* another study of ostensive phonemic substitutions in the limb-kinetic, apraxia of speech component of Broca's aphasia (Keller, 1978) was scrutinized in Buckingham (1982) in an attempt to suggest that there was very likely a motoric underpinning for the phonemic substitutions, and that in reality the phonemic substitu-

tions were encoded by the hearer, whereas the motor asynchrony was produced by the speaker. Keller observed what he felt to be phonemic level vowel substitution in apraxia of speech, whereby patients would select mid vowels for high vowels and low vowels for mid vowels. The phonemic vowel substitutions were in the direction of lower vowels for higher vowels. He explored four hypotheses to account for these vowel lowerings: (a) loss of fine-grained control for the selective contraction of a few single muscles for higher vowels, resulting in a strong simultaneous contraction of the genioglossus and hyoglossus (the two principal intrinsic tongue muscle groups); (b) paradigmatic selection disorders that seem to favor lower vowel counterparts; (c) inaccurate proprioceptive feedback; and (d) incomplete auditory feedback. Each of these hypotheses is problematic. The first would account only for the increased productions of [a] and would not account for errors such as /I/ to /ɛ/. The second appears to be constructed in accordance with the data it seeks to explain. The third and fourth hypotheses do not explain the DIRECTIONALITY of the errors. Keller did not offer a much more likely scenario: auditory impression of vowel lowering caused by abnormal nasalization of the vowels in question resulting from faulty velar control.

Speech apraxic patients such as those studied by Keller (1978) often have "a marked variability in terms of the pattern of velar movements" (Itoh et al., 1979, p. 227). Katz et al. (1988, 1990), in kinematic analyses of anterior aphasia (especially patient EG) with electromagnetic articulography, noted that their patient's velum was relatively low throughout his speech production, although the patient "did not sound overly nasal." Nevertheless, my claim would be that it is quite possible that enough nasality was coming through to affect the formant structure of the vowels so produced. There is experimental evidence that "vowel nasalization is accompanied by an auditory lowering of the vowel" (Wright, 1975, p. 382). In this study, Wright demonstrated that subjects consistently perceived nasalized high and mid vowels as lower. Wright showed that nasalization of high and mid vowels will cause their first formants to rise, thereby imparting an auditory lowering.

Subsequently, Beddor, Krakow, and Goldstein (1986) examined listener misperceptions of the height of nasalized vowels, and their findings are extremely illuminating. They found that listeners misperceive vowel height of nasalized vowels only in those cases where nasalization is motorically inappropriate and where there is no conditioning nasal consonant in the phonological structure of the target word. Although Beddor et al. were seeking listener-oriented explanations for diachronic sound changes, their findings strengthen the likelihood of my contention that the apraxic vowel-lowering phonemic substitutions could very well have been in the

minds of the hearers, that is, phonemic false evaluation. The crucial factor is that the nasalization would be abnormal and inappropriate in the case of the brain-damaged apraxic with disrupted velopharyngeal control. The majority of the "lowerings" would be expected to be in words where there was no nasal consonant in the underlying form, which is precisely the case with apraxic disruption—no underlying nasal phoneme. In addition, Krakow, Beddor, and Goldstein (1988) observed that hearers' perception of height of nasal vowels is influenced as well by the LENGTH of the vowel. More perceptions of lowering occurred when the vowels were longer in duration. Consequently, for the perception of motor aphasic speech, one would expect an even greater tendency to perceive vowel lowering, as these same patients very often produce abnormally lengthened vowels (e.g., Kent & Rosenbek, 1983; Seddoh et al., 1996).

Beddor et al. (1986) disagreed with Wright (1975) that the important acoustic phenomenon is a raising of the first formant. They pointed out that what is happening with nasalized vowels occurs in the region or vicinity of the first formant, rather than that something is affecting the first formant exclusively. For P. S. Beddor (personal communication, 1988), the perceptual lowering of high and mid nasalized vowels is due to the additional nasal formant, which raises the "center of gravity" of high and mid vowels relative to their oral counterparts. As opposed to Wright's first-formant raising theory, Beddor's account also explains the observation that low nasalized vowels will perceptually rise, because the center of gravity of low vowels is lowered by the added nasal formant. Low nasalized vowels should actually lower further, if Wright is correct. Nevertheless, the perceptual consequences of both studies lend credence to my suggestion that phonemic false evaluation is a likely candidate for explaining why certain patients with apraxia of speech from anterior lesions seem to be substituting lower vowels for their higher counterparts. Again, the phonemic selection characterization may only be in the minds of the hearers, whereas the apraxic's problem would be more properly defined as faulty velopharyngeal synchrony with upper articulatory gesturing (Krakow et al., 1988).

Phonemic False Evaluation in Fluent Aphasias

Up to this point, I have focused on phonemic false evaluation as it pertains to the speech substitution errors of patients with anterior lobe, limb-kinetic apraxia of speech. The evidence for this phonetic–phonemic mismatch is rather noncontroversial for the nonfluent speakers. Could there be, as well, a SUBTLE APRAXIC COMPONENT in the fluent left posterior lesion aphasias that might also lead to phonemic false evaluation of segmental substitutive errors? That is, could any of the often observed phonemic sub-

stitutions in Wernicke's aphasia, or in conduction aphasia, be in the minds of the listeners as well?

In several publications, beginning with Blumstein et al. (1980), motor–phonetic abnormalities have, in fact, been charted in the speech of otherwise fluent, posterior aphasics. Subsequent to this publication, there have been several others: MacNeilage, Hutchinson, and Lasater (1981), MacNeilage (1982), Shinn and Blumstein (1983), Tuller (1984), Duffy and Gawle (1984), Ryalls (1986), Kent and McNeil (1987), McNeil et al. (1990), Baum, Blumstein, Naeser, and Palumbo (1990), Code (1998), and other studies listed in Vijayan and Gandour's (1995) critical assessment of subtle phonetic deficits in fluent, posterior aphasias. Different types of phonetic asynchronies (e.g., fricative elongation, abnormal vowel lengthening, range alterations for VOT, preburst stopgap duration differences, initial CV elongations) were found in these studies, some of which have only phonetic consequences, whereas others could very likely have phonological consequences—for hearers, that is. It goes without saying that an unambiguous phonemic error, where the uttered exemplar falls within the allophonic range of that phoneme when it NORMALLY occurs in the same phonetic environment, adumbrates a phonemic level misselection for the aphasic. Although, as Baum and Slatkovsky (1993) point out, "we are still simply inferring the intentions of the speaker" (p. 215). True, indeed. The fact is, however, that not all so-called phonemic selection errors are encoded at the level of phonemic selection by the aphasic. These subtle phonetic disruptions in fluent aphasia are phonetic nonetheless, and, again, some may have phonological consequences and some may not. Ziegler (1987) opts for a very strong version. He claims that "aberrations in the sound pattern of Wernicke's aphasics reflect a dysfunction affecting the discrete structure in the inventory of phonetic plans, whereas patients with apraxia of speech exhibit problems of realizing properly selected phonological units" (p. 177). Moreover, Dogil (1989) demonstrated a startling amount of motor disruption in the early phase of jargon aphasia in a Wernicke's aphasic. Vijayan and Gandour (1995), in contradistinction, claim that, although a subgroup of conduction aphasics may exhibit subtle phonetic deficits,[6] there is little if any evidence that Wernicke's aphasics do.

6. Heterogeneity is fast becoming a signpost of conduction aphasia (e.g., Simmons-Mackie, 1997). Buckingham (1992b) wrote that

> The cross-cutting functional complexities and extreme heterogeneity seen in conduction aphasia present some of the most intriguing and challenging puzzles for language and speech production modelling in aphasiology today, and it would not be surprising if that syndrome ultimately goes the way of agrammatism, holding together as it does at the level of a theoretical category and resisting much patient-to-patient homogeneity. (pp. 112–113)

It is important to note that what we want to chart for phonemic false evaluation are the phonetic alterations that specifically compromise the regions in the acoustic structure that carry crucial cues for phonemic perception. For example, the abnormal prevoicing of Wernicke's aphasics reported in Blumstein et al. (1980) gives us cause to suspect that all is not so fluent with that population, but VOT cues do not rest there for English, and therefore they would not engender any phonemic misperception in hearers. Slight abnormalities in a production of the spectral portion of a stop may be due to a phonetic control problem involving laryngeal manipulation, but it may give rise to phonemic category shifts in the feature [place] for perceivers. Abnormally lengthened voiceless fricatives may induce a hearer to perceive the voiced counterpart, and one would certainly expect abnormal vowel length control before final obstruents to at least occasionally lead to misperceptions of the [+/±voice] phonemic category of those postvocalic coda obstruents. We observed how abnormal nasalization of vowels can affect their perception. Consequently, aphasiologists can no longer simply assume that the only production errors of the posterior aphasic are phonemically based paraphasias. They must instrumentally scrutinize the actual exemplars, use proper control groups, utilize all modes of elicitation (making sure that cross-comparisons use similar confrontational tests), compare LIKE subject populations (which will be difficult, especially with the heterogeneity of apraxia of speech and conduction aphasia), and choose proper sample sizes (Vijayan & Gandour, 1995). And finally, why should the expectancy of some subtle phonetic deficit in the fluent aphasic population in general be so startling? The pars opercularis in the frontal lobe and the planum temporale of the posterior third of the superior temporal gyrus are achitectonically and evolutionarily so similar that researchers such as Galaburda (1982, p. 443) have found it "somewhat surprising . . . that lesions in either region produce such different aphasic syndromes."

Conclusions

In conclusion, it is safe to say that ample historical precedent exists for TWO different neuroanatomical explanations for apraxia: center lesion and disconnection. Liepmann introduced both explanations. Geschwind discarded all but the disconnection syndromes as confusing, ill defined, and counterproductive, leaving no place for a limb-kinetic apraxia of speech. Darley, the progenitor of the Mayo School position, used Liepmann's suggestion that motor aphasia was more likely a limb-kinetic apraxia of the

glossolabiopharyngeal musculature. The neo-Geschwinians have extended Geschwind's work on apraxias to further refinements within the limb apraxias and have suggested more appropriate categories of conduction apraxia and conceptual apraxia (Rothi & Heilman, 1997). Heilman et al. (1997) have crafted a representational system within an information-processing model that extends the picture of apraxia beyond mere connections, but they focus exclusively on nonspeech limb movement processing. Researchers in the tradition of the Mayo School are actively engaged in increasingly sophisticated experimental–instrumental studies of the phonetic, acoustic, physiological, and perceptual parameters in apraxia of speech, in which the speech output that is measured is not necessarily elicited by linguistic command (e.g., Duffy, 1995). Speech samples are garnered by having the subjects repeat words, phrases, or certain carrier phrases within which the target words are embedded, by having the subjects read aloud, name objects, and describe pictured scenes, or by engaging them in spontaneous speech. These studies predictably claim that the cortical regions damaged are those that (somehow) contain the information for proper articulatory timing, whether single or multiple gestures are involved. Although for Liepmann, the limb-kinetic apraxics were essentially caused by damage to the frontal motor zones, other locations for center lesion apraxias have been uncovered, especially the insula, the parietal lobe, and certain structures of the basal ganglia (Dronkers, 1996; Square et al., 1997). The center lesion studies do not focus as much on the stimulus-setting specificity of the connectionists. Rarely do they ask whether a patient with apraxia of speech would demonstrate delays in anticipatory coarticulation when repeating carrier phrases with the target words embedded, but not when reading short sentences, for instance. The focus is rather on the nature of the phonetic disruption and whether or not that disruption is part of a larger motor disorganization (e.g., Hageman, Robin, Moon, & Folkins, 1994). The distinctions between dysarthric, apraxic, and phonemic errors are being brought into sharper focus, although heterogeneity continues to ensure that patients will often mix and match. Both groups of researchers will no doubt continue to use the descriptor "apraxia" to label the maladies they investigate, and why not? Both camps buy into the mechanisms suggested by Liepmann to account for abnormal praxis.

Acknowledgments

In the first edition (1981) of this essay, I expressed my appreciation to Norman Geschwind for having read and commented on the earlier drafts. He sadly died on 4 November 1984. The

revised version (1991) of this essay was dedicated to him. In the spirit of Schachter and Devinsky (1997), this third edition is dedicated as well to the memory of Norman Geschwind. I also express my appreciation to Jason W. Brown and Leonard LaPointe for their helpful remarks on earlier drafts of the essay. Naturally, they are not to be held responsible for any of my interpretations or misinterpretations. This essay is a third updating of the author's paper, which originally appeared in *Brain and Language* (1979), *8*, 202–226.

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9

Aphasia-Related Disorders

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Aphasic patients are heterogeneous with respect to the presence and severity of concommitant cognitive neuropsychological impairment. Although the language deficit may be the most obvious and immediately compelling, additional cognitive problems are likely to coexist. Careful delineation of the nature and extent of associated deficits may therefore clarify issues of diagnosis, inferences concerning localization, and choice of appropriate therapeutic and rehabilitative interventions. The pattern of spared and compromised skills has important implications for a patient's adaptation and recovery. Although nonlinguistic cognitive data have not been used to the same extent in outcome prediction as have similar data in other populations, such as head injury, several studies have been undertaken in this regard. This chapter reviews some of the major associated disturbances that are of historical, theoretical, and clinical importance. These include acquired disturbances of written language (agraphia), calculation (acalculia), right–left orientation, finger localization, visuospatial ability, attention, memory, and executive functions.

These impairments may occur in isolation or in selective clusters as a function of the locus, extent, and etiology of the lesion. Variables including the patient's handedness, history of familial sinistrality, and premorbid abilities may influence the nature and severity of dysfunction. Moreover, to the extent to which verbal coding and semantic representation may play a role in performing certain tasks, the aphasia itself may be a mediating variable. This chapter reviews the disorders listed in the opening paragraph and discusses their presentation in the aphasic individual.

Alexia and Agraphia

Reading, and especially writing, seldom escape unscathed in aphasia. Written language problems can persist chronically even after the disturbance in oral language resolves. The character of the reading and writing disorder tends to show some systematic relationship to the form of the oral language disorder. For example, patients with Broca's aphasia who have agrammatic speech also tend to have difficulties reading and writing grammatical words; patients with conduction aphasia who typically produce literal (phonemic) paraphasias in speech, also often make phonological errors when reading aloud; and patients with Wernicke's aphasia who demonstrate impaired auditory comprehension of word meanings and who produce verbal (semantic) paraphasias in speech, also make semantic errors when reading and writing. The parallels between the oral and written language disorders derive from the fact that these abilities rely, at least in part, on the same elementary language processing components. In addition to engaging central linguistic processing components common to all language tasks, reading and writing also require specialized visual, orthographic, and graphomotor procedures that are separate from the central language system. Thus despite some broad parallels between the patterns of acquired oral and written language disorders, the relationship between alexic, agraphic, and aphasic disorders is not constant. There is significant variability in the degree and type of reading and writing impairment exhibited by patients with the same aphasic syndrome. Also, the lesion site tends to vary considerably within groups of patients with similar types of alexic or agraphic disorders. For these reasons, older classification schemes linking alexic and agraphic disorders to particular aphasic syndromes have been abandoned. Rather, it seems more profitable to describe acquired disorders of reading and writing in relation to the functioning of various cognitive components underlying written language processing.

Cognitive Components of Reading

The initial stage of reading involves PERCEPTUAL ANALYSIS of the visual stimulus. Visual perceptual analysis entails isolating individual letters and encoding their relative ordering in the written stimulus and abstracting the location of the written word in space. The next processing stage involves LETTER RECOGNITION in which the physical shapes of letters are coded into abstract letter identities. Different versions of a given letter (uppercase, lowercase, printed, and cursive) are all coded identically. In competent readers, all of the letters in a presented word, regardless of its length, are

assumed to be processed in a rapid, automatic, and parallel manner (LaBerge & Samuels, 1974).

After these stages of "peripheral" visual processing, central language-processing components are engaged. CENTRAL PROCESSING involves matching the visual stimulus to stored orthographic, phonological, and semantic representations of previously encountered words to derive its pronunciation and meaning. The ORTHOGRAPHIC LEXICON consists of representations of previously encountered strings of letters. Some reading models have assumed that these representations consist of whole word patterns or "visual word forms" (Morton & Patterson, 1980). More recent models conceptualize these representations in terms of activation patterns across a distributed network of orthographic units of varying sizes (Seidenberg & McClelland, 1989; Shallice, Warrington, & McCarthy, 1983).

Subsequent to orthographic analysis, two routes are available for processing the written word. One route involves activation of codes in the PHONOLOGICAL LEXICON that specify pronunciations of letter strings. A second route involves activation of word meanings that are represented in the SEMANTIC LEXICON. The independence of the phonological and semantic lexical processing components is evidenced by the fact that orthographic information can be used to access pronunciations for letter strings whose meanings are not necessarily known (pronounceable nonwords or pseudowords such as *rilt*), and also to access discrete meanings for homophonic letter strings having the same pronunciation (e.g., *made* and *maid*). It is assumed that both semantic and phonological processing systems are engaged in normal reading and that normally there is a bidirectional flow of information between these two processing components, although in brain-damaged patients relationships between the various processing components can become uncoupled.

Varieties of Single-Word Reading Disorders

NEGLECT ALEXIA is one of the most common types of reading disorders. It is thought to be an attentional disorder that results from a disturbance in visual perceptual processing and is manifested in failure to identify letters at one end of the word (Kinsbourne & Warrington, 1962). The neglect is usually more severe when reading pseudowords than when reading familiar real words (Sieroff, Pollatsek, & Posner, 1988). Errors in neglect alexia usually take the form of either deletions (e.g., *can* is read as "an") or letter substitutions (*wine* is read as "mine"). Although cases of both right and left neglect alexia have been reported, inattention in reading most often occurs for letters at the beginning or left side of words. This is similar to findings that elements in the left visual space are also disproportionately in-

volved in visual neglect for other types of material (Heilman, Watson, & Valenstein, 1993).

ALEXIA WITHOUT AGRAPHIA, or PURE ALEXIA, involves disturbed recognition of written words, and often also impaired letter recognition, in the absence of a writing disorder (Dejerine, 1892). Reading is very labored for patients with this disorder, and it tends to be slower and less efficient for longer letter strings compared with short words. Sometimes this leads to a compensatory strategy of reading by naming individual letters serially and then identifying the word on the basis of the named letters, hence the designation "letter-by-letter" reading (Patterson & Kay, 1982). Although impaired in reading visually presented words, patients with this disorder show preserved recognition of orally spelled words (providing auditory verbal span and working memory capacities are not exceeded) and of letters and words traced on the palm, as well as preserved written spelling. These observations suggest that stored orthographic information is preserved in these patients, but it may not be accessible through the visual modality. According to one account, alexia without agraphia reflects a disconnection between the mechanism for visual letter recognition and the orthographic lexicon (Patterson & Kay, 1982). An alternative account suggests that this reading disorder results from a general disturbance in the capacity to process and identify in parallel arrays of simultaneously presented letters and other visual forms (Farah & Wallace, 1991; Friedman & Alexander, 1984).

SURFACE ALEXIA is characterized by specific difficulties reading words whose pronunciations do not conform to typical spelling-to-sound correspondence patterns (Patterson, Marshall, & Coltheart, 1985). Patients with this type of disorder demonstrate impaired reading of words with exceptional spellings, especially those of low frequency (e.g., *cello*), but usually reading of pseudowords (e.g., *chaid*) and words with regular correspondence between orthography and phonology (e.g., *lobster*) is better preserved. "Regularization" errors are common. These consist of incorrect pronunciations that are nonetheless plausible because they are derived from the typical pronunciations of other words with similar spellings (e.g., pronouncing *have* to rhyme with "cave"). Sometimes errors may involve misapplication of familiar spelling-to-sound correspondences (e.g., *lace* pronounced as "lake"; Marshall & Newcombe, 1973). Surface alexia is thought to be caused by a failure to activate established orthographic patterns in memory (Friedman, Ween, & Albert, 1993). In the absence of access to stored orthographic representations, all written stimuli are read as if they were novel pseudowords. Pronunciation is most successful for letter patterns that occur frequently and that are associated with a single invariant pronunciation in many different words (regular spellings), com-

pared with ambiguous orthographic patterns that can yield several different and sometimes conflicting pronunciations (e.g., *pear* could be pronounced to rhyme with "bear" or "hear") and patterns with unique pronunciations (exceptional spellings). Impaired access to orthographic representations also results in a failure of the orthographic information to activate corresponding meanings in the semantic lexicon. Consequently, patients often rely on their pronunciation of the written stimulus to ascertain the word's meaning. This can result in mistaking a written word such as *bear* for an alcoholic beverage, and interpreting the written word *draught* to mean an absence of rain.

PHONOLOGICAL ALEXIA is manifested by impaired oral reading of pseudowords in the face of much better preserved real-word reading (Beauvois & Derouesné, 1979). This disorder is thought to derive from impaired access to or disruption of representations within the phonological lexicon (Friedman, 1995). Unlike real words, which can be identified and pronounced by first accessing their meanings, pronunciations of pseudowords require access to phonological lexical knowledge. In the past it had been claimed that separate routes exist for deriving pronunciations of real words and pseudowords (Morton & Patterson, 1980), but now there is more agreement that pronunciations of real words and pseudowords derive from a single common phonological processing system (Seidenberg, Plaut, Peterson, McClelland, & McRae, 1994). Thus, disturbed functioning of the phonological processing system impairs pseudoword reading more than real-word reading. In phonological alexia, oral reading of words with less stable semantic representation (functors, such as *neither,* serving grammatical functions and words referring to low imagery or abstract concepts) can also become impaired. This results in an effect of "part of speech," in which nouns are read better than verbs and verbs are read better than functors, and/or an effect of "imageability," in which concrete nouns referring to imageable concepts (e.g., *camel*) are read better than nouns referring to abstract concepts (e.g., *moment*).

Impaired processing of information in the semantic system results in a disorder of reading known as SEMANTIC ALEXIA or READING WITHOUT SEMANTICS (Shalice et al., 1983). Despite intact oral reading of all types of real words (and also pseudowords), comprehension of the meanings of written and spoken words is profoundly impaired. Most often this disorder is seen in patients with progressive dementias such as Alzheimer's disease (Friedman, Ferguson, Robinson, & Sunderland, 1992; M. F. Schwartz, Saffran, & Marin, 1980), although it has also been reported in other selected patients (Bub, Cancelliere, & Kertesz, 1985; Glosser, Friedman, & Roeltgen, 1996).

The reading disorders discussed thus far exemplify results of disruption

in relatively circumscribed cognitive components of the written language-processing system. In reality, however, many reading disorders are the consequence of disturbance in more than a single cognitive component. A full discussion of the so-called "multicomponent" reading disorders is beyond the scope of this chapter, and the reader is referred to Shallice and Warrington (1980) and Friedman et al. (1993) for information about different types of multicomponent reading disorders. Here, we review only the most well-recognized multicomponent reading disorder, DEEP ALEXIA (Coltheart, Patterson, & Marshall, 1987; Marshall & Newcombe, 1973). The defining feature of deep alexia is the production of semantic paralexias. These are erroneous responses that are related to the target word in meaning but share no visual, orthographic, or phonological features with the target (e.g., reading *uncle* as *cousin*). Along with the critical symptom of semantic paralexias, patients with the disorder of deep alexia also show a profound disturbance of pseudoword reading, as well as significant effects of part of speech and imageability in real-word reading. It has been suggested that deep alexia results from a combination of a deficit in phonological processing, such as that seen in patients with phonological alexia, and a deficit in semantic processing. Furthermore, the apparent similarities between symptoms in deep alexia and phonological alexia, and the observation that some patients who display a pattern of deep alexia initially after brain injury subsequently recover to show a deficit pattern of phonological alexia, have led to a suggested continuum between these two disorders (Glosser & Friedman, 1990).

Analysis of reading at the single-word level has proven most productive for classifying and understanding the cognitive dysfunction underlying different acquired disorders of reading. Many of the same cognitive symptoms impairing single-word reading also impair reading of sentences and paragraphs, but additional cognitive disturbances must also be considered in understanding reading disorders at the discourse level. For example, sentence and paragraph reading can become disrupted as a result of impairments in the ability to process syntactic relationships between words. Reading beyond the single-word level may also become disrupted as a result of problems with working memory, organizing information thematically, and reasoning inferentially. Although assessment of sentence and paragraph reading is not especially informative about the underlying neurological or cognitive dysfunction, analyses of these performances may be quite important for defining functional and adaptive reading abilities.

Cognitive Components of Writing

Writing depends on the same central linguistic cognitive components as those discussed for reading, as well as other components specific to the

production of letters. In writing to dictation, auditory analysis first decomposes inputs into phonological units that can be matched to representations of familiar auditory word patterns in the phonological lexicon. Processing then proceeds either to activate corresponding letter patterns in the orthographic lexicon or to activate associated meanings in the semantic lexicon. Thus when writing to dictation, the orthographic representations may be accessed either directly from the phonological lexicon or after the word's meaning is accessed in the semantic lexicon. In spontaneous writing, ideational concepts can activate lexical semantic information. This information can also be accessed through sensory systems, as occurs during visual or tactile written naming. Once accessed, semantic information activates the associated orthography.

Written output of the orthographic patterns requires further processing through several "peripheral" transformations that are specific to writing. The GRAPHEMIC or ORTHOGRAPHIC BUFFER refers to a working memory process that maintains the spelling code in a temporary buffer while the physical or ALLOGRAPHIC FORMS of the letters are selected for written output or the letter names are accessed for oral spelling. GRAPHIC MOTOR PATTERNS specify the production of letter shapes corresponding to the orthographic code.

Varieties of Single-Word Writing Disorders

Several writing disorders are assumed to stem from disruption of central linguistic processing components, and these parallel the reading disorders described earlier in terms of their defining symptoms. The hallmark symptom of PHONOLOGICAL AGRAPHIA is an impairment in writing pseudowords to dictation, with better preserved ability to spell real words (Shallice, 1981). In some cases the problem in pseudoword writing is accompanied by difficulties with functors and abstract words, as is seen in phonological alexia (Baxter & Warrington, 1985; Roeltgen, Sevush, & Heilman, 1983). Some patients with impaired pseudoword writing also make semantic paragraphias (e.g., writing *desk* for the dictated word "chair") and demonstrate a constellation of symptoms of DEEP AGRAPHIA (Bub & Kertesz, 1982) analogous to those seen in deep alexia.

LEXICAL AGRAPHIA (Beauvois & Derouesné, 1981), like surface alexia, is characterized by the relatively preserved writing of words with regular spellings in the face of impaired writing of lower frequency words with ambiguous or exceptional sound-to-spelling correspondence. These patients produce regularization spelling errors that tend to preserve the phonological form of the target word (e.g., *chef* is written as *shef*).

Finally, patients with SEMANTIC AGRAPHIA may write real words and pseudowords normally to dictation, but they have difficulties in written

confrontation naming or description (Roeltgen, Rothi, & Heilman, 1986). These patients' difficulties are quite striking when required to write dictated homophones that require the integration of a word's meaning with its spelling. Errors reflect the relative absence of a semantic influence on spelling, as for example, when the word *pair* is written in response to the dictated sentence "She ate an apple and a *pear*." Semantic agraphia can occur with focal lesions (e.g., Rapcsak & Rubens, 1990), but it is most often seen as one of the early linguistic symptoms in Alzheimer's disease (Glosser & Kaplan, 1989).

Reading and other aspects of language functioning may be spared in peripheral agraphias that are assumed to stem from disturbances in procedures for visual and motor output of spelling. In agraphia caused by a GRAPHEMIC BUFFER DEFICIT (Hillis & Caramazza, 1989), spelling performance is similar in oral and written modalities and tends to be independent of lexical and orthographic characteristics of the target stimulus. Spelling difficulties are related to word length, with more errors for longer words than short words. Errors may consist of letter deletions, additions and substitutions, and letter order transpositions that result in phonemically implausible nonwords (e.g., *skip* is spelled as *ksip*). These errors derive from a disturbance in the specialized working memory process that maintains spelling representations in a temporary store prior to written or oral output.

In the agraphic disorders described thus far, accuracy does not differ noticeably between written and oral spelling, or for that matter in typing or spelling with block letters. Impairments in the peripheral mechanisms for written output, however, can result in a dissociation between preserved oral spelling and disturbed written spelling. Patients with APRAXIC AGRAPHIA, previously termed PURE AGRAPHIA (Dubois, Hécaen, & Marcie, 1969), demonstrate disturbed writing in the presence of intact speech, reading, oral spelling, and visuoconstructional abilities. This disorder is believed to involve a problem in accessing the allographic codes and/or selecting the appropriate program specifying the movements needed to form letters (Friedman & Alexander, 1989). This can result in incorrect letter selection, production of poorly formed letters (sometimes to the point of illegibility), inability to maintain a particular case or script consistently, and a variable range of difficulty copying letters and words, as is seen in other forms of apraxia (Baxter & Warrington, 1986; Ellis, 1982; Patterson & Wing, 1989). The difficulty accessing familiar motor programs for writing, however, often dissociates from other types of apraxia (Coslett, Rothi, Valenstein, & Heilman, 1986). SPATIAL AGRAPHIA can be manifested in various types of problems in attention to the spatial organization of the written output. There may be errors in the orientation of words on the page, hemi-

spatial neglect, spacing errors, or repetitions of strokes within letters (Hécaen & Marcie, 1974). This disorder, like neglect alexia, is most often associated with pathology in the nondominant hemisphere (Hécaen, Penfield, Bertrand, & Malmo, 1956).

As with reading, writing beyond the single-word level can become affected by disruptions in a broad range of cognitive systems. Chedru and Geschwind (1972), for example, were the first to note the exceptional vulnerability of writing in acute confusional states. They described patients with confusion due to a variety of pathophysiological processes whose writing deteriorated to senseless scrawls. As another example, Goodglass and Hunter (1970) demonstrated parallels between utterance length, fluency, and features of agrammatism in the speech and writing of patients with Broca's aphasia.

The relationships between cognitive components of reading and writing are still being debated (Bub & Chertkow, 1988; Shallice, 1988). According to one view these systems are totally independent (Beauvois & Derouesné, 1981). An alternative view posits that lexical orthographic and/or phonological representations are shared by the reading and spelling systems (Coltheart & Funnell, 1987). Although this debate is not yet resolved, it is generally agreed that the character of reading and writing disorders seen within a single patient may differ substantially. The most famous example of this type of dissociation was reported in the case of a patient who had a selective difficulty in reading pseudowords (phonological alexia), but whose writing errors occurred mostly on words with irregular spelling, indicative of a lexical agraphia (Beauvois & Derouesné, 1981). Such clinical observations can be accommodated by several different theoretical accounts (Bub & Chertkow, 1988), although the explanations of these phenomena differ depending on assumptions about the relationship between the cognitive mechanisms for reading and spelling.

Acalculia

Loss of mathematical abilities can have a profound effect on a patient's ability to manage such basic activities as making change, balancing a checkbook, cooking from a recipe, or planning activities that involve the quantification of time. The concept of calculations actually encompasses a broad range of cognitive tasks; some involve rote retrieval of number facts, others are procedural (e.g., specifying and applying the steps in correct sequence), and others are strategic (e.g., using magnitude estimation to double-check roughly the results obtained by a calculation procedure) (Clark & Campbell, 1991; McCloskey, 1992). Deficits may occur in specific do-

mains of calculation or may be more global. The potential dissociation of these skills is discussed later in this section.

The term ACALCULIA was introduced by Henschen (1919) to designate an acquired calculation disorder distinct from an aphasic inability to read and write numbers. Before Henschen, there had been a number of case reports of acquired impairment of calculation overshadowing other co-occurring impairments. Peritz (1918) proposed the left angular gyrus as the center for calculation, a localization endorsed by Henschen (1919).

Berger (1926) described three cases of acalculia with dominant hemisphere lesions that did *not* involve the angular gyrus (two occipital and one temporal). These "pure" cases (PRIMARY ACALCULIA) were distinct from SECONDARY ACALCULIA, that is, calculation disturbances resulting from aphasia, alexia, agraphia, or problems in cognitive functioning, such as attention, memory, or visuospatial information processing. Secondary acalculia therefore occurs more frequently than pure or primary acalculia.

Appreciation of magnitude may exist at a preverbal level, as is evident from studies of magnitude awareness in animals and preverbal children. Gallistel and Gelman (1992) suggested that this system coexists actively with subsequently acquired numeracy skills, permitting quick approximations as well as rough verification of the accuracy of a computation. The co-occurrence of severe constructional difficulties noted in some cases (e.g., Singer & Low, 1933) led some investigators to consider the role of a spatial component (Critchley, 1953).

In 1961, Hécaen, Angelergues, and Houillier proposed the following classifications based on a study of 183 acalculic patients with retrorolandic lesions:

1. Digit alexia and agraphia may or may not be accompanied by an aphasia, alexia, and/or agraphia. In this type of acalculia, the PARALEXIC substitutions for presented numbers (stimuli) and the PARAGRAPHIC errors during computation (response) preclude a correct answer. Benson and Denckla (1969) demonstrated intact computational ability in two patients with digit alexia and agraphia by presenting multiple-choice solutions for arithmetic problems that had been originally "solved" incorrectly.
2. Spatial acalculia leads to incorrect solutions due to misalignment of numbers, misordered numbers, number reversals, directional confusion, sign confusion (e.g., + for x), visual neglect, or oculomotor disturbances. Here, too, basic computational skills may or may not be demonstrated to be intact.
3. Anarithmetria is a primary impairment of calculation. This does not imply an isolated impairment, but rather one that does not have as its source an alexia or agraphia for numbers or a spatial organizational problem.

Of the three types of acalculia, digit alexia, agraphia, and anarithmetria occurred predominantly with left-hemisphere lesions, whereas spatial acalculia rarely occurred with left-hemisphere lesions (10%), but occurred in 73.3% of patients with right-hemisphere lesions (Hécaen & Angelergues, 1961).

Within each hemisphere, varieties of acalculic disorders have been identified in virtually all regions of the brain (Grewel, 1969). Moreover, acalculia is a frequent early symptom in the progressive dementias, and may present developmentally as a specific learning disability and thus occur in the absence of a focal lesion. Variability in lesion loci involving different neural substrates has been attributed to premorbid individual differences in processing mathematical information (Leonhard, 1979). Conversely, the nature and severity of the calculation disorder are largely determined by the specific dysfunctions referable to different lesion loci. These in turn have differential impact on various aspects of the complex function of calculation (Benson & Weir, 1972; Cohn, 1961; Gerstmann, 1940; Hécaen et al., 1961). Whalen, McCloskey, Lesser, and Gordon (1997) described a case in which simple multiplication was selectively disrupted by cortical stimulation of a single site in the left anterior parietal area. They interpreted their findings as providing evidence for a dissociable role of this region in retrieval of arithmetic number facts.

Grafman, Passafiume, Faglioni, and Boller (1982) noted that patients with posterior left hemisphere lesions were particularly likely to show severely impaired calculation even in the presence of intact reading and writing, although patients with both left and right hemisphere lesions did manifest calculation deficits. Several studies have demonstrated dissociation of number fact retrieval from application of number procedures (Lampl, Eschel, Gilad, & Sarova-Pinhas, 1994). Warrington (1982) reported a case of a patient with a posterior left hemisphere lesion whose acalculia was specific to fact retrieval for all four basic arithmetic operations, despite satisfactory auditory comprehension and spared skills in other arithmetic domains. Delazer, Ewen, and Benke (1997) argue that arithmetic facts are a distinct, domain-specific component of the cognitive system, stored independently both of other math skills (e.g., calculation procedures) and of other semantic knowledge. Their study supports their theory that cognitive implementation of arithmetic facts involves "an interrelated network of associations in long term memory." Calculation may be impaired differentially as a function of the modality of presentation, the modality of response (Benton, 1963), and the nature and number of operations involved. Because acalculia encompasses many separate tasks, it is important to delineate the precise nature and extent of the impairment if anatomical–clinical correlations are to be meaningful.

Diagnostic tests of calculation skills readily available from commercial

sources (e.g., Key Math Diagnostic Arithmetic Tests, Connolly, 1988; or Arithmetic subtests of achievement tests such as the Wide Range Achievement Tests-Third Edition, Wilkinson, 1993; or *Wechsler Individual Achievement Test*, 1992) are generally designed and referenced for assessing grade-appropriate skills mastery. These tests do include basic numerical operations as well as problems involving fractions, decimals, percentages, simple linear equations, and geometry. Qualitative examination of items passed and failed on such tests can yield useful information. Langdon and Warrington (1995) introduced the VESPAR, a test of arithmetical reasoning in which attentional and language factors have been minimized. Investigators, including Deloche et al. (1994), have designed experimental batteries for assessing calculation and number processing in adults based on cognitive theories of calculation. The Deloche et al. battery includes, in addition to standard items, tasks such as number transcoding (from Arabic numerals to number words), timed estimation of magnitude (as might frequently be required in real-world situations), and fact and operation verification. Subsequent research (Deloche, Dellatolas, Vendrell, & Bergego, 1996) has established credible ecological validity for this battery in comparison with a questionnaire of numerical activities of daily living.

Although written calculations are certainly important in the assessment of acalculia, they are not sufficient to capture the entire domain of possible impairment. Conversely, some individuals may fail written calculations because of format, when the underlying arithmetic skills are intact, as may be the case with spatial acalculia.

Finger Agnosia

The inability to recognize or otherwise identify fingers on one's own hands, on those of the examiner, or on model hands was believed by Gerstmann (1924) to be a consequence of a more general disturbance of the body schema and attributable to a lesion in the parieto–occipital junction around the angular gyrus of the dominant hemisphere. It should be noted that when finger agnosia is diagnosed, the severity of the impairment in finger naming or comprehension must exist above and beyond any impairment that may coexist in body-part identification in general.

As is the case in each of the specific disorders (e.g., alexia, agraphia, and acalculia) described earlier, finger agnosia is manifested in a variety of ways. Performance may be impaired as a function of modality of presentation or response, that is, whether the modality of stimulation is auditory, visual, or tactile, and the response verbal or nonverbal (Benton, 1959; Critchley, 1966; Ettlinger, 1963). Although nonverbal finger recognition

was found to be impaired in left (18%) and right (16%) hemisphere lesions, most of the left hemisphere damaged patients showed evidence of either an aphasia or general mental impairment (Gainotti, Cianchetti, & Tiacci, 1972).

In a study of nine patients with nonverbal impairment of finger identification, Kinsbourne and Warrington (1962) found a high degree of association with visuoconstructive disorders. Schilder (1935) similarly reported the frequency of co-occurrence of such constructional defects as drawings (especially of face and hands in the human drawings). Gerstmann (1940) also recognized the frequency of co-occurrence of a constructional apraxia. Goodglass and Kaplan (1972) found finger agnosia to correlate best with arithmetic (.58), three-dimensional block constructions (.52), and stick construction (.52), and a general loading of .74 with a parietal lobe factor.

Finger agnosia rarely occurs as an isolated finding. Gerstmann (1924) suggested that finger agnosia is the primary deficit in a cluster of symptoms (agraphia, acalculia, and right–left disorientation). This tetrad of symptoms, known as the Gerstmann syndrome, is discussed later.

Right–Left Disorientation

The inability to identify the right and left sides of one's own body, as well as those of another person seated opposite, or on a schematic figure, has been long recognized to occur with lesions lateralized to the left hemisphere and in the presence of an aphasia (Bonhoeffer, 1923; Head, 1926). Like finger agnosia, right–left disorientation may be selectively impaired as a function of the modality of stimulus and the required response.

Verbal tasks (e.g., naming, responding to verbal command) and nonverbal tasks (e.g., imitation, indicating right vs. left on pictorial stimuli) should be studied separately, as should tasks of varying levels of complexity, such as identifying single lateralized parts on one's own body, executing double uncrossed (e.g., touching right eye with right hand) and crossed (e.g., touching right eye with left hand) commands on one's own body, and pointing to parts of the examiner's body (Dennis, 1976; Sauguet, Benton, & Hécaen, 1971).

Head (1926) and McFie and Zangwill (1960) attributed verbal and nonverbal (imitation) impairment of right–left orientation to a left-hemisphere lesion and an associated aphasia. Sauguet et al. (1971) supported this lateralization for orientation only to one's own body. They found impaired imitation of lateral movements to occur in 38% of patients with right hemisphere disease compared with 48% of aphasic patients. Luria (1966) defined the inability of patients with frontal pathology to make reversals on

the examiner, that is, to identify the right side of the examiner as the left side (especially on tasks of imitation), as ECHOPRAXIC. Benton (1969) viewed such errors as conceptual, owing to an inability to understand the relativistic nature of the right–left concept.

Tests of right–left orientation in extrapersonal space, such as the Road Map Test (Money, 1965), make greater demands on visuospatial functions and are as demanding for patients with either right or left hemisphere damage. Increasing the spatial complexity of the task (e.g., presenting a schematic figure in different spatial orientations) demands the capacity for mental rotation. Ratcliff (1979) found the presentation of an inverted manikin (upside down and back to front) especially sensitive to posterior right hemisphere involvement. Again, right–left disorientation is immediately related to the demands of the test. As in acalculia and finger localization (described earlier), the complexity of the task requires a sensitive sorting out of the components that may be selectively impaired as a function of the lesion site.

The Gerstmann Syndrome

The tetrad of symptoms described in the preceding sections (agraphia, acalculia, finger agnosia, and right–left disorientation) were, as a complex, believed to represent a distinct neuropsychological syndrome (Gerstmann, 1930). Gerstmann assumed that finger identification is central to the development of calculation. Strauss and Werner (1938) demonstrated a definite relationship between the ability to articulate the fingers and the early development of the number concept. Finger localization and calculation are presumed to be the precursors of right–left orientation and the ability to write.

In addition to the prominence of this cluster of symptoms, Gerstmann noted cases with constructional problems, word finding difficulty, mild reading difficulty, color naming problems, and/or absence of optokinetic nystagmus. The presence of any or all of these associated problems constitute a secondary syndrome implicating more extensive involvement of the parieto–occipital junction of the left hemisphere. The sudden appearance of either the primary or secondary syndrome has been associated with space-occupying lesions. Kertesz (1979) identified 9 out of 556 aphasics and controls who were observed to have the four components of the syndrome distinct from other deficits. Seven of the nine cases had a left parieto–occipital lesion, one had bilateral lesions, and one was a trauma and had a negative scan.

The infrequent occurrence of the specific tetrad of Gerstmann and the

greater frequency of the symptoms occurring separately (Heimburger, Demeyer, & Reitan, 1964) has raised questions about the existence of this syndrome. In 1961, Benton addressed this question in his paper, "The Fiction of the 'Gerstmann Syndrome.' " He concluded, as did Poeck and Orgass (1975), that the intercorrelations between the four elements of the Gerstmann syndrome were not any greater than correlations between any one element and defects not included in the syndrome. Strub and Geschwind (1974), on the other hand, argued that the infrequency of occurrence of a specific combination of symptoms is what clinically defines a syndrome. For those who accept the entity of Gerstmann syndrome, it localizes the lesion to the region of the angular gyrus of the left hemisphere.

There have been a number of reports of a developmental Gerstmann syndrome. Benson and Geschwind (1970) reported two cases of good readers having the tetrad of symptoms along with constructional difficulties. Rourke and Strang (1978) found children, referred to a clinic for deficient calculating ability, who demonstrated a pattern of deficits analogous to the Gerstmann syndrome, that is, problems in arithmetic, right–left orientation, writing, and finger gnosis, who also read at or above expected grade level. J. Schwartz, Kaplan, and Schwartz (1981) identified 22 dyscalculic students (1% of a fifth and sixth grade population). Ten of these children were found to have all of the components of Gerstmann Syndrome as well as constructional difficulties. Qualitative analysis of their strategies and errors were consistent with performance of children with a dysfunctional right hemisphere. This raises the possibility of a developmental Gerstmann Syndrome that, unlike in adults, does not implicate left hemisphere dysfunction.

Constructional Disorders

Impaired performance in producing drawings or geometric configurations is frequently referred to as CONSTRUCTIONAL APRAXIA. The original conception (Kleist, 1912), however, was reserved for those faulty productions that were not the result of either impaired visual or motor executive function, but rather a defect in the transmission of the visual information to the motor system. The early descriptions (Kleist, 1912; Poppelreuter, 1914–1917) implied this disconnection mechanism. Current use of the term constructional apraxia no longer reflects the presumed underlying mechanism. For some investigators (e.g., Arrigoni & De Renzi, 1964; Piercy & Smith, 1962), constructional disorders after right- and left-hemisphere lesions reflect the same basic disturbance except that it is more frequent and more severe in patients with right-hemisphere than left-hemisphere le-

sions. It may reflect larger right-hemisphere lesions, since left-hemisphere lesions probably come to the attention of a physician earlier because of the associated aphasic symptoms.

Benton (1969) demonstrated differences as a function of the nature of the constructional task (e.g., drawing, assembling) and concluded that constructional difficulty does not represent a unitary disorder. Even on a single constructional task, the completion represents the final integration of multiple abilities and skills rather than a single cognitive function. Goodale and Milner (1992) have distinguished between neural pathways for visual–perceptual analysis and those that mediate visually guided manipulation of objects. Disorders of planning and conceptual reasoning may also play a role.

A number of investigators have observed distinctive lateralized differences in the quality of performance. Warrington, James, and Kinsbourne (1966), and Hécaen and Assal (1970) demonstrated that patients with left-hemisphere lesions improve with practice, whereas those with right-hemisphere lesions worsen with practice. Patients with left-hemisphere lesions draw more right angles than are present in the target drawing of a cube. Patients with right-hemisphere lesions underestimate angles in a star; those with left-hemisphere lesions overestimate angles. In drawing, patients with right-hemisphere lesions tend to oversketch by producing more lines and more details, whereas patients with left-hemisphere lesions tend to oversimplify drawings and delete details. Inattention to the left side of space is far more characteristic of patients with right-hemisphere lesions. Gainotti, Silveri, Villa, and Caltagirone (1983) did not find the type or severity of aphasia to be predictive of the ability to draw objects from memory.

The Block Design subtest of the Wechsler Adult Intelligence Scales is another measure of visuoconstructive ability that is performed poorly but qualitatively differently by patients with lesions lateralized to opposite hemispheres. A study by Kaplan, Palmer, Weinstein, and Baker (1981) found that patients with lateralized lesions tend to begin working on a block design in the hemiattentional field contralateral to their noncompromised hemisphere; that is, patients with left-hemisphere lesions work significantly more often from left to right, whereas patients with right-hemisphere lesions work significantly more often from right to left. Errors en route to a final solution as well as in the final product are more prevalent in the hemiattentional field contralateral to the dysfunctional hemisphere; that is, patients with left-hemisphere lesions make significantly more errors on the right side of the design, and patients with right-hemisphere lesions make significantly more errors on the left side.

The tendency for patients with right-hemisphere lesions to use a piecemeal approach (Paterson & Zangwill, 1944), without integrating component parts, results in a remarkable inability to maintain the 2×2 or 3×3 matrix (broken configuration). Broken configurations evident in productions of patients with right-hemisphere damage were virtually absent in the productions of patients with left-hemisphere lesions. Similar qualitative hemispheric differences were also noted in drawings (e.g., Wechsler Memory Scale Visual Reproductions, Rey–Osterrieth Complex Figure; Kaplan, 1988), and in the drawing of clocks (Freedman, Leach, Kaplan, Winocur, Shulman, & Delis, 1994). Patients with left–hemisphere lesions were noted to deal more effectively with contour information than with internal features or details, whereas the reverse obtained for patients with right-hemisphere lesions. It should be noted that assessment of visuoconstructive performance in many aphasic patients is complicated by hemiparesis of the dominant hand, so that the patient is obliged to use the less skilled hand. This in itself may reduce the quality of motor realization, as it would even in normals, but the lateralized information-processing style is still clearly evident.

The California Global–Local Learning Test (Delis, 1989) provides another opportunity to observe lateralized differences in spatial information processing. This test consists of visual hierarchical stimuli, that is, a larger letter or shape constructed from numerous dissonant smaller letters and shapes; for example, a large *S* composed of numerous small *j*'s. Aphasic patients tend to reproduce the global *S*, whereas patients with right-hemisphere damage tend to reproduce the local *j*'s while distorting the global aspect of the stimulus. This dissociation is noted when these patients copy the design as well as when they recall it from memory after either a short or long delay.

The preceding illustrations underscore the importance of qualitative analyses of the strategies that patients with lateralized lesions spontaneously use to compensate for their deficits. The strategies that are observed not only inform the therapist but may be relevant for the development of both test materials and therapeutic interventions (Kaplan, 1988).

Lyon and others (Bauer & Kaiser, 1995; Lyon, 1995; Lyon & Helm-Estabrooks, 1987) have proposed that because drawing may provide an alternative representation of conceptual thought that does not depend on access to linguistic symbols, it may be possible to train some severely aphasic patients in communicative drawing as a substitute or adjunct to language. Lyon (1995) underscores the fact that in the select patients for whom this proves possible, communicative drawing must be explicitly trained and encouraged, as it is unlikely to be offered spontaneously or to generalize.

Apraxia: Disorders of Gestural Behavior

APRAXIA may be defined as a disturbance in the deliberate execution of learned purposeful movement to command, out of context, that is not attributable to elementary motor or sensory defects; incoordination; poor motivation, attention, or comprehension of verbal commands; or intellectual deterioration. Apraxia may also involve the inability to imitate meaningless gestures accurately. Liepmann (1908) distinguished between the following types of apraxia: limb–kinetic, ideomotor, and ideational.

1. Limb-kinetic. This concept is rarely invoked, as many authors have noted that it is particularly difficult to distinguish from more basic pyramidal or extrapyramidal motor disorders (Goodglass, 1993). It has been characterized as a deficit involving clumsiness and loss of finely grained movements. Loss of kinetic melody, temporal disordering, and decomposition of movement have also been described.

2. Ideomotor. Basic limb kinetics are intact but are not adequately informed by a motor plan for the target behavior. The majority of observations of apraxia could be described as characteristic of the ideomotor variety. Rothi, Mack, Verfaellie, Brown, and Heilman (1988), and Poizner, Mack, Verfaellie, Rothi, and Heilman (1990) have classified typical errors as postural, spatial orientation, and spatial movement. Limb apraxias can be divided further along the dimension of transitive or intransitive, that is, whether or not the gesture involves the use of a tool or implement (Goodglass & Kaplan, 1963). An alternative movement may be substituted for the target motor response (PARAMIMIA), or the posture may be largely correct but incomplete or spatially displaced. The responsible lesion is typically lateralized to the left, or dominant, hemisphere and/or to the corpus callosum.

Heilman and Rothi (1993) posit the existence of visuokinesthetic motor engrams, or praxicons, which are stored in the inferior parietal lobule of the dominant hemisphere. These code the spatial and temporal patterns of learned skilled movements and program the motor areas for production of gestures, and they are also crucially involved in the comprehension and discrimination of gestures. According to Heilman et al., ideomotor apraxia involves an impairment in the selection, sequencing, and spatial orientation of gestural movements. In the posterior variant, the area of the inferior parietal lobule containing the engrams is damaged, whereas the anterior type involves an anterior disconnection of the engrams from premotor and motor areas.

3. Ideational. Given a complex sequential task, with the object(s) present, patients may be able to perform each step in isolation but are unable to program the full sequence correctly. Heilman, Watson, and Rothi (1997)

review evidence for what they term CONCEPTUAL APRAXIA. Patients with this disorder are described as having lost knowledge that governs the selection or appropriate use of tools and objects. Thus, they may choose the wrong tool for a specific task, or given a tool, may pantomime or demonstrate the usage appropriate to a different tool altogether. Such patients are also likely to show deficits in other tasks involving conceptual knowledge related to tools, such as associating a tool with a related object (e.g., a hammer with a nail).

The type of apraxia is distinguished by the types of errors made, as well as by the means of eliciting the desired movement (command or imitation). Different levels of performance are seen with different body parts (buccofacial, limb, or whole body; Alexander, Baker, Naeser, Kaplan, & Palumbo, 1992). Geschwind (1965, 1975) described the consequence of a premotor and a callosal lesion as follows: An auditory command to perform a movement, such as "Show me how you would brush your teeth with a toothbrush," is comprehended in Wernicke's area; however, destruction of the premotor area necessary to program and initiate the movement precludes performance with either the right or the left hand since the information has no way of getting to the right premotor area. McCarthy and Warrington (1990), however, observed that such patients also have deficits when the task stimuli are presented visually; Geschwind's model would have to imply verbal mediation in performing the visually guided task.

Patients with a disconnection syndrome, as reported by Geschwind and Kaplan (1962) and Gazzaniga, Bogen, and Sperry (1967), are capable of performing the target movement with the right hand (left premotor region is intact), but are incapable of performing it with the left hand because the fiber tract from the left premotor to the right premotor region is destroyed. Another disconnecting lesion occurs in the left arcuate fasciculus. Here the lesion in the fiber tract deep to the parietal lobe connecting Wernicke's area to the premotor area precludes the decoded command from reaching the premotor area (as well as resulting in a conduction aphasia). Ochipa, Rothi, and Heilman (1994) identified a syndrome they named CONDUCTION APRAXIA. They described a patient with left-hemisphere damage whose performance on pantomime to verbal command was superior to pantomime imitation. They suggest that gestural imitation involves a nonlexical gesture-processing route.

Ideomotor apraxia may result from lesions in the supplementary motor, inferior parietal, and callosal, and in several reported subcortical sites, including the basal ganglia and white matter (Pramstaller & Marsden, 1996). Apraxia following a nondominant hemisphere lesion, though rare, has been reported.

Liepmann's attribution of motor programming to the hemisphere dominant for handedness, as well as the earlier localization of language to the left hemisphere (in right-handed individuals) by Broca and Wernicke, explains the frequent co-occurrence of apraxia with aphasia. A case reported by Heilman, Coyle, Gonyea, and Geschwind (1973) supports Liepmann's proposition that praxis is localized to the hemisphere dominant for handedness. Their patient was left handed and suffered damage to the motor and premotor region of the right hemisphere. He sustained left-sided hemiparesis without an aphasia and a severe apraxia involving the right limb; although this patient's intact left hemisphere was obviously dominant for language, he was apraxic secondary to the lesion of the right hemisphere (dominant for handedness). This case dramatically supports the separate functions of language and praxis and argues for their co-occurrence in aphasia as a result of the proximity of structures affected by one lesion.

Goldstein (1948) proposed that aphasia and apraxia coexist as components of a central communication disorder. Patients who could not communicate orally or in writing also could not use the gestural channel. Goodglass and Kaplan (1963) tested the notion of a central communication disorder. The finding that the severity of aphasia did not correlate with the severity of the gestural disturbance argued against a central communication disorder. The inability to imitate the gestures demonstrated by the examiner supported Liepmann's position that the aphasic's gestural deficit represented an apraxic disorder. In contrast, Pickett (1974), using the Porch Index of Communicative Ability (PICA) and 10 commonly used objects in addition to the 10 items used in the PICA, concluded that gestural ability is related to severity of aphasia rather than limb apraxia.

Poizner and Kegl (1992) have reported interesting dissociations of aphasia and apraxia among deaf patients who were fluent in American Sign Language (ASL) prior to their strokes. These patients were heterogeneous in ability to produce both meaningful (i.e., communicative but nonlinguistic) and nonrepresentational gesture to command. Corina et al. (1990) reported a globally aphasic deaf signer who was able to express and comprehend pantomime. He was not able to comprehend the ASL signs that corresponded to the pantomimes, and was noted to attempt spontaneously to use pantomime and gesture to augment communication.

Duffy, Duffy, and Pearson (1975) found pantomime recognition to be significantly correlated with auditory comprehension, naming ability, and overall linguistic competence and argued for the concept of a central communicative disorder. Opposing this view are the findings of Zangwill (1964) and Alajouanine and Lhermitte (1964), who supported the prevalence of defective pantomime recognition in aphasics but not a correlation

with the severity of the aphasia. Gainotti and Lemmo (1976) found a high degree of relationship between the comprehension of symbolic gestures and semantic errors on a verbal comprehension test. Although it was again clear that understanding symbolic gestures was more impaired in aphasics than in other brain-damaged patients, there was a minimal relationship between comprehension and production of symbolic gestures (a finding contrary to Duffy et al., 1975). Finally, Varney (1978) found that deficits in pantomime recognition always co-occurred with reading deficit (but not vice versa). Pantomime recognition was only weakly associated with auditory comprehension and naming ability. Varney concluded along with Vignolo (1969) that modality-specific factors may underlie the relationships that have been obtained (in this study, the visual modality). Despite severe inability to engage in gestural representation to verbal command, apraxic patients perform relatively well in the context of real action with implements.

Body-part-as-object (e.g., use of a body part to represent an absent implement, such as using the index finger as if it were a toothbrush and vigorously rubbing teeth with it) was commonly noted in the apraxic aphasic (Goodglass & Kaplan, 1963), and was later found by Kaplan (1968) to be a characteristic response of 4-year-old normal children. In her developmental study, a distinct developmental progression was noted in the acquisition of gestural representation of absent implements. Using the example of representing brushing teeth with a toothbrush, young children between 2½ and 4 years of age rely on deictic behavior (pointing to the locus of the action; e.g., pointing to the mouth) and manipulation of the object of the action (rubbing the teeth). At age 4, body-part-as-object is the most characteristic mode of representation. By age 8, children are pretending to hold the absent implement but the movement is too close to the object of the action (e.g., the teeth) and may degrade into a body-part-as-object response. By age 12, children are performing like adults, pretending to hold the implement and utilizing empty space to represent the extent of the absent implement. It may be inferred that the use of body-part-as-object circumvents the difficulty apraxic patients have in positioning the hand and reproducing the movement veridically. Gestural representation in dementing patients, in intellectually inferior adults, or in the elderly tends toward more concrete representation and the use of body-part-as-object as in the immature child.

Thus far we have discussed impairment of limb movements to command. Patients with arcuate fasciculus lesions as well as left premotor lesions will have similar difficulty carrying out buccofacial movements. These may include facial and upper respiratory movements, such as licking the lips, blowing, coughing, or sipping to command. Buccofacial aprax-

ia may occur in the absence of limb apraxia. As with limb apraxia, providing a real object often facilitates performance of a gesture that could not be pantomimed in the absence of the real object.

In all apraxic patients, there is a relatively spared class of movement—axial or whole body movements (e.g., stand up, turn around twice, and sit down). Geschwind (1975) suggested that axial movements are controlled by nonpyramidal systems arising from multiple regions in cortex; whereas the pyramidal systems controlling unilateral movements arise primarily from the precentral gyrus.

Global aphasic, apraxic patients have been demonstrated to have the capacity to use nonorthographic visual stimuli for comprehension as well as for communication (Gardner, Zurif, Berry, & Baker, 1976; Glass, Gazzaniga, & Premack, 1973). Helm-Estabrooks, Fitzpatrick, and Barresi (1982), guided by this body of evidence, developed a therapeutic program (Visual Action Therapy). Beginning with actual implementation, global aphasics were trained to produce symbolic gestures to represent absent stimuli. On pre–post testing, these patients showed significant improvement on PICA pantomime and auditory comprehension subtests. The results of this study hold promise for therapeutic intervention in apraxic aphasic patients.

Nonlinguistic Cognitive Abilities in Aphasic Patients

Van Mourik, Verschaeve, Boon, Paquiers, and Van Harskamp (1992) described a group of diagnostic procedures they call, collectively, the Global Aphasia Neuropsychological Battery (GANBA). The subtests of this battery represent adaptations of standard neuropsychological tests of attention and concentration, recognition memory for objects and faces, visual–perceptual tasks including perceptual matching, visual search, figure–ground discrimination, recognition of visual stimuli from novel views, recognition of audiotaped environmental sounds, and language comprehension. Van Mourik et al. (1992) concluded that globally aphasic patients demonstrated a diversity of patterns of cognitive performance. They used information gleaned from the GANBA to determine whether these patients were appropriate treatment candidates, and, if so, whether the treatment of choice should be a language-based program or a nonverbal therapy. The latter included "visual–perceptual training, visualization of a 'dialogue' with pictograms, basic drawing skills, [or] nonverbal semantic training such as categorization with pictures" (p. 491).

Biber, Helm-Estabrooks, and Fitzpatrick (1983) retrospectively studied the neuropsychological evaluations of 21 globally aphasic patients who were enrolled in the Visual Action Therapy language program. The neuropsychologist, blind to outcome, proved successful in predicting the outcome of all 21 patients on the basis of performance on *both* visual–perceptual and memory tasks. Gardner et al. (1976) found that the Picture Arrangement subtest of the Wechsler Adult Intelligence Scale (WAIS) was the best predictor of success in a visual communication program (VIC) utilizing nonlinguistic symbols. Gallagher, Fitzpatrick, Helm-Estabrooks, and Albert (1993) found significant correlations between WAIS-Revised Performance IQ measures and BDAE scores shortly after stroke as well as at follow-up intervals of up to 15 years postonset. Despite the significant correlations, initial WAIS-R parameters were less predictive of long-term language outcome than were the initial language scores.

The lesions that produce aphasia typically spare the anatomical regions that subserve long-term memory, including the mesial temporal lobe and the diencephalon. Risse, Rubens, and Jordan (1984) demonstrated that aphasic patients with left inferior frontal lobe and basal ganglia lesions were severely deficient in acquisition and retention of word lists relative to patients with posterior temporo-parietal lesions. The reverse was true for digit span, a measure of working memory, and the authors discuss their finding in terms of the dissociation between the anatomical and functional systems subserving short-term and long-term memory.

Assessment of verbal learning and memory in aphasic patients is obviously complicated by the problems they may have in comprehending the stimuli or expressing their recall of it. We have found that multiple-choice probing can frequently demonstrate relatively intact acquisition and retention of new material (anterograde memory) even in the presence of severe language impairment. Glosser, Goodglass, and Biber (1989) developed a nonverbal, visuospatial analogue to word list learning. They found that performance on this visual learning task in aphasic patients was independent of naming ability, and that even patients with moderate to severe aphasia could achieve normal learning curves and recognition scores.

Disorders of attention and executive function, particularly with lesions that involve the frontal lobe and its subcortical connections, may impair communication insofar as skills such as selective and sustained attention, integrating and analyzing incoming information, and organizing and monitoring one's own verbal production may be impaired. Perseveration, or the intrusive interference of previous responses, is also a problem for many aphasic patients. Although these processes are not linguistic per se, they may interact with any existing language deficits to undermine the

residual adaptive potential. Glosser and Goodglass (1990) found that aphasic patients with left frontal lobe lesions were significantly more impaired on nonverbal tests of executive control than were those aphasic patients whose lesions were retrorolandic or mixed. They argued that these impairments were specific to left frontal or prefrontal lesions, and were independent of language or visuospatial deficits.

Finally, consideration of aphasia-related disorders would not be complete without reflection on the emotional consequences. Depression may suppress ability to obtain maximum benefit from treatment by affecting activation, arousal, and motivation. Patients with aphasia are obviously at a disadvantage in discussing their feelings, and neurogenic changes in facial musculature may make it difficult to interpret the usual cues. Moreover, in some cases, disinhibition of affect or superficial displays of tearfulness or laughter may occur which are not reflective of the patient's true mood state. Aphasic patients can have catastrophic emotional reactions to their losses (Robinson & Benson, 1981; Robinson, Starr, Lipsey, Rao, & Price, 1985) and should be given the means to communicate these feelings so that they can be addressed therapeutically. A simple, nonverbal means for this is the Visual Analog Mood Scale, which correlates with verbal measures such as the Depression scales of the Minnesota Multiphasic Personality Inventory (MMPI) and Profile of Mood States (POMS) (Stern, Rosenbaum, White, & Morey, 1991).

It is important to evaluate how combinations of deficits may interact to create complex problems, and how treatment in one area may affect other areas of dysfunction. Improvement of an attentional deficit, for example, may have a positive impact on other problem areas, including comprehension or ability to focus on and profit from therapy. In assessing a group treatment program that emphasized functional communication skills, Bollingen, Mussen, and Holland (1993) found that Communicative Activities of Daily Living (CADL) performance improved, but the Porch Index of Communicative Abilities (PICA) did not. They noted that "the data from this study would suggest that the careful structuring of group communication intervention enables a strengthening of the nonlanguage and language cognitive processes that underlie meaningful communication treatment strategies that directly assess attention, tracking, nonverbal memory, shifting and categorization, improving those performances that are important underpinnings of the communication process" (p. 312).

In this chapter, we reviewed specific components of nonlinguistic cognitive domains that may be selectively impaired in patients with acquired aphasia. It is our hope that a fuller understanding of the dynamic interaction between these processes and the language impairment will enhance treatment, rehabilitation, and quality of life for aphasic patients.

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10

Intelligence and Aphasia

KERRY HAMSHER

Early Controversies

The Discursive Arguments

The purpose of this chapter is to identify and review issues concerning the relation between aphasia and intelligence. The debate that surrounds this topic is far from being concluded and remains a major topic for continuing research in the field of aphasia. This issue has at its heart two underlying themes: first, what is the relation between language and thought; and second, to what extent does an aphasic disorder transcend a mere disorder of speech. The first theme has manifold ramifications ranging from contested philosophic debates to questions about vocational rehabilitation for victims of cerebral disease. The second theme is a restatement of the first using the terms and concepts of clinical neurology and neuropsychology. As we shall see, these concepts of "thought" and "language" are much like Siamese twins: At their points of interface, it may not be possible to say where one stops and the other begins; yet each generally operates independently and possesses a unique character.

In Chapter 1, Benton and Anderson describe the history of concern about the effects of aphasia on intelligent thought, a concern that is deeply rooted in the earliest theories of aphasia. From those early clinical observations of patients with acquired aphasia, two major principles were derived: (a) aphasia was more than a paralysis of the muscles involved in speech and therefore must involve some mental or cognitive capacity, and (b) the problems manifested by aphasics in the activities of daily living and the limitations in communication with others in the environment seemed

greater than what could be accounted for by the speech defects alone. In response to the first observation, hypotheses were offered to link a cognitive defect with the consequent disturbances in speech. Bouillaud and Lordat, for example, supposed there was damage to the "organ of memory for words," leading to a concept of aphasia as a specific amnestic defect. Broca, Wernicke, and Lichtheim spoke of defects in association processes that link words with their referent objects or actions. Logically, this concept would allow that both memory for words and a meaningful appreciation of the environment could be preserved but dissociated in aphasia.

The second observation led to questions about the effect of aphasia on thought processes. Trousseau first emphasized the apparent impairment in thought processes in aphasia, followed by J. Hughlings Jackson and Baillarger. Pierre Marie spoke of the necessary impairment of limited but important aspects of intelligence in aphasia. Arnold Pick (1931/1973) declared that "speech symbols represent an important aid to thought" (p. 136). In describing thought processes as cascading events in which the early stages of formulation guide the later stages, Pick suggested that a deficiency in language would degrade the formulation of thought at its earliest stages and thus could give the appearance of intellectual retardation. By this comment one may suppose that Pick felt the real substance of intelligence may remain intact in aphasia but left in waiting for the return of linguistic skills. Findelnburg, Kurt Goldstein, and Head believed aphasia could be conceived as a defect in symbolic thinking. Whether printed or sounded out, words are a convenient way to refer to elements of one's environment and thus are very clearly symbols.

In some senses, these are all appealing concepts of aphasia. It is not clear, however, that any one of them can adequately account for either the variety of cognitive deficits that are associated with aphasia or the variability among aphasics in their patterns of cognitive symptomatology. For example, whereas language represents a special use of symbolic thinking—namely, "the use of symbols for purposes of communication" (Benton, 1965, p. 298)—this does not appear to be the only aspect of language that is vulnerable to brain disease. The four major syndromes of aphasia share the symptom of an impairment in naming: In Broca's aphasia, effortful and labored attempts to name result in phonemic paraphasias; in so-called conduction and Wernicke's aphasia, naming is non-effortful and fluent, but there are prominent phonemic and semantic paraphasic errors; in nominal aphasia, paraphasic errors are rare and the defect in naming appears to represent a failure in word retrieval (Goodglass, 1980). Thus, one can see how difficult it would be to explain these various characteristics by one mechanism.

Empirical Formulations

An understanding of the contribution of the right hemisphere to the performance of certain intellectual tasks or functions was largely missing during the early stages of the development of a concept of aphasia. This lack of appreciation for the role of the so-called minor hemisphere persisted despite the contributions of J. Hughlings Jackson, who at the time of Broca's discoveries was suggesting that symptoms of visuospatial impairment, which he called "imperception," could result from right-hemisphere lesions, particularly in the posterior zone (Benton, 1977a). Yet, even Jackson lacked a formal theory of intelligence, and none of the early theorists proposed a view of intelligence as an objective and measurable behavioral capacity. The work of Weisenburg and McBride (1935) ushered in both a new approach to the question of the relation between aphasia and intelligence and a new speculation. Their objective assessment of both verbal and nonverbal cognitive performances in aphasic, nonaphasic brain-damaged, and control subjects led them to believe that individual cognitive styles or strategies must play a role. Persons accustomed to solving problems by verbal means might, by this formulation, be the ones most likely to exhibit nonverbal defect in the context of aphasia. Weisenburg and McBride's contribution marked the onset of a new era in which formal conceptualizations of intelligence, and the method of assessment, were brought to bear on the question of the effect of aphasia on intelligence.

Intelligence

The Concept of Intelligence

A definition of the concept of intelligence is at once both controversial and complex. To emphasize here the points of disagreement would be self-defeating, for we cannot realistically evaluate the issues at hand if a consensus definition is circumvented. At the same time, some of the complexity must be preserved if we are going to do justice to the term and meaningfully test the limits of the concept of intelligence in the context of aphasia.

Sir Cyril Burt (1955) ascribed the origin of the concept of intelligence to Plato and Aristotle and credits Cicero with the coinage of the term. The use of tests to assess intelligence, as well as the application of statistics to these data, was introduced by an English scientist, Sir Francis Galton. Galton was interested in supporting the theories of his first cousin, Charles Dar-

win, by demonstrating that the principles of hereditary descent applied to intellectual as well as physical attributes. To this end he published his study *Hereditary Genius* in 1869 (Galton, 1887). But as Zangwill (1964) pointed out, the real work on the exploration and development of this concept began with the development of intelligence tests for the purpose of addressing socioeducational problems. This work was begun by Alfred Binet, a French lawyer and natural scientist by education, who became a psychologist largely through self-tutoring.

A different conception of the term INTELLIGENCE is sometimes encountered among the general public, where it is taken to mean a state of above-normal thinking capacity. In scientific psychology, intelligence is conceived as a largely but not exclusively biologic characteristic that is expressed in behavior, which varies from one person to the next and accounts for some individual differences in behavior. Therefore, it is a quantitative concept, as are the concepts of height and weight. At the same time, there appears to be some naturally occurring upper limit to human intelligence, but this limit is not explicitly defined. Unlike height and weight, intelligence cannot be directly observed, touched, or measured in physical terms. It is more well defined and less arbitrary than such concepts as beauty and creativity. Also, the characteristics one looks at to assess intelligence in a subjective fashion are more consistent than, say, the characteristics that might be employed to assess "athleticism" in ping-pong players, football players, gymnasts, and runners.

A distinction is made by most authorities between the application of the concept of intelligence to represent one's ability versus one's actual performance. A highly intelligent individual could on some occasions perform poorly on an intelligence test for a variety of reasons, such as anxiety or preoccupation. Clearly one could fake a bad performance on an intelligence test, and obviously such an event would not in any real sense lower that person's intellectual competence. Thus, this distinction between capacity and performance is crucial and something to be addressed later in this chapter.

Wechsler (1958) described general intellectual ability as the "global capacity of the individual to act purposefully, to think rationally, and to deal effectively with his environment" (p. 7). Intellectual behavior or functional intelligence, he said, depends on general intellectual ability plus the way in which specific cognitive abilities are combined and such nonintellectual factors as the person's drive and the incentive offered by the situation in which intelligence is being assessed.

Thus, intelligence is a construct that implies an underlying reality, but this reality must be inferred from behavioral observations (Wechsler, 1971). The parallel to this is a working definition of intelligence as a complex trait

that is measured by intelligence tests (Wechsler, 1971). Because psychology has developed methods to determine whether a particular test is a measure of what we intend to call intelligence, the circularity of the working definition is no longer problematic. These methods derive from our current understanding of the structure of intelligence.

The Structure of Intelligence

Two differing views of the structure of intelligence were developed during the first half of the twentieth century. One view, which was articulated by British psychologist C. Spearman (1927), holds that intelligence is a unitary trait that is expressed to greater or lesser extent on most any cognitive task. The opposing view is that general intelligence is merely the sum (or average) of a collection of different primary abilities. This point of view was championed by American psychologist L. L. Thurstone (1938). Since Thurstone's primary abilities are not independent of each other and, in fact, are intercorrelated, this position is not fundamentally different from Spearman's position that besides the general factor there are also specific intellectual factors (Piercy, 1969). Modern psychology therefore views intelligence as having both unitary and multiple or factorial aspects. The history that led to this conclusion is described by Matarazzo (1972, pp. 24–62).

Intelligence is generally conceived as being organized in a hierarchy. This notion is borrowed from the facts of evolution, especially with regard to the development of the central nervous system. According to this model, in the normal brain, general ability exerts a downward influence on the next level of organization, which consists of several major or group factors (primary abilities). The group factors, in turn, comprise several specific factors that may be specific to the type of task or to the method of assessing performance. Whether there are two, three, or more levels of organization within the various spheres of cognitive activity has not been fixed in fact or theory. The question of the number of levels of organization is an arbitrary one, as the answer varies depending on the number and types of cognitive behaviors sampled, the methods of assessment, and the type of statistical analysis applied.

A concrete example may elucidate the theoretical view of the structure of intelligence. First, an extensive battery of cognitive tests is administered to a cross-section of the general population. The finding that all the test scores correlate positively with each other provides the basis for inferring the presence of a general factor of intelligence, often called *g* after the manner of Spearman. When the influence of each test's correlation with *g* is statistically removed, one can determine to what degree two tests are related to each other in a way not accounted for by their shared correlation with

g. This leads to the observation that there are clusters of tests that tend to correlate with each other to a high degree and show a low correlation with the remaining clusters of tests. This clustering is how major or group factors are identified. To determine an individual test's correlation with the statistical definition of *g*, each subject's score on that test is correlated with the subject's score on the entire battery. Examining these correlations, one will find that some tests correlate with (predict) *g* better than others. Those tests that correlate most strongly with *g* are considered the best representatives of measures of general intelligence within the test's group factor. Among verbal tests, vocabulary and general information show this strong relationship with *g*. Among nonverbal intellectual tasks, Raven's Progressive Matrices and the Block Design subtest from the Wechsler Adult Intelligence Scale-Revised (WAIS-R) are among the best predictors of *g*.

The Validity of Intelligence

This psychometric framework allows behavioral scientists to develop and validate measures of intelligence within various cultures. Thus, when we speak of measures of general intelligence, we are denoting measures that predict how well a person will likely perform on a wide variety of cognitive tasks. Although eloquently conceived, this view of intelligence would be rather trivial if it were not also true that intelligence, defined and measured by objective means, predicted or coincided with other real-world events and biologic patterns.

When the concept of general intelligence is expressed as a summary score derived from an intelligence test battery, then the technical term INTELLIGENCE QUOTIENT (IQ) may be used. A quotient score is a way of expressing performance level in a standard fashion. By convention, quotient scores have a mean of 100 and a standard deviation of 15 points. The IQ score states a person's standing relative to some reference group. Since performance on intelligence tests progressively increases with age during the developmental years and may progressively decline with age during late life, separate reference groups are needed for various age categories.

Childhood IQ's starting from age 7–8 correlate with adult IQ in the range of $r = .70–.85$, suggesting a fair degree of stability. Childhood IQ's correlate with adult occupational level ($r = .40–.60$). Similarly, adult IQ scores correlate with adult educational and occupational levels in the same range (approximately $r = .50$) (McCall, 1977). We may infer from these findings that, although performance on an intelligence test battery seems to have an important relationship to adult education and occupational achievements, other factors must also be involved that are not accounted for in the IQ score. Because our concept of intelligence does not require that

it account for everything, these summary findings reported by McCall can be considered supportive of the validity of measured intelligence.

Substantial evidence indicates that objective measures of intelligence are in part under hereditary control. For example, the highest correlations in IQ's are found among identical twins (about $r = .90$). As expected, there are highly significant but smaller correlations between the IQs of siblings and between parents and children, all of whom share fewer genes than identical twins. There is also evidence that, although these correlations are attenuated when the pairs in these correlations live apart from each other, the pattern of correlations (e.g., identical twins vs. fraternal twins) remains much the same. Moreover, the magnitude of these correlations are similar to those of other complex, multiply determined traits, such as height and weight. Since the IQ is not a perfectly reliable score, varies within the same individual on different occasions, and is subject to the influence of such personality and environmental factors as drive and incentive, the obtained correlations may be considered minimal correlations. The magnitude of the contributions of heredity and environment is a question that is still at issue; for our purposes, knowing that the IQ is in part under hereditary control is sufficient with regard to the question of the validity of the concept of intelligence expressed as an IQ score (Matarazzo, 1972, pp. 298–317; Vandenberg, 1971).

So far, we have seen that intelligence as a concept has progressed (in a scientific sense) from an abstract concept to a concrete IQ score. This score appears valid in the sense that IQ scores tend to covary with the trappings of the abstract concept of intelligence, such as educational and occupational attainment and performance, and heredity. Further evidence of its validity comes from the observation of its susceptibility to decline with brain damage.

Intelligence and Brain Disease

When speaking of brain disease, one may be tempted to think of something akin to an inflammation, but DISEASE is a broad term that carries no particular implication for the etiology of the diseased or abnormal state. Brain disease may be acquired through such mechanisms as heredity, brain tumors, prenatal trauma or metabolic disorders, postnatal vascular events such as stroke, a shearing of the brain from sudden rotation, and missile wounds such as from a gunshot. Brain disease also can be a secondary consequence of a disease of another organ system, such as renal failure or liver disease. Likewise, the term LESION is not a specific term. A lesion is a site of abnormality. It may be visible radiographically because it alters brain

structure or causes a breakdown in the blood–brain barrier, but this is not required. A lesion may also be said to be present as a result of microscopic changes in brain tissue or even changes in intracellular metabolism. For the most part, however, the kinds of lesions referred to in studies of the effects of focal brain lesions on intelligence are ones that have been demonstrated radiographically (on x-ray) or through electrophysiology (i.e., on the electroencephalogram), or have been visualized by a neurosurgeon and occasionally a neuropathologist.

General Intelligence and Brain Disease

Karl S. Lashley, a physiological and behavioral psychologist, brought together psychological and statistical concepts of intelligence, neurological concepts of dementia and mental retardation, and some sparse neuroanatomical observations. From this mixture of data and conjecture, he enunciated two major principles: EQUIPOTENTIALITY and MASS ACTION. EQUIPOTENTIALITY means that the cerebral cortex is considered undifferentiated in its contribution to the performance of intelligent behavior; that is, the contribution of one section of cortex is presumed to be as good as, and equal to, the contribution of some other section. MASS ACTION means that all regions of the equipotential cortex work together, en masse as it were, to produce intelligent behaviors so that a loss in efficiency consequent to brain damage will be proportional to the loss in cortical mass (tissue) regardless of its locus. According to Lashley, these principles applied only to nonlocalized behavioral functions such as learning and intelligence. Lashley (1929) supported these concepts with data on the maze-learning performances of rats in relation to the locus and amount of cortical tissue that was surgically removed. By today's standards, several of his correlations would not be considered statistically significant (reliably greater than zero), so in retrospect his arguments were not as sound as they were thought to be at the time. These principles had value, however, in that they generated research in attempts to defend or refute Lashley's laws.

In a study that was the human analogue of Lashley's classic study, Chapman and Wolf (1959) correlated IQ scores with the neurosurgeon's estimated amount of cerebral tissue removed during operations for the treatment of brain tumors and arteriovenous malformations. Evidence for mass action was found with the stricture that the relationship between intellectual and cerebral tissue loss was much more firm in those patients in whom the cortical excisions were postrolandic (involving the parietal, occipital, and temporal lobes). In an equally monumental study, Blessed, Tomlinson, and Roth (1968) examined the brains of deceased demented patients most of whom suffered from Alzheimer's disease, and counted the number of

senile plaques found in several brain regions. Senile plaques are present in excessive amounts in the brains of demented patients relative to age-matched nondemented controls. Prior to these patients' deaths, they were administered a battery of cognitive tests, and behavioral ratings were obtained from relatives. These investigators found a significant but modest correlation between mean plaque count and scores on the mental test battery; the plaque count correlated more highly with the ratings of a decline in the patients' personal and social habits. The difference in the magnitude of these correlations may be due to an artifact of assessment, since the behavioral ratings were a direct rating of decline in behavioral status, whereas the mental test scores may have been partially confounded with premorbid ability level. Nevertheless, these and similar findings lend credence to the notion that some summary index of acquired intellectual impairment derived from a battery of cognitive performances provides at least a rough index of the status of the cerebrum (Benton, 1980; Loeb, Gandolfo, & Bino, 1988), particularly the posterior association areas in the parietal and temporal lobes (Duara et al., 1986; Foster et al., 1983).

Focal Brain Lesions and Intellectual Performance

So far we have seen how the concept of intelligence as having both multifactorial and unitary aspects developed from studies of neurologically normal individuals. When a broad array of cognitive abilities are sampled in the investigation of general intelligence, one frequently observes individual patterns of cognitive performance, that is, areas of relative strengths and weaknesses as opposed to equivalent scores across all samples of cognitive performance. Because of what was earlier described as a downward influence of general ability on more specific abilities, if two individuals have broadly separated IQ scores, even in the case where one person's weakness is the other's strength, the person with the lower IQ will seldom surpass the higher IQ individual on any of these more specific intellectual factors. Another way to view this is to say that to some extent particular abilities, such as spatial, verbal, or numerical abilities, are dissociable within the general population. Some evidence suggests that an individual's pattern of specific abilities in the context of their general ability level plays a role in occupational selection. For example, on average, architects may outperform mechanics on measures of general ability, but they share a relative superiority in mechanical and visuoperceptive skills compared with other abilities such as numerical or verbal (see Matarazzo, 1972, pp. 168–174).

With *g* as a summary statement of intellectual standing in the normal population, the concept of dementia may be viewed as a pathological

counterpart to *g*. In behavioral terms, dementia denotes a generalized decline in all aspects of cognitive functioning, which is neurologically represented by widespread cerebral disease or dysfunction (Benton, 1980). In short, dementia implies a significant decline in *g* from some previous (premorbid) level due to brain disease. Between these two extremes of an intact and devastated cerebrum are the effects of focal brain lesions.

The WAIS, like its predecessor, the Wechsler-Bellevue Intelligence Scale, and its revised version, the Wechsler Adult Intelligence Scale—Revised (WAIS-R), comprises 11 subtests covering a variety of cognitive abilities. Scores from six of the subtests are grouped to yield a verbal IQ score (VIQ), and the remaining five subtests are grouped to yield a performance IQ score (PIQ). The full-scale IQ (FSIQ) represents the sum of performances across all subtests. Several statistical studies using factor analysis (see Matarazzo, 1972, pp. 261–276) suggest that the VIQ is composed of two factors, one of which could be called verbal–conceptual abilities (information, comprehension, similarities, vocabulary) and the other attention–concentration (arithmetic reasoning, digit span). The PIQ is composed of tests assessing perceptual–constructional abilities (block design, picture arrangement, object assembly, picture completion) and psychomotor speed (digit symbol substitution).

The selection and grouping of the WAIS subtests into the verbal and performance scales was largely done on a rational rather than an empirical basis. Although not specifically designed to detect or localize brain disease, this test battery has some usefulness along these lines (Benton, 1977b; Fogel, 1964; McFie, 1976; Wilson, Rosenbaum, & Brown, 1979). For the sake of argument, the VIQ and PIQ will represent two major intellectual factors. Studies of the WAIS performances of patients with focal or at least largely unilateral brain lesions suggest that the lesions of the left hemisphere often result in a VIQ deficit, whereas lesions of the right hemisphere result in greater impairment of the PIQ. Not all studies are in agreement with this summary statement, but the trend has appeared often enough in many separate clinical settings that it has become accepted with qualifications (G. Goldstein, 1974; Kløve, 1974; Lezak, 1983, pp. 181–224; Smith, 1975; Walsh, 1978, pp. 282–331). Furthermore, McFie (1960, 1969; McFie & Thompson, 1972) reported specific patterns of deficits on the Wechsler subtests that are associated with the intrahemispheric locus of focal brain disease (e.g., frontal vs. temporal vs. parietal lobes). Although these latter findings with the Wechsler tests have not been fully substantiated, the fact that patterns of specific cognitive deficits may be associated with lesions in specific brain areas (foci) is well established (McFie, 1969; Newcombe, 1974; Piercy, 1964; Walsh, 1978).

These illustrative points help to build a conceptual framework for un-

derstanding how focal brain lesions may affect intellectual performance. First, depending on its locus, the lesion may severely compromise a given test performance. Because that test shares some properties with the other test with which it is factorially grouped—which is the nature of factorial grouping—the effect of that lesion may be seen as partially affecting related test performances. Both the specific test defect and shared deficits on related tests would act to lower the major factor or test group score (e.g., the PIQ). This, in turn, would result in a lowering of the summary or general intellectual score. According to this model, in pathological conditions, specific cognitive defects may exert an upward influence through the structure of intelligence, and its influence would tend to be diminished at higher stages in the hierarchy. To take the analogy one step further, suppose there were a single test that incorporated several of the more specific cognitive abilities, all of which were required for the successful performance of the task. First, in the general population, we may expect such a task to have a high correlation with *g* because of its multifactorial composition. Second, we might expect that focal lesions in different regions of the brain may impair test performance because of impairment of one or several of the more specific abilities incorporated in the test. Studies of the effects of focal brain lesions on cognitive performance may also provide an alternative to factor analysis to identify the structure and hierarchical organization of intellectual abilities.

It has been asserted that when *g* is conceived as the composite sum of various performances that individually may be sensitive to aphasic, apraxic, and agnostic disorders, it may not be possible to assess general intelligence meaningfully in the presence of cerebral disease (Messerli & Tissot, 1974). For instance, a patient with a premorbid IQ of 110 has suffered a stroke resulting in aphasia. After the stroke, this patient obtains a VIQ of 78 and a PIQ of 108 with a resulting FSIQ of 93. Can we say the FSIQ represents a fair and adequate assessment of the patient's general intelligence? It certainly would not reflect the patient's spatial strengths or the severity of the verbal deficits. At this point we reach a theoretical and empirical impasse, for to date we are without an adequate conceptualization of intelligence that can accommodate the findings obtained from normal individuals, demented patients, and patients with focal lesions. As an attempt to arrive at a pragmatic solution, the custom is to use an IQ score derived from nonverbal tests for patients with aphasic disorders and to use a measure of verbal IQ for patients with visuoperceptive or spatial disorders. The supporting reasoning is that specific cognitive defects, such as aphasia or constructional apraxia, are considered to be acquired disabilities much like acquired blindness, deafness, or hemiplegia, which interfere with the usual method of assessing general intelligence.

Language and Intellectual Development

Deafness and Illiteracy

Because of the intimate relationship between language and thought in our everyday experiences, it may take a moment of reflection to disengage the two concepts. True, much of our thought processes are expressed verbally, as in the classroom or when writing an exam, or in talking to ourselves to work through solutions to various problems (so-called inner speech). But we can also recall situations and social relations in the form of visual memories, like silent movies, and in this mode we may use visual imagery to replay and rearrange events in our minds. We may find the best route on a map by visual inspection with the aid of spatial judgments, and we may conceive of repairing an automobile engine or an electric appliance without the aid of words, but not without thought. To refrain from attempting to put a large square peg through a small round hole surely represents not only perceptual discrimination but also some form of conceptual thought (Piaget, 1936).

Through the use of pantomime, we can communicate nonverbal thoughts to others, although, granted, this may be a tedious and inefficient process compared with oral speech. Infrahuman animals, such as dogs, can be trained to perform many tasks, for example, to fetch a stick and return it to the master in exchange for a pat or some treat. When the same dog spontaneously brings an object in an effort to initiate the game or to receive a reward, we must assume this, too, represents thought as well as learning in the absence of language. Yet those examples do not reveal what level of complexity can be achieved by humans without the aid of words.

Pitner and Lev (1939) compared the intellectual performances of partially deaf children and hearing children in grades 5–8. The mean performance of the hearing-impaired children was 8 IQ points below that of normal children on verbal intellectual measures but only 3 IQ points lower on nonverbal tests. Upon a review of the literature, Furth (1964; Furth & Youniss, 1975) noted that on a variety of tests of reasoning and problem solving, deaf children are often able to perform very closely to the level of hearing children of the same age. Although the retardation in oral and written language acquisition that results from deafness is a formidable obstacle, the many points of intellectual similarity between the deaf and their hearing peers would seem to negate the necessity of language for the development of normal, or near normal, intelligence and conceptual thinking. It has also been noted that environmental circumstances may be pivotal in the degree to which deafness and other forms of sensory deprivation may retard or place limits on learning processes (Kodman, 1963).

One must ask to what extent others in the deaf person's environment have tried to communicate using preserved sensory channels, such as the visual, tactile, and proprioceptive modalities. The developmental history of Helen Keller provides strong testimony to the importance of this environmental issue.

The assumption that deaf children, who are without oral or written language, actually lack ANY form of language, however, may not be valid. Since deafness is a peripheral sensory impairment, there is no reason to suppose that the deaf lack or have an impairment of the neural structures that subserve language in the hearing and speaking population. Pilot observations of deaf children between 1.5 and 4 years of age who have not been exposed to a manual sign language suggest they may spontaneously develop their own structural sign system that contains the basic properties of spoken language (Goldin-Meadow & Feldman, 1977). The lack of aural exposure to language with the consequent failure to develop oral speech or its graphic representation may not preclude the development of the fundamental properties of language or the capacity for intelligent thought.

Illiteracy (due to social rather than intellectual restrictions), represents a similar but more limited form of environmental deprivation for an aspect of language in the context of presumably normal neurological mechanisms to acquire it. Despite the incompleteness of the illiterate's language repertoire, thinking, linguistic processing, hemispheric specialization for language, and vulnerability to neurological impairments of language, for the most part, do not appear to differ from those of literate individuals (A. R. Damasio, Castro-Caldas, Grosso, & Ferro, 1976; A. R. Damasio, Hamsher, Castro-Caldas, Ferro, & Grosso, 1976; H. Damasio, Damasio, Castro-Caldas, & Hamsher, 1979).

Developmental Aphasia and Intelligence

DEVELOPMENTAL ASPHASIA (or CONGENITAL APHASIA) is a term used to describe the condition in which a child shows a relatively specific failure in the acquisition of language functions and manifests abnormalities either in expressive speech alone or in both comprehension and oral speech (Benton, 1964). With rare exceptions—for instance, congenital auditory imperception (Worster-Drought & Allen, 1929)—language functions are similarly affected in the auditory, visual, and tactile modalities. By definition, language abilities must be more severely impaired than other cognitive functions so as to distinguish this specific syndrome from general mental impairment (mental retardation or amentia). Likewise, other causes for a failure to exhibit normal language for age must be excluded (Benton, 1964; Zangwill, 1978), such as deafness or more pervasive neuropsychiatric dis-

ease in which disturbances in verbal communication represent but a subset of core symptoms, as in childhood autism (A. R. Damasio & Maurer, 1978).

It is reasonable to query whether the criteria for this behavioral diagnosis begs the question of intellectual deficit in the context of developmental aphasia. To a limited extent it does, since investigators are not likely to argue points of concern to developmental aphasia using data from subjects who are also severely impaired in nonverbal intellectual performance. Also, at very low levels of cognition, numerous technical problems adhere to mental measurements. Inasmuch as the diagnosis requires only a relative disparity between verbal and nonverbal abilities, rather than some absolute level of nonverbal intelligence, the case material reported in the literature can still be informative on this point.

The issue of the status of cognitive functioning in the presence of developmental aphasia is as complex as in the case of adult acquired aphasia. The rarity of the childhood disorder makes it particularly difficult to arrive at statistically reliable empirical formations. The failure of investigators to describe adequately the symptomatology of the subjects under study makes comparisons across different studies tenuous at best, as does the failure to report or account for demographic variables that influence expected intellectual values, as discussed earlier in the section on the validity of intelligence. The indiscriminate lumping together of children with quite variable patterns of speech and language impairments certainly clouds and may confound experimental findings as well. Two broad classifications are minimally indicated: children with primarily expressive speech defects and children with expressive and receptive language deficits (Benton, 1978).

From the available findings, we may conclude that children with developmental aphasia (or developmental dysphasia), in addition to speech defects, are impaired in verbal intelligence relative to their performance on nonverbal intellectual measures and are more variable than normals in their nonverbal IQ's, which tend to be lower than expected given the children's family backgrounds (Benton, 1978). Some of these children do obtain high average to superior nonverbal IQ's, but a larger proportion are in the low average to dull normal categories. Although the evidence is insufficient to make a definite statement, generally children in the primarily expressive speech defect category show little impairment in nonverbal intelligence, whereas those in the receptive–expressive group are at much greater risk for these additional deficits.

Benton (1978) reviewed three hypotheses to account for nonverbal intellectual deficits in developmental aphasia. The first view holds that the two types of deficits are fundamentally independent. The extent to which

developmental aphasics are impaired in nonverbal intelligence depends on the extension of the underlying neurological disease to areas beyond the language zone. This places developmental aphasia on a neurological continuum with mental retardation, the latter manifesting cognitive symptoms of bilateral and more symmetric hemispheric dysfunction.

The second hypothesis is similar to that discussed earlier in the section on the concept of intelligence, namely, that developmental aphasia is a handicap that limits and may distort the child's appreciation of his or her environment and his or her communication with others and, in turn, this may have a retarding influence on intellectual development. This implies that through special environmental intervention the handicapping factor may be minimized. On the whole, the evidence does not strongly support this concept. Yet there is a case report by Landau, Goldstein, and Kleffner (1960) of a child with receptive–expressive language impairments who showed a 19-point increment in nonverbal intellectual performance following intensive and protracted language training.

The third hypothesis holds that developmental aphasia represents the expression of an impairment of a more general or higher order cognitive function that is not strictly verbal and may encompass certain nonverbal abilities. Accordingly, the language deficits are not seen as the core disturbance, but rather as a reflection of a disability in a more major cognitive function. This position finds support in the case of the receptive–expressive form of developmental aphasia in which a higher-level, auditory–perceptual deficit is held to be primarily responsible for the failure in language development (Benton, 1964; Tallal & Piercy, 1978; Worster-Drought & Allen, 1929). However, agreement on the specificity of the defect to the auditory modality is controversial (Zangwill, 1978), and others insist that developmental aphasia is specifically a defect in the structure of language (Cromer, 1978).

It is well known that language is acquired on the basis of audition and that the infant must first learn that the mother's spoken language has symbolic significance before the instrumental use of expressive language develops. It is fundamental that we must first be able to perceive something before we can come to know it, let alone make use of it for our own purposes. It must be very frustrating and perhaps confusing to children with expressive developmental aphasia to be able to hear and come to understand language without being able to imitate it faithfully and effortlessly. Considerably more sympathy and understanding must be held out for the child with receptive–expressive developmental aphasia who is deprived of these early experiences demonstrating the symbolic and communicative uses of language. Furthermore, because "communicating with oneself is an important aspect of thinking and that to this extent language is also a

tool of thinking" (Benton, 1965, p. 299), it is not surprising that a receptive–expressive disorder will hamper the development of nonverbal problem-solving skills. What is remarkable is how little impairment some of these children show. These observations suggest the tentative conclusion that the neural mechanisms that subserve nonverbal intelligence must be independent of those that subserve language. However, inasmuch as the two cerebral hemispheres and their various lobes work in concert (Zangwill, 1974)—usually harmoniously—brain lesions affecting language operations, particularly receptive ones, which have the consequence of disrupting internal communications may also have a disharmonious impact on nonverbal operations. To overcome this, one may have to learn not to attend to faulty "verbal" insights and selectively rely on nonverbal perceptions, reasoning, and innate intuitions. This is likely a far easier task for the deaf child, who has the neurological mechanisms for language, than for the aphasic child, whose apparatus for language is partly nonfunctional or functioning abnormally.

Acquired Aphasia and Cognition

Performance on Tests of Intelligence

Weisenburg and McBride (1935) provided sound evidence that some aphasics show defects in visuoperceptive and spatial abilities and thus seem to have acquired cognitive deficits that exceed the concept of aphasia as strictly a disorder of language. On the other hand, Weinstein and Teuber (1957) reported that the patients who showed the greatest deficit on the Army General Classification Test in their studies of soldiers with missile wounds were those with left temporoparietal lesions, some of whom were aphasic and some of whom were not. Perhaps, then, these extralinguistic symptoms are not so much an expression of aphasia as they are a coincidence of anatomy. If there were areas in the left hemisphere that mediated nonverbal cognitive abilities and if these were situated near the language zone (e.g., Newcombe, 1969, pp. 98–102) or overlapping with it (Basso, De Renzi, Faglioni, Scotti, & Spinnler, 1973; Goodglass, 1974), then it would seem that some aphasics are at risk for extralinguistic cognitive deficits by virtue of the size and locus of their lesions. This formulation would deny that these extralinguistic defects are psychologically (i.e., functionally) related to the aphasic symptoms. Such cognitive symptoms could be as incidental to aphasia as are visual field defects, hemiplegia, or sensory–discriminative impairments. If aphasia, or some forms of it, were psychologically related to nonverbal mental impairment, then language

may be viewed as having some paramount role in intelligent thought—perhaps. If not, as suggested by the anatomical explanation, then we could conceive of the cerebrum as composed of several compartments, each of which subserves some different and relatively independent primary mental ability. To try to link aphasic symptoms to nonverbal cognitive deficit would be pure folly if the anatomical hypothesis were correct.

Many investigators have reported that aphasics are more impaired than nonaphasic left-brain–damaged patients on the nonverbal portions of the Wechsler intelligence scales (Orgass, Hartje, Kerschensteiner, & Poeck, 1972). Particularly, it appears to be those aphasics who manifest signs of constructional apraxia who are at greatest risk for nonverbal intellectual impairment (Alajouanine & Lhermitte, 1964; Basso, Capitani, Luzzatti, & Spinnler, 1981; Borod, Carper, & Goodglass, 1982). This association between constructional apraxia and nonverbal intellectual impairment seems to hold, however, for patients with both left- and right-sided lesions (Arrigoni & De Renzi, 1964; Kløve & Reitan, 1958), and therefore is not peculiar to aphasia. Given that the Block Design subtest from the WAIS is often employed to assess constructional praxis (Warrington, 1969), it is not clear how one can separate constructional praxis from nonverbal intelligence. It is also not clear that constructional praxis is as unitary a concept as some clinical investigators would have us believe. The various ways an examiner may test for constructional apraxia, such as by having the patient copy drawings or geometric designs or build forms out of sticks and models out of blocks, are not highly correlated in brain-damaged patients (Benton, 1969). Each one of these tests for apraxia may be tapping one or more specific abilities that belong to a more general spatial factor which may not be fundamentally different from the spatial factor comprising the nonverbal intellectual measure. Therefore, there is no compelling reason to attach special significance to the observation that the symptom of constructional apraxia is predictive of impairment in nonverbal intelligence.

Raven's Progressive Matrices (RPM) test has been demonstrated to be a measure of general intelligence obtained through nonverbal means. The test requires subjects to examine a visual array from which a subsection appears to have been cut out. By means of either an oral or a pointing response, the subject indicates which of several multiple-choice alternatives is the correct one. Some items involve the simple completion of a pattern, such as diagonal stripes, and other items incorporate a progression or sequence of figures, such as the systematic rotation or elaboration of figures, which must be discerned and understood in order to make the correct choice. This task calls on various cognitive abilities, including visuoperception, abstract reasoning, spatial relations, counting, and mental flexibility.

Significant impairment in the performance of the RPM test is seen in patients with both right- and left-hemisphere lesions, particularly in association with constructional apraxia (Arrigoni & De Renzi, 1964; Zangwill, 1975). In right-hemisphere disease, impairment on this task is associated with defects in block building and visual pattern matching, but not in left-hemisphere disease. Among patients with left-hemisphere damage, impairment on the RPM test is associated with the presence of aphasia (Basso et al., 1973), and among aphasics, such defects are primarily restricted to patients with language comprehension impairment (Archibald, Wepman, & Jones, 1967; Costa, Vaughan, Horwitz, & Ritter, 1969; Zangwill, 1969). However, there are patients with severe receptive language deficits who may perform rather well on this task (Kinsbourne & Warrington, 1963; Zangwill, 1964).

Constructional Apraxia

Constructional apraxia was originally viewed as a symptom of cerebral disease in the left posterior quadrant, but numerous studies have since shown that patients with lesions in the right posterior quadrant have the highest incidence and produce the most severe form of this cognitive symptom (see Benton, 1967; Warrington, 1969). When present, it is often associated with general mental impairment (Benton, 1962; Benton & Fogel, 1962). Although general mental impairment may result in the appearance of constructional apraxia, this is not a consistent consequence, and constructional apraxia may occur as a relatively isolated cognitive defect, that is, outside the context of general mental impairment. Obviously, there is no relationship between language impairment and constructional apraxia among patients with right-hemisphere lesions as there is in patients with left-hemisphere lesions. Specifically, in left-hemisphere disease, constructional apraxia is associated with the presence of receptive language impairment, and the more severe the receptive disorder, the more likely constructional apraxia will occur (Benton, 1973). The relationship between constructional apraxia and receptive language disorders with left-hemisphere disease may be described as a one-way relationship. There is a high probability of receptive impairment when constructional apraxia is present, but only about one-half of patients with moderate to severe comprehension defects manifest constructional apraxia (Benton, 1973). This probabilistic relation between the severity of the linguistic defect and the likelihood of constructional apraxia, while consistent with the notion of a functional relation between the two, does not contradict the anatomical hypothesis described earlier. The size of the lesion may be another structural

factor that relates to both the locus of the lesion and the severity of the language comprehension defect (Borod, Carper, Goodglass, & Naeser, 1984).

Visuoperceptive and Visuospatial Performances

Nonverbal intellectual measures, such as the performance scale from the WAIS or the RPM test, as well as measures of constructional apraxia, are all in part dependent on visuoperception. Whether on the left or on the right, brain lesions producing constructional disabilities typically result in associated visuoperceptive deficits (Dee, 1970). One standardized visuoperceptive task is a facial recognition test that calls for the matching of photographs of unfamiliar persons taken from different angles and under different lighting conditions (Benton, Hamsher, Varney, & Spreen, 1983). In patients with focal brain lesions who show no evidence of general mental impairment, defective performance on this test is associated with right-hemisphere lesions and with lesions of the left hemisphere but only in the context of an aphasia syndrome in which language comprehension is significantly impaired (Hamsher, Levin, & Benton, 1979). Aphasic patients without significant comprehension deficits as assessed by objective tests (Benton & Hamsher, 1989) and nonaphasic left-hemisphere–damaged patients perform on a par with hospital control subjects who are without history or evidence of neurological disease. Similarly, a disturbance in perceptual association involving colors is associated with aphasic impairments in language comprehension and not with "pure" measures of color perception (such as color matching or color discrimination); the opposite pattern is obtained with right-hemisphere lesions (De Renzi, Faglioni, Scotti, & Spinnler, 1972; De Renzi & Spinnler, 1967). The ability to associate colors with objects may be assessed by having patients select colored pencils to shade a drawing of, say, an apple, or to point to colors on a multiple-choice array. Interestingly, impairment in this type of performance in the setting of aphasia is correlated with deficits in conceptual performances rather than on tests of facial matching or identifying figures hidden in complex drawings (De Renzi et al., 1972). Among patients with Broca's aphasia, color association correlates with sorting performance on Weigl's Sorting test; among patients with Wernicke's aphasia, color association is correlated with performance on Raven's Coloured Progressive Matrices (Basso, Capitani, Luzzatti, Spinnler, & Zanobio, 1985). Color association defects have been found to be more closely related with language comprehension deficits in the visual than in the auditory modality (Varney, 1982a), even in the very rare instance where such defects occur in the absence of aphasia (Varney & Digre, 1983).

One example of a visuoperceptive performance that is not apparently disrupted in the context of receptive aphasia is global stereopsis. Stereopsis is the ability to appreciate that two objects lie at different distances from the observer based on the fact that each eye receives slightly different retinal images of these objects. If one looks through a stereoscope, which presents separate images to the two eyes, and sees two forms with one eye and the same with the other except with a different horizontal separation, then when both eyes are used one form will appear closer than the other. Global stereopsis is similar, except that the two images must be extremely complex, being composed of such things as randomly placed dots. It is impossible to see any forms at all with either eye alone, but when viewed binocularly, part of the background stands out from the rest and the viewer then perceives a form in space. To achieve this visual feat, one must call on certain higher level visuoperceptive abilities (Hamsher, 1978a). Lesions of the right hemisphere can impair one's capacity to achieve global stereopsis, whereas patients with left-hemisphere lesions perform at the level of hospital control patients (Carmon & Bechtoldt, 1967) even in the context of objectively demonstrated receptive language impairment (Hamsher, 1978b).

Aphasics have been reported to perform more poorly than other brain-damaged groups on a task involving the use of maps, both in the visual and tactile modalities (Semmes, Weinstein, Ghent, & Teuber, 1963). In aphasia this type of apparent spatial deficit is not associated with generalized spatial disorientation as it is in right-hemisphere disease (McFie & Zangwill, 1960). Yet, on what must be considered a rather pure measure of "spatial thinking," namely, the Judgment of Line Orientation test (Benton et al., 1983), which merely calls for matching of lines having the same spatial orientation, aphasics perform very nearly at the level of non–brain-damaged control patients, whereas a sizable proportion of patients with right-hemisphere lesions show severe impairment (Benton, Hannay, & Varney, 1975; Benton, Varney, & Hamsher, 1978). These findings agree with previously described observations suggesting that aphasics show little or no impairment on simple and direct measures of visuoperceptive and spatial capacities, whereas on more complex tasks that often involve an amalgam of spatial, perceptual, and conceptual abilities, severe cognitive disability may emerge in some forms of aphasia, especially in the context of language comprehension deficits. Regrettably, as yet, we cannot say why this is so. Perhaps in receptive forms of aphasia, the victim loses the ability to integrate several specific mental processes, although individually these processes are retained. The difficulty levels of the more specific tests do not seem to provide a useful explanation, since, for example, normal

persons describe the spatial orientation task as more difficult than the constructional praxis task, the opposite of the order of difficulty in aphasia.

Conceptual Performances

Conceptual thinking is something of an amorphous concept that has been applied in such various ways that it is difficult to separate it from either general intelligence or some specific verbal ability. In fact, some theorists (e.g., Bay, 1962) would subsume the nonverbal cognitive deficits in aphasia under the rubric of a defect in conceptual or categorical thinking. It is only when we restrict our usage of the term to its basic elements that we can employ it in a definable and tractable fashion. So we shall use CONCEPTIONAL ABILITY to mean the fundamental ability to generate a concept of a class of things and to discriminate those things that belong to the class from those that do not. Tasks calling for the sorting of objects, colors, forms, and so forth, were thought by Kurt Goldstein to be good representatives of conceptual thinking (K. Goldstein, 1948; K. Goldstein & Scheerer, 1941). One often-used task is the Weigl Color—Form Sorting test. The test uses tokens of several colors in the shapes of circles, squares, and triangles. The subject's task is to form a principle and sort the tokens accordingly and then form a second principle and re-sort the tokens to fit it. Thus the task also involves a conceptual shift.

In an early study using the Weigl sorting task, McFie and Piercy (1952) reported that defective performance was associated with left-hemisphere lesions, based on the observation that failure on this task occurred in 52% of 42 patients with unilateral left-sided lesions and in only 6% of 32 patients with right-sided lesions. Within the left-hemisphere cases, these investigators reported no relationship between conceptual failure and aphasia. A reanalysis of their data, however, suggests that this conclusion may be inaccurate if one considers frontal lobe patients separately. In the left–frontal-lobe group, there were 7 defective performances out of 12, and 6 of the patients with defective performances were nonaphasic. Statistically, there was only a trend for aphasia and defective sorting to be dissociated in left–frontal-lobe disease (the Fisher exact probability of chance association was .14). On the other hand, in patients with lesions outside the frontal lobe, aphasia was positively associated with impaired sorting (Fisher exact probability was .047); that is, only 2 of the 15 defective performances were produced by nonaphasic patients (one of whom had a frontoparietal meningioma). Excluding aphasics, there were 8 cases with left-sided lesions and defective sorting out of 17, and 6 of these patients had frontal or frontoparietal lesions. Thus, in the absence of aphasia, it is likely there ex-

ists a positive association between defective performance and frontal lobe involvement (Fisher exact probability was .041).

These analyses highlight an interesting point: Within a single hemisphere, lesions in different loci may result in the same defective cognitive performance but for different reasons. It is now well known that lesions in certain regions of the frontal lobes outside the language zone produce disturbances in sorting behaviors and in the ability to perform conceptual shifts (Milner, 1963). These reanalyses also bring McFie and Piercy's (1952) classic study in line with the results of other investigators who have found, on the whole, that defects on sorting tasks are related to the presence of aphasia and, more specifically, to aphasia involving receptive language impairment (De Renzi, Faglioni, Savoiardo, & Vignolo, 1966).

Language Comprehension Impairment

Nonverbal Communicative Performances

Up to now we have followed the usual custom of thinking of, or at least referring to, language comprehension impairment and its synonyms as if it were a single entity; however, this may no longer be justified. It is fitting to conclude this chapter on intelligence and aphasia with a discussion of language comprehension impairments as it is primarily in association with such defects that nonverbal intellectual performance is most variable and most vulnerable to impairment. The emphasis here is on processes leading to, or occurring with, receptive impairment, with special attention given to the types of errors made by aphasics. We will look for clues that might help resolve the enigma of nonverbal intellectual impairments in aphasia. If the anatomical hypothesis is correct, then this discussion is largely for naught.

In attempting to explain the basis for the occurrence of nonverbal intellectual deficits in receptive aphasia, both the anatomical hypothesis and the psychological hypothesis suffer from certain inconsistencies. It is often stated that if a symptom of cognitive impairment occurs with equal frequency with lesions of either hemisphere, then it bears no essential relationship with language, which in most individuals resides in the domain of the left hemisphere. That the symptom may have a close association with aphasia in the context of left-sided lesions could be attributed to a coincidence of anatomy. This dictum may not be warranted, however, unless the investigator can demonstrate that the cognitive symptom has the same correlates regardless of whether the lesion is on the right or the left. Of course, because of anatomical coincidence, there may be different addi-

tional correlates depending on the side of the lesions, but, at the same time, a core set of related cognitive disabilities should remain invariable. This requirement is derived from the concepts of the structure of intelligence as described earlier, and it is on this point that the anatomical hypothesis is in conflict with the data. The psychological hypothesis stresses the position that behaviors, especially complex cognitive behaviors, have multiple determinants and therefore are subject to distortions for various reasons and from several sources. Exactly what psychological (cognitive, mental) process is the cause of the nonverbal symptoms is the subject of much debate. It may lie in the ability to manipulate symbols or categories. If this were the root cause of both the linguistic and the nonverbal symptoms, however, then it is difficult to explain the substantial proportion of patients with apparently severe receptive deficits and no evidence of a compromise of nonverbal abilities. To postulate the existence of some conceptual center that lies near, or partially overlaps with, the language zone would provide a compromise to the two countervailing hypotheses, but it would also require the assumption of the existence of a set of cognitive abilities that have not previously been shown to have unique properties. For these reasons, the issue of the status of intelligence in aphasia remains unresolved.

Language, as a more or less codified system of symbols used to express or communicate ideas and information, has several forms for expression (e.g., oral speech, written language, gesture). For each mode of expression, a corresponding mode of reception must exist for communication to take place. LANGUAGE is usually used to refer to the use of verbal symbols (words), but we may also think of a nonverbal form of language that allows an individual to communicate with his or her environment.

Spinnler and Vignolo (1966) experimentally demonstrated that defects in the recognition of nonverbal meaningful sounds, such as thunder, the ringing of a door bell, and animal sounds, are associated with aphasia and specifically with impairment in aural comprehension of spoken language. By using a multiple-choice assessment technique, they were also able to show that the types of errors made by aphasics were not random; instead they were predominantly errors involving semantic slippage. For example, if an error were to be made by an aphasic patient in response to a stimulus such as a canary singing, the most likely response choice would be one from the same semantic class, such as a cock crowing, rather than an acoustically similar response, such as a man whistling, or an irrelevant response, such as a train in motion. This impairment is not due to an acquired hearing loss and does not represent a defect in sound discrimination, since receptive aphasics who are impaired on its measure show no defect in the discrimination of meaningless sounds, whereas patients with right-hemisphere lesions may show the opposite pattern (Faglioni, Spinnler, & Vi-

gnolo, 1969). Not all aphasics with receptive deficits show a defect in the recognition of meaningful environmental sounds. Those who do, however, have a high frequency of impairment on the Weigl sorting task and on the RPM test. These findings have been interpreted as demonstrating the presence of a "cognitive–associative" deficit in comprehension-impaired aphasics (Vignolo, 1969).

Subsequently, Varney (1980) replicated these findings and added three new observations. First, sound recognition impairment is specifically associated with aural comprehension as opposed to the comprehension of written language in the visual modality (reading). Second, sound recognition deficits, when present, appear to represent a primary determinant of aural comprehension impairment rather than an expression of it, because defects in aural comprehension of at least equal severity always accompany sound recognition deficits, whereas there are patients with severe impairments in the understanding of oral speech who are not impaired in recognizing the meaning of environmental sounds. Third, Varney and Benton (1979) demonstrated that a defect in the ability to discriminate phonemes represents another, relatively independent, source of aural comprehension deficits. Thus, the aural comprehension of speech may be impaired for either semantic–associative or for perceptual–linguistic reasons. The perceptual comprehension defects are most commonly seen in the acute phase of aphasia and tend to recover (Varney, 1984a). On the other hand, defects in the semantic–associative aspects of aural comprehension suggest a much poorer prognosis (Varney, 1984b).

Deficits in the ability to comprehend the meaning of pantomimes represents another form of nonverbal comprehension impairment in aphasia. Goodglass and Kaplan (1963) observed such impairments in the context of aphasia. Because there was not a close association between the severity of aphasia and the severity of the disturbances in gestures and pantomime, they were led to the conclusion that such deficits must in some sense be independent of aphasia. Subsequently, Varney (1978) demonstrated that, although there was no special correlation between pantomime recognition defects and the severity of the disorder in aural comprehension or verbal expression (naming), failure in pantomime recognition was closely allied with disturbances in reading comprehension (i.e., reading for meaning rather than reading aloud). Impairments in the comprehension of pantomimes always occurred in association with defects in reading comprehension of at least equal severity; however, one could be alexic, even severely so, without showing any impairment in the recognition of pantomimes. Thus, in the visual modality, there is likely to be more than one determinant for an acquired impairment in the comprehension of written language. Varney and Benton (1982) also demonstrated that the major er-

ror type committed by aphasics was to select from among four response alternatives a response that was semantically associated with the pantomimed stimulus rather than selecting an irrelevant item or one that was visually similar but semantically unrelated to the stimulus. Similar results were found by Duffy and Watkins (1984). These findings suggest a parallel organization of verbal and nonverbal language processing in the visual and auditory modalities.

Nonverbal Intelligence and the Receptive Aphasias

Do the defects in sound and pantomime recognition associated with aphasia tell us anything about the fundamental structure of language, or are they merely instances of a host of findings of nonverbal deficits in comprehension-impaired aphasics? It has been argued that the abilities to comprehend gestural communication and to appreciate the significance of environmental sounds represent the selective advantages that fostered the evolutionary development and refinement of the neural structures in humans that serve these functions. They are considered selective advantages because they may be directly related to one's ability to survive in a primitive and hostile environment. Thus, the human brain may have become preadapted for language through the evolutionary development of these pristine nonverbal communicative abilities (Varney & Vilensky, 1980). Arguments of this sort are necessarily discursive, for when empirical proof cannot be obtained, one must rely on analytic reasoning. Because oral speech arises from audition, it would be natural to expect that mechanisms for understanding sounds as meaningful stimuli must precede the development of speech. Also, it would be difficult to maintain, in view of the very recent and rapid development of reading abilities in human evolution, that the capability for understanding written language in the visual modality was acquired on the basis of a selective advantage that augmented the probability of survival.

The final task before us is to relate these recent developments in identifying modality-related subtypes of comprehension impairments to the problem of explaining nonverbal intellectual deficits in aphasia. In our present and limited state of knowledge, it is more reasonable to look for clues rather than solutions to the puzzle. We owe to J. Hughlings Jackson the concept of two classes of behavioral symptoms of brain disease: the negative and the positive. Negative symptoms represent the loss of an ability to produce behaviors that were previously in one's behavioral repertoire. An example is the inability to recall the name of an object. Positive symptoms represent the emergence of new behaviors that were not previously in the behavioral repertoire. An example of a positive symptom is

misnaming, such as the utterance of semantic or phonemic paraphasias in the attempt to name an object (e.g., calling a knife a fork). Whether the errors described here as semantic (conceptual, associative, symbolic) slippage are negative or positive symptoms may be more an academic argument than a real distinction. If characterized as a loss in the ability to discriminate among members of the same semantic class, they could be considered negative symptoms; if characterized as an active error tendency, perhaps as the result of a derailment in thinking, they could be considered positive symptoms. Since aphasic patients who show these errors of semantic slippage may make their response choices with great conviction and an air of success, it may be convenient to think of these errors as positive symptoms resulting from an active process that causes one to err in a particular direction.

If we were to accept the proposition that some comprehension-impaired aphasics suffer from some form of semantic slippage, we might ask if this error tendency could provide an explanation for the occurrence of nonverbal intellectual deficits in aphasia. Applying this model to the problem at hand, we could conceptualize nonverbal intellectual deficits as the consequence of a newly acquired active error tendency that disrupts performance on complex tasks rather than as the result of a loss of the cognitive abilities the tasks were intended to measure. One convenience of this model is that it does not require postulation of the bilateral representation of visuoperceptive, spatial, and constructional abilities or other components of nonverbal intelligence. Unfortunately, such a simple model appears inadequate; that is, when Varney (1982b) tested for a relationship between pantomime recognition defects and performance on the WAIS Block Design subtest, none was found. Thus, the hypothesis that a "general asymbolia" could account for both types of nonverbal defects does not appear tenable. At the same time, there is no evidence that the semantic–associative disturbances with regard to either sound recognition defects or pantomime recognition defects in aphasia are dependent on a specific lesion locus (Varney & Damasio, 1986, 1987). Both types of defects are associated with lesions in a number of cortical and subcortical loci (all of which are associated with aphasia).

The demonstration that there are at least two types of acquired deficits that may result in language comprehension impairment may help to explain the exceptional cases of patients who do not understand oral speech but who show none of the deficiencies in nonverbal intellectual performance that are associated with receptive language impairment. Another dichotomy in subtyping language comprehension impairment is the distinction between single-word or lexical defects and syntactic processing (Vermeulen, 1982); however, it is not clear that this distinction differs from

the perceptual and semantic associative distinctions made earlier. This is an area that needs further exploration.

Probably, we will need new and more complex hypotheses to explain the high degree of variability in the pattern of nonverbal deficits exhibited by groups of aphasic patients who previously were thought to share the same, unitary symptom of comprehension impairment (Benton, 1982, 1985). The development of brain-imaging techniques should help to refine these hypotheses (H. Damasio & Damasio, 1989). Substantial evidence now shows that both size and locus of lesion affects language comprehension and recovery in aphasia (Kertesz, Harlock, & Coates, 1979; Knopman, Selnes, Niccum, & Rubens, 1984; Lomas & Kertesz, 1978; Selnes, Knopman, Niccum, Rubens, & Larson, 1983; Selnes, Niccum, Knopman, & Rubens, 1984; Tramo, Baynes, & Volpe, 1988; Yarnell, Monroe, & Sobel, 1976). Other, more physiological studies may also be necessary. Another imperative may be to specify the syndromes of aphasia to include nonverbal symptomatology that heretofore has been omitted in the assessment and classification of aphasic disorders (Benton, 1982, 1985).

Summary

Intelligence, conceived abstractly, represents one's innate ability to generate ideas and solve problems of varying complexity and to communicate effectively with others and with one's environment. In an effort to approximate this abstract concept, psychologists have developed batteries of cognitive tests from which an IQ score can be obtained. Evidence gathered from various sources shows that intelligence, expressed concretely as an IQ score, meets psychometric criteria and predicts demographic characteristics of individuals that are incorporated in the abstract notion of intelligence.

Neurological disease affecting the cerebrum often results in corresponding impairments in intellectual functioning. In the usual case, disease of the right hemisphere may be reflected in acquired impairments of nonverbal intelligence, whereas disease of the left hemisphere may result in verbal intellectual deficits. In a significant proportion of aphasic patients having receptive language defects, however, nonverbal intellectual performance appears to be compromised.

The hypotheses offered to account for these incongruous findings fall into two general classes. One general hypothesis is that some portion of the cerebral cortex in the left hemisphere, which lies near or overlaps with the language zone, subserves the same general mental functions as mediated by the right hemisphere. If lesions resulting in aphasia happen to invade

this region as well, then nonverbal intellectual deficits will result. This hypothesis denies the existence of a functional relation between nonverbal intelligence and aphasia. The other general hypothesis suggests the opposite and links disturbances in language with disturbances in thought processes. So far, the evidence on this issue is mixed, and if sampled selectively, it can be used to support or refute either hypothesis.

It is argued that linguistic aspects of language were derived from pristine nonverbal communicative abilities. Recent investigations of the capacity of aphasics to comprehend environmental sounds and pantomimes has disclosed the presence of error tendencies, described here as semantic slippage, in both the auditory and visual modalities. In aphasia, these error tendencies may disrupt response selections in the performance of nonverbal intellectual tasks, whereas in right-hemisphere disease, acquired deficits in stimulus processing may be the root causes of nonverbal intellectual impairment. Neural mechanisms to account for these error tendencies have remained elusive.

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11

Artistry and Aphasia

ELLEN WINNER and CATYA VON KAROLYI

Language is our central mode of communication. Although the symbol systems of the arts are almost as universal and as well developed among humans as is language, much less is known about the representation of artistic skills in the brain. Moreover, little is known about the relationship of language and artistic modes of communication. In what follows, we review what is known about the relationship between aphasia and abilities in three major art forms: music, the visual arts, and literature.

Music

Music bears many parallels to language. Both involve a written notation and are based on combinatorial rules. In addition, both involve more than one role: speaker, writer, reader, or listener (language), and performer, composer, critic, or listener (music). Each role can be selectively impaired. For instance, Botez and Wertheim (1959) studied an accordion player who, after removal of a tumor in the second right frontal convolution, suffered several severe disturbances in music. Although able to sing individual pitches, he could not combine them into a song. His repetition of rhythmic and melodic material was poor, and he could no longer play the accordion. Yet he had perfectly preserved perceptual and receptive capacities: he could recognize pieces he knew, he could spot errors, and he was able to criticize his own performance. Suggesting an analogy to expressive aphasia, Botez and Wertheim described this patient as having expressive amusia. A contrasting case was reported by Wertheim and Botez (1961), in

which a violinist became aphasic after left-hemisphere injury. He also lost his absolute pitch, had difficulty recognizing changes in tempo, was unable to analyze chord structure, and could not name familiar pieces. However, he could pick out pieces on the violin with his nonparetic hand. Continuing the analogy to the aphasias, here was a patient whose receptive problems were more evident than his expressive ones.

Organization of Musical Skills in the Brain

A pioneering study by Milner (1962) documented a right hemisphere superiority for certain components of music. Subtests of the Seashore test battery were administered both pre- and postoperatively to epileptic patients who underwent removal of one of their temporal lobes. After removal of the right temporal lobe, there was a significant drop in scores on subtests measuring sensitivity to timbre and intensity, and in tonal memory. Patients with left temporal lobe removal were generally comparable in sensitivity to their preoperative levels.

Subsequent dichotic listening studies have confirmed the greater importance of the right hemisphere in the processing of melody, chords, tone contours, and complex tones (Kimura, 1964, 1967), and for individual pitches, intervals, and directionality of pitches (Peretz & Morais, 1987). Evidence from brain damage has confirmed and extended these findings. For example, right-hemisphere damage impairs the discrimination of complex tones (Sidtis & Volpe, 1988). Anterior right temporal lobectomy impairs timbre discrimination (Samson & Zatorre, 1994). And although earlier studies reported by Milner, Kimura, and Taylor (1965) and Zatorre (1985) found that both temporal lobes play a role in melody recognition, a more recent study showed that only the right temporal lobe is involved (Samson & Zatorre, 1991; see also Samson & Zatorre, 1988). In the earlier studies, target versus foil melodies were very different, whereas in Samson and Zatorre's (1991) study, target and foil melodies differed only in pitch intervals. Thus, the right temporal lobe may play a critical role in processing pitch interval information. Another study demonstrated the role of the right hemisphere in recognizing familiar melodies: Gardner and Denes (1973) found that right-hemisphere, brain-damaged (RBD) patients were impaired in their ability to recognize the situation in which particular pieces (such as "Hail to the Chief") are normally heard. These patients only performed well when the correct answer was based on the lyrics (not given) of a song: for instance, they could match the melody of "Row, Row, Row Your Boat" to a picture of a boat.

The right hemisphere is also involved in making musical judgments. When asked to rate how well a final note completed a simple melody, RBD

patients made less use of tonal goodness than did left-hemisphere, brain-damaged (LBD) patients (Brownell, Postlethwaite, Seibold, & Gardner, 1982). Patients with right anterior damage were unable to detect errors in familiar pieces (Shapiro, Grossman, & Gardner, 1981), demonstrating an impaired internal representation of familiar melodies. Further evidence for such a deficit comes from a case study in which an amateur RBD musician proved unable to tell whether the first note of a familiar piece was higher or lower than the second (Judd, Gardner, & Geschwind, 1983). Despite this deficit, his ability to answer challenging theoretical questions about music was unaffected.

Positron-emission tomography (PET) studies have continued to show the primary role of the right hemisphere in processing music. For example, listening to melodies results in cerebral blood flow to the right superior temporal and right occipital cortices, and judging pitch results in right frontal lobe activation and decreases in blood flow to the left primary auditory cortex (Zatorre, Evans, & Meyer, 1994). Thus, specialized neural systems in the right superior temporal cortex play a role in the perception of melodies, and pitch comparisons are performed by the right prefrontal cortex. These results were not simply a function of auditory processing: control tasks showed that processing of nonmusical noise did not produce these same effects.

Finally, evidence from split-brain patients has also implicated the right hemisphere in the perception of harmony and in the discrimination of timbre. Tramo and Bharucha (1991) presented chords that were either in tune, or out of tune because one pitch has been altered. Only the right hemispheres of the two split-brain patients tested showed evidence of a chord priming effect, demonstrating that only the right hemisphere was making judgments of chord harmony. Tramo and Gazzaniga (1989) showed that the right hemisphere of split-brain patients was superior to the left on the Timbre Test of the Seashore Measures of Musical Talents (for reviews of the role of the right hemisphere in musical processing, see Hodges, 1996; Peretz, 1985; Zatorre, 1984).

The right hemisphere also is dominant for musical performance. For instance, under right hemispheric anesthetization, melodies were distorted (Bogen & Gordon, 1971) and patients proved unable to vary pitch and tone when singing but retained their rhythmic ability (Borchgrevink, 1982). After right intracarotid injection, patients sang monotonously, without tonal control, although rhythm was far less affected; in contrast, speech (including pitch and stress) remained unimpaired (Gordon & Bogen, 1974). Measurements of right- versus left-sided lip opening when singing versus speaking also prove revealing. When speaking, individuals normally show greater lip opening on the right, demonstrating left hemisphere activation

during speech (Graves & Landis, 1985, 1990). In contrast, when singing without words, individuals show no asymmetry of lip opening, indicating that the right hemisphere is now playing a role (Cadalbert, Landis, Regard, & Graves, 1994). When aphasics sing (again without words), they show greater left-sided lip opening, indicating right-hemisphere dominance for singing melodies (Graves & Landis, 1985, 1990). This right-hemisphere advantage for singing has been used in speech therapy for aphasics. Yamadori, Osumi, Masuhara, and Okubo (1977) found that Broca's aphasics with intact right hemispheres can improve speech when singing, probably because singing recruits the right hemisphere. This finding has been successfully used in Melodic Intonation Therapy (Albert, Sparks, & Helm, 1973; Helm-Estabrooks, 1983).

The one component of music for which the left hemisphere of the ordinary individual has been implicated is rhythm, a time-dependent sequencing task (Borchgrevink, 1982; Brust, 1980; Gordon, 1978; Gordon & Bogen, 1974; Mavlov, 1980; Natale, 1977; Robinson & Solomon, 1974; Zatorre, 1984; but see Craig, 1980, Gregory, Harriman, & Roberts, 1972, and Shapiro et al., 1981, who showed rhythm processing in the right hemisphere, and Peretz, 1990, and Prior and Troup, 1988, who showed no lateralization for rhythm).

With formal musical training, the left hemisphere takes on more of a role in music processing. Bever and Chiarello (1974) found that individuals without musical training recognized melodies better when they were presented to the left ear (indicating a right-hemisphere advantage); individuals with musical training showed less of a right ear advantage, indicating the involvement of both hemispheres. Musicians also out-performed nonmusicians on an excerpt recognition task, which requires breaking down melodies into their constituent parts. Bever and Chiarello concluded that individuals with formal training in music approach music more analytically, and their left hemispheres become more involved. However, not just any kind of formal training may suffice: a later study showed that a right-ear advantage in musicians was found only among those who could transcribe music, but not among those who could simply read and play music (Johnson et al., 1977).

Perhaps the most dramatic evidence of left-hemisphere dominance for music in musicians is the case of Martha Curtis, a violinist who developed severe epilepsy at age $3\frac{1}{2}$. As shown on the television program, "Sixty Minutes" ("Martha Curtis' Story," 1996), Curtis has had four brain surgeries to control the epilepsy, and has had large portions of her right hemisphere removed. However, her music has remained entirely unaffected and she continues to flourish as a professional violinist.

Increased involvement of the left hemisphere (and use of an analytic strategy) may occur not only as a function of training, but also as a function of the task. For example, when nontrained individuals were asked to detect pitch changes in familiar melodies (an analytic task), a right-ear advantage was found (Gates & Bradshaw, 1977). Similarly, a right-ear advantage was found when nontrained individuals were asked to discriminate melodies differing only by a single note (Gaede, Parsons, & Bertera, 1978). Peretz and Morais (1980, 1987) showed that although nontrained individuals discriminate melodies by attending to their overall contours (thus primarily calling on the right hemisphere), when the task forces them to rely on more local cues, the left hemisphere takes over.

The role of the right hemisphere in global processing and of the left hemisphere in local, analytic processing of music have been confirmed in studies of brain-damaged patients. Peretz (1990) asked patients with unilateral brain damage to determine whether pairs of melodies were the same or different in cases in which the contours were preserved but pitches violated (an analytic task), in cases in which contours were violated (a global task), and in cases in which the melodies were transposed, resulting in the same contour but different pitches (a global task). The RBD patients were worse than the LBD patients at the global task of contour violation. In contrast, LBD and RBD patients were equally impaired on the analytic task of discriminating melodies differing only in pitch or intervals. Thus, RBD patients were impaired in BOTH global and analytic processing, whereas LBD patients were impaired ONLY in analytic processing. Peretz concluded that the processing of local intervals is dependent on intact contour processing. Only after the right hemisphere processes the contour can the left hemisphere process the local information. Thus, music processing is hierarchical: local processing (a left-hemisphere task) cannot take place until a global framework is established (a right-hemisphere task).

Further evidence for the two hemisphere's contrasting roles in music processing comes from a study investigating the ability to match musical fragments with geometric patterns (Gardner, Silverman, Denes, Semenza, & Rosenstiel, 1977). Patients were asked, for example, to match ascending passages with lines pointing either up or down, or continuous tones with either intact or fragmented circles. The LBD patients were better at matching passages to patterns showing the temporal course of the piece (e.g., regularity, irregularity) than they were at matching passages to patterns capturing gestalt aspects of the piece (e.g., continuity, discontinuity). In contrast, those with left anterior damage were better at matching passages to pictures capturing global properties of the piece than they were at matching to pictures showing temporal aspects.

Amusia and Aphasia

As the left hemisphere plays a role in music processing (in individuals with formal training, when individuals adopt an analytic processing style, and perhaps when rhythm is being processed), left-hemisphere damage may impair some aspects of music. Thus, in some cases, aphasia will be accompanied by amusia.

The relationship between aphasia and amusia is not straightforward. In a review of 89 cases, Marin (1982) found 33 cases of amusia together with aphasia, and 9 cases of verbal and musical alexia. The composer, Maurice Ravel, had mild Wernicke's aphasia, along with alexia and agraphia. He lost his ability to compose, to sight read, and to play either from a score or by heart, yet he retained the ability to appreciate music (Alajouanine, 1948; Baeck, 1996; Sergent, 1993). Ravel commented poignantly, "I will never write my *Jeanne d'Arc;* this opera is here, in my head, I hear it, but I will never write it. It's over, I can no longer write my music" (Nichols, 1988, cited in Sergent, 1993). Ravel's impairment was selective. He could not translate musical representation into another form of representation. That is, he could not translate musical notation into motor form by playing, and he could not translate his auditory imagery of a piece of music into a visual form (by notating it) or into a motor form (by playing by heart). Yet he could use each of these modalities separately: he could play scales (using motor representation) and he could detect errors when listening (using auditory representation; for other examples of amusia accompanied by aphasia, see Botez & Wertheim, 1959, and Hofman, Klein, & Arlazoroff, 1993).

Subtle language disturbances not detected by standard aphasia batteries may also bring with them disturbances of music perception. Patel (1996) studied two amusic patients with bilateral damage with no significant aphasias. The patients were tested on their ability to make same–difference judgments about sentence pairs and melody pairs. When the sentence pairs differed, they differed only in intonation or rhythm (e.g., "Take the TRAIN to Bruge, Anne" vs. "Take the train to BRUGE, Anne"). The melody pairs had the same pitch and temporal patterns as the sentence pairs. One patient performed well on both tasks; her deficit involved long-term memory for melodies and the perception of tonality. But the other patient, whose deficits were tied to damage to the left primary auditory cortex and to the right prefrontal cortex, performed poorly on both tasks. Thus the perception of prosody in language and in music may depend on shared neural resources.

In Marin's (1982) review of cases, there were also 12 instances of aphasia without amusia. The Russian composer, V. I. Shebalin, had Wernicke's aphasia with no loss of musical ability (Luria, Tsvetkova, & Futer, 1965).

Another example is that of the French organist and composer, Jean Langlais, who was aphasic, alexic, and agraphic, but his music was unaffected (Signoret, Van Eeckhout, Poncet, & Castaigne, et al., 1987). In addition, it is quite possible for an individual to lose the ability to read linguistic text but still be able to read musical notation (Soukes & Baruk, 1930; for other examples, see Assal, 1973; Basso & Capitani, 1985; Bouillaud, 1865; Clynes, 1982; Critchley & Henson, 1977; Polk & Kertesz, 1993; Wallin, 1991).

Finally, Marin (1982) uncovered 19 cases of amusia without aphasia. Disturbances of music without an accompanying aphasia typically result from right-hemisphere brain damage, whereas amusia accompanied by aphasia results from left-hemisphere damage (e.g., see McFarland & Fortin, 1982; Polk & Kertesz, 1993). Amusia can be highly selective, leaving the perception of environmental as well as speech sounds intact. This was demonstrated in the case of a woman with bilateral lesions of the rostral auditory association cortex (Peretz et al., 1994). She suffered no aphasia but had impaired music perception. She complained that singers sounded like they were talking, not singing, and that she was not able to recognize familiar pieces, nor was she able to name musical instruments from their sounds. However, she had no difficulty recognizing environmental sounds or emotional tone conveyed by speech prosody. Another patient described in the same study suffered bilateral damage that left a Wernicke's aphasia from which the patient later recovered. After recovery, this patient had no difficulty in naming environmental sounds, but could not recognize familiar music and had lost pleasure in listening to music. Like the first patient, he also was impaired in the ability to recognize emotional tone conveyed by speech prosody, but could use linguistic prosody to determine the syntax of a sentence (e.g., interrogative vs. declarative). Both of these patients had difficulty recognizing pitch variations, but had no trouble recognizing rhythmic variations, showing spared processing of rhythm in the face of impaired processing of melody, despite the fact that these processes are integrated in the normal brain (Peretz & Kolinsky, 1993).

Both of these cases show striking dissociations between music and speech and indicate a distinction between kinds of auditory agnosia. Clearly, then, there is no general mechanism whose disruption causes all types of auditory agnosia. These cases also show that music is processed differently not only from speech but also from environmental sounds. It would be of interest to determine whether there are cases in which music is preserved in the face of recognition of both impaired speech and environmental sounds.

With the possible exception of Ravel, musicians with left-hemisphere damage and aphasia have retained the capacity and desire to engage in cre-

ative musical activity. In contrast, right-hemisphere damage in a musician can alter the person's relationship to music, and render the person unable to produce or perform music except in a very limited and uninspired manner. These double dissociations, in which amusia can exist without aphasia, and aphasia without amusia, show that music and language (and the perception of environmental sounds) are mediated by distinct neural areas (Benton, 1977) and are functionally autonomous. How, then, can we explain the not infrequent co-occurrence of aphasia and amusia, as in the famous case of Ravel? The answer to this must be either that music and language share some neural substrates, or that they share contiguous neural substrates.

Positron-emission tomography studies shed light on this question. Sergent, Zuck, Terriah, and MacDonald (1992) PET scanned 10 professional pianists while they performed three different activities: reading music notation, playing from notation, and listening to the music of the score without looking at it or playing it. Both hemispheres were involved. Playing scales with the right hand activated the left motor cortex (area 4), the right cerebellum corresponding to the motor representation of the right hand, and the left premotor cortex (area 6). Listening to scales activated the secondary auditory cortex (area 42) in both hemispheres, as well as the superior temporal gyrus of the left hemisphere (area 22). Listening to a musical piece activated the same areas and also the right superior temporal gyrus, indicating bilateral involvement of the temporal cortex. Reading scores resulted in bilateral activation of the extrastriate visual areas (since this task requires processing of visual information). However, the areas in the left hemisphere normally engaged in reading words were not activated by reading music. Instead, the left occipito–parietal junction was activated. This can be better understood by recognizing that whereas word reading requires a feature analysis (of each letter), note reading is carried out by analysis of the spatial location of notes, that is, their relative heights on the staff (Sloboda, 1985).

When reading the score and listening simultaneously, the superior and posterior part of the supra marginal gyrus (area 40) in the inferior parietal lobule of the hemisphere is activated. This area may be involved in mapping the notation to the sound. In the case of reading words, the INFERIOR parietal lobule carries out this mapping function, and when this area is destroyed, alexia and agraphia result. For music, it appears to be the SUPERIOR part of the supramarginal gyrus that is involved, and when this area is impaired, reading of words is spared (Roeltgen & Heilman, 1984). Thus, the mapping of music notation to its auditory representation is performed by areas distinct from, but adjacent to, structures underlying the mapping of visually presented words to their auditory representations (see Judd et

al., 1983, for further evidence of the independence of reading of words and musical notes).

When reading, playing, and listening were performed together as one activity, the superior parietal lobule (area 7) of both hemispheres was activated. This may be due to the spatial nature of music notation. This area of the parietal cortex also mediates visually guided skilled action (e.g., required for correct finger positioning in piano playing). These conjoint activities also activated the left premotor cortex (area 6) and the left inferior frontal gyrus (area 44), both of which are just above Broca's area.

Sergent et al. (1992) demonstrated that sight-reading and piano playing depend on a cerebral network widely distributed over the four cortical lobes and the cerebellum. The areas activated are independent of the areas used for language: hence the many cases of aphasia in musicians with no amusia. However, Sergent et al. (1992) argue that the proximity of the areas underlying language and music makes it likely for both to be impaired when brain damage is extensive. Musical activities activated cortical areas distinct from but adjacent to those underlying similar linguistic functions. This may explain why brain damage in musicians may or may not affect both verbal and musical functions: whether both functions are impaired may depend on the size and location of the area of damage (see also Patel, 1996, for evidence that music and language share neural substrates).

The pattern of findings reviewed here, although complicated and conflicting, allow the following generalizations. Both hemispheres are clearly involved in music, but each hemisphere makes a different kind of contribution. Although the right hemisphere plays the most important role, it is relatively rare for an individual with severe aphasia to not also suffer some loss in musical capacity. The right hemisphere seems particularly important in four areas: processing the components of music (e.g., recognizing pitch and timbre), internally representing melodies so that errors can be detected, producing music, and reacting emotionally with pleasure and inspiration to music. The left hemisphere seems particularly important in those aspects of music most closely allied to language—reading musical notes and naming notes and pieces. But even when the ability to read music and to recognize and name pieces is severely impaired, underlying musical intelligence may be spared.

Drawing

We know a great deal about the effects of brain damage on drawing because drawing is part of most neuropsychological assessments (Lezak, 1983). In addition, quite a few artists who have suffered brain damage have

been studied (Alajouanine, 1948; Ball, 1983; Gardner, 1975a, 1975b; Jung, 1974; Schweiger, 1988; Wapner, Judd, & Gardner, 1978; Zaimov, Kitev, & Kolev, 1969).

Organization of Drawing Skills in the Brain

There is far more dissociation between graphic and linguistic ability than there is between musical and linguistic ability. Most research demonstrates that the ability to draw can remain uncompromised despite significant aphasia, although this is not always the case. Research also consistently demonstrates that drawing can be compromised with no correlative language impairment.

Despite the considerable independence of drawing and language, both the left and the right hemisphere play a role in drawing ability. Drawings by LBD patients have characteristics highly distinguishable from those by RBD patients (Gainotti & Tiacci, 1970; Gasparrini, Shealy, & Walters, 1980; Hécaen & Assal, 1970; Hécaen, de Ajuriaguerra, & Massonet, 1951; Kimura & Faust, 1985; Kirk & Kertesz, 1989; McFee, Piercy, & Zangwill, 1950; Piercy, Hécaen, & de Ajuriaguerra, 1960; Swindell, Holland, Fromm, & Greenhouse, 1988; Warrington, James, & Kinsbourne, 1966). Drawings by LBD patients are simplified and lacking in detail, whereas those by RBD patients are elaborately detailed (Caplan, 1988; Kirk & Kertesz, 1989); LBD drawings are spatially coherent, showing the overall framework of what is depicted, and RBD drawings are piecemeal and fail to capture the overall framework, spatial relations, and proportions (Caplan, 1988). These are the two most commonly reported differences between drawings by LBD and RBD patients.

There are other fairly consistent differences as well. Drawings by LBD patients, particularly those with anterior lesions, are smaller and less easily recognizable than those by patients with right damage (Kimura & Faust, 1985). The LBD patients draw slowly and with difficulty (note that right-handers must draw with their nondominant hand), whereas the RBD patients draw energetically and fluently. LBD patients have greater deficits in spontaneous drawing than do RBD patients (Kimura & Faust, 1985). However, even though aphasic patients have difficulty drawing objects from memory (objects are often so devoid of critical details as to be unrecognizable), their ability to copy from a model may be only mildly impaired (see Goldenberg, Dettmers, Grothe, & Spatt, 1944, in which aphasics scored only slightly below control subjects on the Rey-Osterrieth copy task). Similarly, aphasics often select the wrong colors when drawing (Cohen & Kelter, 1979), yet are able to choose the correct hue in a match-to-sample test (De Renzi & Spinnler, 1967). Although LBD patients improve their draw-

ing when working from a model, RHD patients do not. LBD patients rarely show right-sided neglect of the picture, whereas RBD patients (with right parietal lesions) often have severe left neglect and leave drawings incomplete or completely blank on the left side (Caplan, 1988). LBD patients recover their drawing abilities more quickly and more completely than do those with RBD (Swindell et al., 1988).

Thus the left hemisphere provides the details of a graphic representation, and the right hemisphere captures the general configuration and organization of a drawing (Kaplan, 1982). This conclusion is supported by perceptual studies showing that the left hemisphere attends to features, and the right hemisphere attends to the overall spatial configuration of a drawing. For example, D. W. Zaidel (1990) showed two split-brain patients pictures of faces with correct features in incorrect places (e.g., a nose where an eye should be). The patients were asked to point to facial features. When the left hemisphere saw the face, patients pointed accurately to the misplaced features; when the right hemisphere saw the face, they pointed to the locations where the features SHOULD have been. Similarly, Gardner (1975a) showed that RBD patients grouped paintings by subject matter (i.e., they attended to the features), whereas LBD patients grouped paintings by overall style.

Site of lesion within each hemisphere also predicts they type of drawing deficit. Caplan (1988) reported that patients with left inferior parietal lesions draw oversimplified figures with few details, but are able to copy drawings with little difficulty. These patients often have naming problems and/or alexia with agraphia. Left superior parietal lesions leave some patients with deficits in drawing people and body parts, concomitant with their difficulties in naming and describing these. Frontal lobe lesions in either hemisphere lead to carelessness and poor planning in executing drawings: such patients often leave drawings unfinished (Caplan, 1988).

With respect to the right hemisphere, Grossman (1988) reported that damage to the right central (primarily parietal) area is associated with particular impairments in depicting shape. In contrast, damage to the right posterior (primarily temporoparietal) area is associated with difficulty in depicting color. Damage to the right anterior area is associated with impairments in depicting shape, color, and relative size. In addition, subcortical lesions have been shown to play a role in drawing difficulties: Kirk and Kertesz (1993) found no differences in drawing impairment when comparing patients with right cortical versus subcortical lesions, and when comparing patients with left cortical versus subcortical lesions. Subcortical lesions may interrupt pathways between cortical areas needed for drawing.

Drawing Impairment and Aphasia

Most researchers argue that the drawing disability associated with right-hemisphere damage is a consequence of impaired visual–spatial skills (e.g., Gainotti & Tiacci, 1970; Warrington et al., 1966; but see Arena & Gainotti, 1978, who argue that drawing impairment is due to a perceptual disorder independent of side of lesion.) For instance, Kirk and Kertesz (1989) administered the Ravens Coloured Progressive Matrices test (a test of visual–spatial cognition) to both LBD and RBD patients. They found a correlation between level of drawing ability and score on the Ravens test for BOTH kinds of patients, suggesting some visual–spatial deficit underlying the drawing impairment caused by damage to either side of the brain. However, the association was stronger in the case of patients with right-sided damage, showing that a visual–spatial deficit contributes more to the drawing impairment associated with right-hemisphere damage. RBD patients clearly have an understanding of the objects they draw, given the richness of detail that they supply. Their difficulty lies in synthesis, in putting together the parts to make up the whole.

Drawing disability associated with LBD has been argued to result from a deficit in executive function, causing difficulty in planning and performing complex motor sequences (Gainotti & Tiacci, 1970; Warrington, James, & Kinsbourne, 1966). For instance, Kimura and Faust (1985) found that drawings by LBD patients were particularly impaired and difficult to recognize in the case of apraxic patients, who are known to have difficulty in executing planned sequences of fine motor movements. Drawing disability associated with left-sided damage may also be related to semantic–lexical deficits that accompany some aphasias. Gainotti, Silveri, Villa, and Caltagirone (1983) found a relationship between aphasics' ability to draw from memory and degree of impairment at the semantic–lexical level. Kirk and Kertesz (1989) showed that two subtests of the Western Aphasia Battery were significantly related to drawing impairment in LBD patients: verbal comprehension and aphasia quotient. They concluded from this that although dominant hand paresis contributes to left brain-damage drawing difficulty, linguistic deficits also play a role. In particular, deficits in verbal comprehension and associated deficits in concept formation may account for the oversimplified nature of drawings of LBD patients. This account is consistent with the finding that aphasics who cannot draw or color from memory can draw from a model and can select appropriate colors on a matching task. What is impaired, therefore, is not visual–spatial understanding but an understanding of the object they are drawing (Gainotti et al., 1983).

To determine whether aphasics' difficulty in drawing and coloring from memory is due to difficulty in planning and executive functions, or to a

loss of knowledge about how things look, Goldenberg (1995) tested aphasics' ability to distinguish correct from incorrect graphic representations of familiar objects. These aphasics had difficulty detecting errors in the drawings, showing that aphasics' drawing impairment is not due to a problem with planning and executing a drawing but rather to a loss of knowledge about how objects look. Goldenberg (1995) argues that left-hemisphere damage can lead to an impaired semantic understanding, resulting in difficulties producing and comprehending names of objects, and reconstructing and recognizing how objects look.

The ability to generate images is a function of the posterior left hemisphere (Farah, 1984, 1989; Farah, Gazzaniga, Holtzman, & Kosslyn, 1985; Kosslyn, Farah, Holtzman, & Gazzaniga, 1985). Hence, aphasics' drawing impairments may be related to problems in image generation consequent to left-hemisphere damage. Many computations are involved in imaging, however, and both hemispheres make a contribution (Kosslyn et al., 1985). The left hemisphere plays a dominant role when the locations of image parts are specified precisely and categorically, since the left hemisphere encodes information categorically and linguistically; in contrast, a right hemisphere superiority occurs when the parts are less precisely specified (Kosslyn et al., 1985).

Use of Drawing in Aphasia Therapy

Even though comprehension deficits in aphasia may contribute to oversimplified drawing, there have been many attempts to recruit drawing as a vehicle of communication for aphasics (Hatfield & Zangwill, 1974; Lyon, 1995; Van Eeckhout, 1993; Ward-Lonergan & Nicholas, 1995; for a review, see Lyon & Helm-Estabrooks, 1987). Pictures have also been used as means of communication in the rehabilitation of aphasic patients. Even severely aphasic patients are able to communicate through VIC, a visual communication system consisting of simple (predrawn) visual symbols (Gardner, Zurif, Berry, & Baker, 1976; Steele, Weinrich, Wertz, Kleczewska, & Carlson, 1989; Weinrich, Steele, Carlson, & Kleczewska, 1989). In some cases, aphasics have been able to create their own pictures in order to communicate what they cannot say. When drawing ability is less impaired than language, drawing can facilitate communication (Cubelli, 1995). Visual Action Therapy (VAT) teaches global aphasics to draw to communicate (Helm-Estabrooks, Fitzpatrick, & Barrei, 1982). Other forms of treatment based on drawing include Promoting Aphasics' Communicative Effectiveness (PACE), and Back to the Drawing Board (BDB). These training methods have been shown to improve drawing ability in LBD patients (Lyon and Sims, 1989; Ward-Lonergan & Nicholas, 1995).

Although training in drawing can clearly provide the aphasic patient with a means of communicating, there is no evidence that such training can improve skills in aphasia. Moreover, drawing is not always the royal road to communication in aphasia. Sometimes drawing is too impaired, and thus cannot serve as a direct way of showing thought (Goldenberg, 1995).

Effects of Brain Damage on the Art of Visual Artists

LEFT-HEMISPHERE DAMAGE

The left hemisphere of the painter may play a less crucial graphic role than it does in the ordinary person. A major French painter studied by Alajouanine (1948) was rendered severely aphasic by a left-hemisphere stroke, but his art was unaffected. The painter poignantly described this split between his artistic self and his other selves:

> There are in me two men, the one who paints, who is normal while he is painting, and the other one who is lost in the mist, who does not stick to life. . . . I am saying very poorly what I mean. . . . There are inside me the one who grasps reality, life; there is the other one who is lost as regards abstract thinking. . . . There are two men, the one who is grasped by reality to paint, the other one, the fool who cannot manage words any more. (p. 238)

For other cases in which artistry remained unimpaired in the face of severe aphasia, see Gardner, Winner, and Rehak (1991), Van Eeckhout (1993), and Zaimov et al. (1969).

As is evident in Figures 11-1 through 11-4, the work of a patient referred to as GM showed some change in style subsequent to a left-sided stroke (Gardner et al., 1991). Before the stroke, his work (Figure 11-1) shows complex use of form and line, and attention to depth and the interplay of shapes. His painting subsequent to the stroke (Figure 11-2) continues to be concerned with these graphic elements, but on a somewhat less sophisticated level: the forms seem amorphous and there is little sense of depth to the painting as a whole. His drawings show a similar but less dramatic change. GM's earlier drawing (Figure 11-3), created just a few months before the stroke, relies on sharp contrast and clearly drawn forms. Although his poststroke drawing (Figure 11-4) also shows sharp contrast, the forms seem less masterfully drawn, and there seems to be less control of depth and layering of shapes. Note, however, that GM retained a good sense of overall composition. GM did not seem to show the typical finding of a loss of graphic detail consequent to left-hemisphere damage, although it is difficult to make judgments about loss of detail in the case of nonrepresentational art.

GMs work did not diminish in expressiveness or power. In fact, one

FIGURE 11.1. *This example of GM's prestroke painting shows complexity of form and line, attention to depth, and interplay of shapes.*

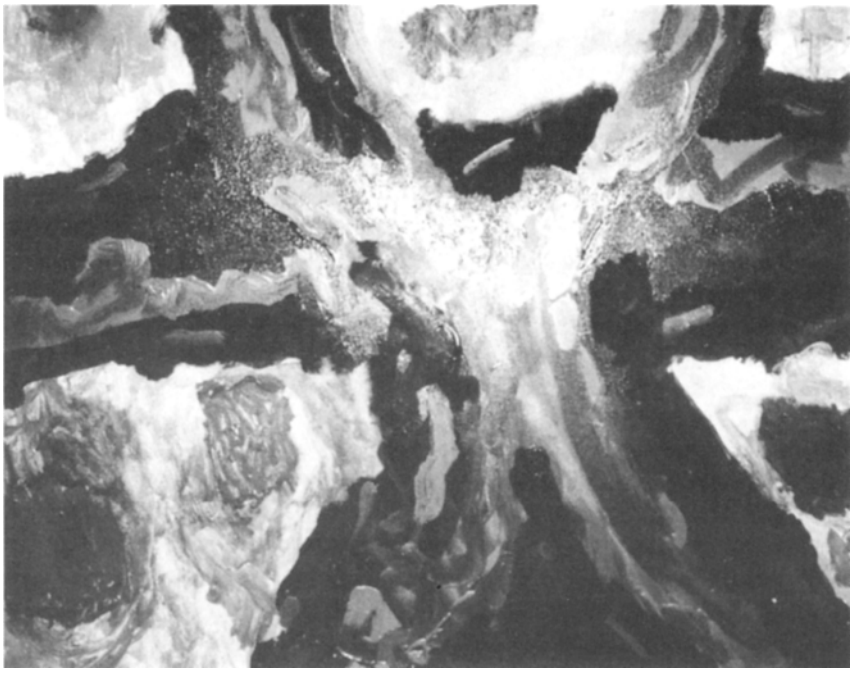

FIGURE 11.2. *This example of GM's poststroke painting has amorphous forms and little sense of depth.*

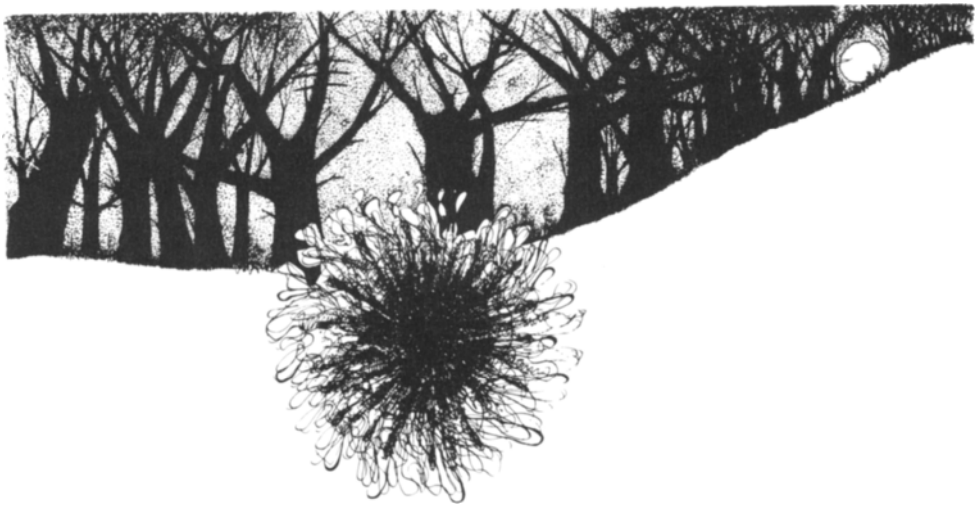

FIGURE 11.3. *This example of GM's prestroke drawing shows sharp contrast and distinct forms.*

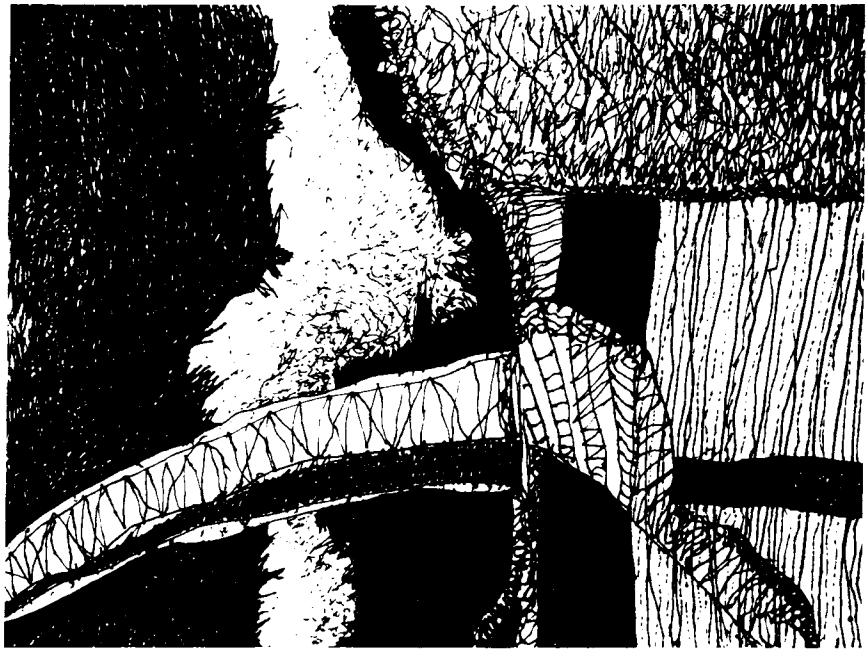

FIGURE 11.4. *This example of GM's poststroke drawing exhibits less control of depth and layering of shapes.*

might argue that the poststroke work is more interesting and more emotionally evocative then the earlier work. (See also Hskus, 1995, for a report of an aphasic stained-glass maker who learned to draw with his left hand and whose drawings became freer, more impressionistic, and less detailed.) In addition, GM remained critical of his own work and that of others, communicating his opinions through gesture and facial expression.

Left-hemisphere damage does sometimes affect artistic ability. (The patient, IK, had a left posterior parietal tumor that left him transiently aphasic (Marsh & Philwin, 1987). His painting showed right-sided neglect and constructional apraxia. A painting produced during IK's illness had recognizable human figures only in the lower left hemifield, with progressive simplification from the left to the right side of the painting. These findings indicate that such a tumor can damage visual–spatial perception and constructional ability. Such radical changes have not been reported in other cases of left-hemisphere disease, and may result as much from the nature of the tumor and its possible effects as from its focal location. IK's paintings showed little change in style or emotional quality; rather the changes seemed to result only from his perceptual difficulties.

These cases reveal that artists' graphic skills can function independently of language and most other left-hemisphere skills. This finding is less true of nonartists, in whom LBD results in impoverishment of detail. Whether the visual analysis of detail is preserved in the LBD artist because this skill has been overlearned, or because this skill is more widely represented in the brain, is not known. In addition, we do not have a group of nonartists matched in terms of lesion site with whom to compare the artists. Thus no strong conclusions can be drawn about the difference between nonartists and artists concerning the role of the left hemisphere in drawing ability. Drawing ability in an artist may also be impaired as a consequence of visual agnosia. Wapner et al. (1978) report a visually agnostic artist who could draw parts of objects realistically but was unable to draw whole objects because he was unable to see them in their entirety.

RIGHT-HEMISPHERE DAMAGE

A few cases have been reported of painters who have suffered right-hemisphere strokes. Schnider, Regard, Benson, and Landis (1993) described an artist with a right parieto-temporo-occipital stroke, resulting in hemispatial neglect, hallucinations, and bipolar disorder (all well-known consequences of right middle cerebral artery infarction). His poststroke paintings reflected all of these symptoms.

Lovis Corinth and Anton Raderscheidt, both German expressionists, resumed painting after partial recovery from right-hemisphere strokes (Jung, 1974). Initially, their paintings included left-sided neglect, irregular contours, misplaced detail, and fuzziness of depiction. With recovery, neglect was reduced, but the paintings continued to appear fundamentally different from their prestroke style. The poststroke style was less inhibited, more emotional, primitive, sensual, and bizarre, featuring rough lines and grotesque effects (Gardner, 1975b). Art historians and critics at the time assumed these changes were a reaction to severe illness. However, the style changes may have reflected altered emotional appropriateness consequent to right-hemisphere disease. A similar change in style following right-hemisphere damage occurred in the case of the American artist Reynold Brown, whose style became bolder and less detailed, and in the case of the artist Loring Hughes, whose style became less realistic and more emotionally expressive (Heller, 1994).

OTHER CASES

Other kinds of brain damage can also leave visual artistry intact or alter it in rich and wonderful ways. A striking example is the work of Willem de Kooning after Alzheimer's disease. His late life work was featured in a major exhibit in 1997 at the Museum of Modern Art in New York (Smith, 1997). After Alzheimer's disease, his paintings were described as declining

in "de Kooningness" but not in visual strength and assurance (Smith, 1997). The paintings were loose and free, dramatic and challenging.

Recently the strange case of Jon Sarkin was reported, a chiropractor who had surgery to correct a distended blood vessel pushing against the acoustic nerve in his left ear (Corsello, 1997). Unexpected bleeding post-surgery led to another operation, this time resulting in the removal of part of the cerebellum. When Sarkin recovered, he was a changed person. He was now disorganized and chaotic and manic in energy, driven to philosophical ramblings and poetry, and intricate, strange, humorous paintings. He became obsessed with color, and felt that for the first time he could really SEE color. He had a powerful, uncontrollable urge to paint. It was as if a filter that had previously reined in Sarkin's artistic drive had been lifted, and he was left in an unfocused state in which he painted without knowing what he was doing.

Neither the case of de Kooning nor of Sarkin have been adequately explained. We do not know what it is about their altered brains that led to their altered artistry (or, in the case of Sarkin, to his new-found artistry). Nonetheless, these two cases show us in startlingly ways that artistry can exist and flourish in the face of devastating brain injury.

In summary, studies of both artists and nonartists with unilateral brain damage show that even though both hemispheres play a role in the visual arts, the right hemisphere is dominant. In the case of nonartists, the left hemisphere is important for rendering the details of a represented object, and the right hemisphere is important for capturing the overall form. The graphic capabilities of the right hemisphere have been put to use in several aphasia therapy programs, all of which encourage aphasics to communicate through drawing.

In the case of artists, the role of the left hemisphere is less clear. Painters with severe aphasia have been able to continue painting, although the quality and style of their poststroke work changed. In one case, a left posterior parietal tumor led to deficits in visuospatial and constructional skills. Despite the variety of sequelae of left-hemisphere damage in artists, it is clear that graphic skills in an artist can and do function independently of linguistic and other left-hemisphere skills. When an artist suffers right-hemisphere damage, on the other hand, the paintings become altered in at least two ways. There is a noticeable neglect of the left side of the pictorial space, and the style typically becomes more emotional and direct.

Literature

For a long time it was assumed that only the left hemisphere was involved in language processing. But there is by now a large body of evi-

dence that the right hemisphere also makes important (and different) contributions to language. The right brain is capable of uttering overlearned phrases (Taylor, 1932); it can process vowels, intonation contours, and affectively tinged language (Blumstein & Cooper, 1974; Cicone, Wapner, & Gardner, 1980; Heilman, 1976; Kimura, 1973); and it may even possess some vocabulary and syntax (Gazzaniga, 1970; Sperry, 1974; E. Zaidel, 1973, 1977, 1978a, 1978b). The right hemisphere also makes a far more important contribution to literary skill than does the left hemisphere.

In evaluating the effects of brain damage on literary skills, the important questions do not concern performance on aphasia batteries or other standard linguistic tasks. Literary skill requires far more than syntax, semantics, or phonology. What is important is that ability to go beyond the literal and appreciate metaphor, irony, and humor. Equally important is the ability to recognize a story, to grasp the rules of narrative structure, and to be aware of the boundary between fact and fiction.

Given the centrality of the left hemisphere in language, one might expect that aphasia would be associated with poor performance on tasks assessing literary skill. After all, such tasks tap higher-order levels of language, which might depend on more basic components of language. However, left-hemisphere damage appears to be far less destructive of literary abilities than is right-hemisphere damage.

Literary Ability after Left-Hemisphere Damage

Presented with a description of a person having a "heavy heart," aphasics have difficulty explaining the meaning of this metaphor (Winner & Gardner, 1977). But this difficulty stems simply from an inability to put their understanding into words. Provided with a nonlinguistic response mode in which they may simply point to a picture that goes with such a metaphoric statement, aphasics performed nearly as well as normal individuals (Winner & Gardner, 1977). Similar findings were reported by Stachowiak, Huber, Poeck, & Kerchensteiner (1977): aphasics could match a picture with a story ending even when the end of a story contained a metaphorical expression. Aphasics also performed well when asked to match a picture to an idiomatic sentence (e.g., "He's turning over a new leaf"), despite performing poorly when asked to match a picture to an unusual but literal sentence (e.g., "He's sitting in the bubbles"; Van Lancker & Kempler, 1987; see also Myers & Linebaugh, 1981, for evidence that left-hemisphere damage does not impair idiomatic understanding). Thus it appears that the analysis of the figurative meaning of a metaphor or an idiom is dissociated from left-hemisphere sentence parsing and is carried out by the preserved right hemisphere.

It has been suggested that LBD patients may use nonsyntactic cues to understand idioms (Burgess & Chiarello, 1996). For instance, take the idiom, "He kicked the bucket." Because this idiom is a nonreversible sentence (unlike "The boy kicked the horse," in which either boy or horse could be doing the kicking), patients may recognize that the bucket is the thing being kicked on the basis of real-world rather than syntactic knowledge. They may then access in memory the concept of bucket as object, and thereby "bootstrap" their way to a figurative interpretation.

Aphasics also do not appear to lose sensitivity to narrative structure. They are able to extract the main ideas from a text (Brookshire & Nicholas, 1984; Stachowiak et al., 1977), and they can understand familiar scripts such as going to the doctor (Armus, Brookshire, & Nicholas, 1989). Thus, they have retained their sensitivity to the macrostructure of discourse, even if they have lost semantic or syntactic knowledge. Although aphasia may thus leave relatively intact the ability to APPRECIATE figurative language and narrative, such damage cripples the ability to PRODUCE literature. Aphasics have been shown to have difficulty summarizing stories concisely, they often insert extraneous propositions (Ulatowska, Allard, & Chapman, 1990), and they produce "coherence violations" when telling a story (Christiansen, 1995; see also Kukkonen, 1993, 1995).

In no case has an aphasic writer been able to continue to write. Charles Baudelaire, the nineteenth-century French poet, became severely aphasic after a left-hemisphere stroke. He never wrote again, and the only words he could utter were an oath: "*cré-nom*" (Alajouanine, 1948). Of course, if a writer recovers from aphasia, writing may resume: William Carlos Williams was able to go back to writing poetry after a partial recovery from aphasia (Plimpton, 1977).

Literary Ability after Right-Hemisphere Damage

On the surface, RBD patients seem to possess intact language. Yet closer inspection reveals a different story. Patients with lesions outside of the traditional language areas, who show no deficits on traditional aphasia batteries, have been found to perform poorly on a number of higher-order language tasks: they are pervasively literal minded and have difficulty making the inferences necessary to understand a story (for a review, see Joanette, Goulet, & Hannequin, 1990). These are patients with damage in the right hemisphere, or in the frontal lobes, or both (see McDonald, 1993b, for the difficulty in distinguishing between right-hemisphere and frontal lobe language disorders).

Unlike LBD patients, those with right-hemisphere damage often speak in a manner that sounds metaphoric (e.g., a patient might joke about a par-

alyzed arm, calling it his or her "old fin"). Yet, when asked to paraphrase the metaphoric phrase "heavy heart," they were resistant, insisting that such language is not proper English (Winner & Gardner, 1977). Asked to point to the picture that went with the description, they were as likely to choose a literal depiction as a metaphoric one. Moreover, unlike patients with left-hemisphere damage or non–brain-damaged controls, they failed to find the literal pictures amusing. However, these RBD patients revealed a dissociation: although they could not reliably select the metaphoric interpretation shown in a picture, they were, upon prodding, able to provide an appropriate verbal paraphrase. Thus, their problem does not seem to be a loss of metaphoric understanding per se, but rather a RESISTANCE to metaphorical interpretation. They also did not notice any conflict between their verbal paraphrase of, for example, "heavy heart" as "sad" and their literal picture choice of a person lugging a heart-shaped object.

The same resistance to metaphor in those with right-hemisphere damage was shown by Gardner and Denes (1973) on a task in which pictures such as up and down arrows had to be matched with verbal concepts such as "wealth" or "poverty." These patients often objected that the task was impossible. Given triads of words such as *warm, loving,* and *cold,* and asked to judge which two words were most closely associated, LBD patients grouped metaphorically (e.g., *warm* with *loving*), whereas RBD patients grouped antonymically (e.g., *warm* with *cold*; Brownell, Potter, Michelow, & Gardner, 1984).

The difficulty the patients in Brownell et al.'s (1984) study had might have been due to a difficulty in retrieving the subordinate meanings of words rather than to a specific difficulty with metaphorical meanings per se. Such an interpretation is consistent with research showing that retrieval of subordinate word meanings takes longer in the right than in the left hemisphere (Burgess & Simpson, 1988b). However, a later study by Brownell and his colleagues demonstrated that RBD patients have particular difficulty with subordinate meanings that are metaphoric (Brownell, Simpson, Birhle, Potter, & Gardner, 1990). Patients were again given triads of words and asked to group the two most similar ones. One half of the triads allowed a match between a word and a subordinate, metaphoric meaning (*deep* with *wise*) and the other half allowed a match between a word and a subordinate, nonmetaphoric meaning (*suit* with *trial*). RBD patients performed worse than LBD patients only on the metaphoric matches.

The studies described thus far demonstrating RBD patients' impairment on metaphor have all assessed deliberate, conscious (off-line) processing. Given tasks that assess automatic, nonconscious processing, RBD patients show no impairment. Tompkins (1990) demonstrated this using a lexical decision task. Patients saw a string of letters and had to decide

whether the string was an English word, a task which requires no overt judgment about word meaning. When the target letter string was preceded by a prime related metaphorically in meaning to the target, RBD patients showed normal priming effects: For example, a prime such as "loving" facilitated recognition of "warm" as a word. However, patients were impaired in their ability to show conscious understanding of metaphorical meaning (see also Brownell & Malloy, 1990). Thus, RBD impairs conscious access to metaphorical interpretations, but leaves unconscious metaphorical connections intact.

The role of the right hemisphere in metaphor comprehension has been confirmed in a PET study with normal subjects (Bottini et al., 1994). Understanding metaphoric sentences activated not only the left hemisphere but also a number of sites in the right hemisphere—the prefrontal cortex, the middle temporal gyrus, the precuneus, and the posterior cingulate. Comprehension of idioms is also impaired by right-hemisphere damage, but again, only when comprehension is assessed in an off-line task. When asked to demonstrate conscious understanding of idioms by selecting a picture that illustrates the idiomatic meaning, RBD (but not LBD) patients did poorly (Van Lancker & Kempler, 1987). However, RBD patients had no difficulty assessing idiomatic meaning on an on-line, automatic task (Tompkins, Boada, & McGarry, 1992). Patients were asked to detect target words in idiomatic and literal sentences. For example, they had to detect the word *rat* in "My lawyer was studying my contracts. When he smelled a rat, he warned me," and in "My cat was hunting one night. When he smelled a rat, he attacked it." Both LBD and RBD patients' responses to target words were faster when they were in idiomatic contexts. Thus, the difficulty that RBD patients have with idioms is not at the initial unconscious stage, but at a later stage. (For evidence of right-hemisphere–damaged patients' difficulty with proverb understanding, see Van Lancker, 1990, and Hier & Kaplan, 1980). Patients with RBD also have difficulty understanding ironic, sarcastic, or joking language. Ironic utterances, like metaphoric ones, are not intended literally. To understand them one must ignore the literal meaning and realize that the speaker means (roughly) the opposite of what was said (Winner, 1988). When RBD patients were given stories ending with ironic comments which were sarcastic and nasty, or light and joking, they failed to use explicit mood information to determine that the utterance was intended nonliterally (Brownell, Carroll, Rehak, & Wingfield, 1992). When given stories ending in ironic utterances or in white lies, RBD patients were unable to use knowledge about the speaker–listener relationship to determine whether the speaker was telling a white lie (to be nice) or making a sarcastic comment (to be hostile; Kaplan, Brownell, Jacobs, & Gardner, 1990). When given stories that ended either in ironic jokes

or in lies, RBD patients performed inconsistently and unreliably (Winner, Brownell, Happé, Blum, & Pincus, 1998). In addition, their difficulty in distinguishing ironic jokes from lies was related to a problem in making inferences about what the speaker believed about what the listener knew. Thus, RBD patients seem to have an impaired ability to make mental state inferences, inferences which are needed to determine whether a false utterance is ironic (joking) or deceptive (see also Happé, Winner, & Brownell, 1997).

Sensitivity to context is not only important in understanding various kinds of figurative language, it is also important in understanding fictional narrative. Understanding a narrative requires an ability to enter into the story, attend to the story line and its coherence, and infer events that are implied but not directly stated. RBD patients have difficulty with many of these higher-order processing skills. They do not make appropriate use of contextual knowledge in making assumptions or predictions about what a speaker means. Furthermore, they are often reluctant to revise their initial interpretations of sentences or situations when presented with new contextual information (Molloy, Brownell, & Gardner, 1990).

A number of studies provide further evidence of the deficits shown by RBD patients in the processing of contextualized language. These patients have difficulty understanding discourse in which a speaker abruptly shifts the topic of conversation; they fail to realize that the speaker is trying to change the subject, probably because they do not appreciate that coherence has been violated (Rehak, Kaplan, & Gardner, 1992). They also have difficulty interpreting indirect requests (Foldi, 1987; Hirst, LeDoux, & Stein, 1984; Weylman, Brownell, Roman, & Gardner, 1989) because they make inadequate use of the context provided. Hirst et al. (1984) showed LBD and RBD patients videotaped episodes in which one actor asked another, "Can you *X*?" The other actor responded by either doing what was requested or by saying, "Yes." Aphasics understood that it was appropriate to respond to such a question with an action if the context indicated that it was meant as an indirect request, but they proved unable to understand the literal meaning of the question. In contrast, RBD patients always interpreted the question literally, regardless of the context in which it was asked. Foldi (1987) also found that, relative to both aphasics and controls, RBD patients preferred literal interpretations of indirect requests over pragmatically appropriate ones. In Weylman et al.'s (1989) study, subjects heard short stories with contextually appropriate indirect requests at the end; both RBD and LBD patients had significantly more difficulty than controls in responding correctly to the requests, but aphasics were at a clear disadvantage because no visually presented information accompanied the text.

Right-hemisphere damage also impairs both the ability to understand

and to generate narratives. RBD patients are insensitive to key structural features of stories (Delis, Wapner, Gardner, & Moses, 1983; Gardner, Brownell, Wapner, & Michelow, 1983; Joanette, Goulet, Ska, & Nespoulous, 1986; Moya, Benowitz, Levine, & Finklestein, 1986; Wapner, Hamby, & Gardner, 1981). These patients have no difficulty understanding and retaining the facts of a story, but they have trouble ordering events correctly, deriving the main point or the moral of a narrative, and distinguishing between fact and fabrication. This last point was shown in a study by Gardner et al. (1983) that assessed sensitivity to the insertion of noncanonical elements into stories. Normal controls generally judged these elements to be bizarre and did not incorporate them into their retellings of the stories. Aphasics altered the information so that it made sense within the context of the story. RBD patients, however, accepted the bizarre elements as normal parts of the story and included them in their retellings, sometimes fabricating elaborate scenarios in an attempt to integrate all of the information. Thus RBD patients have lost a sense of what elements make up the canonical form of a story.

Narrative comprehension difficulties also manifest themselves in a failure to make appropriate inferences. For instance, when asked to select the main theme for a paragraph-long story, RBD patients succeeded when the theme was explicitly stated at the beginning but not when the theme was given only near the end (Hough, 1990; Hough & Pierce, 1993). Thus they had difficulty in providing their own thematic organization of a brief story. Similarly, Schneiderman, Murasusi, and Saddy (1992) found that RBD patients had difficulty arranging sentences into a coherent paragraph. Unlike patients with left-hemisphere damage, they were not aided by the presence of a theme sentence containing the macrostructure of the paragraph.

One reason why RBD patients may have difficulty with story understanding is that they resist or are unable to revise their interpretation of a story as it unfolds (Brownell, Potter, Birhle, & Gardner, 1986). This inability to revise also impairs RBD patients' appreciation of jokes. Bihrle, Brownell, Powelson, and Gardner (1986) presented patients with the first three frames of captionless cartoon strips. The task was to select the final frame that would make the cartoon strip funny. RBD patients often chose nonsequitur final frames—frames which preserved the element of surprise necessary for humor but lacked coherence with the body of the joke. In contrast, although aphasic patients sometimes confused humorous final frames with nonhumorous but sensible ones, they rarely chose nonsequitur final frames. Right-hemisphere damage even appears to impair story inferencing ability in an automatic, on-line task: Beeman (1993) showed that RBD patients are not helped in deciding whether a string of letters is

a word even when that word is related to an inference that should have been made during comprehension.

Nonetheless, RBD patients do not suffer from an across-the-board deficit in story processing. Rehak, Kaplan, Weylman et al. (1992) showed that these patients retained a sense of what is going to happen next in canonically structured (suspense) stories, a finding that is consistent with the fact that they have a preserved knowledge of scripts (Roman, Brownell, Potter, Seibold, & Gardner, 1987) and a preserved understanding of the structure of jokes (Birhle et al., 1986). Rehak, Kaplan, Weylman et al. found that RBD patients were better at story comprehension when the story is interesting, suggesting that they CAN attend to and make sense of a story if they are sufficiently interested. RBD patients are quite impaired in the ability to generate even very simple "stories." For instance, Myers and Brookshire (1996) asked patients to explain illustrations by Norman Rockwell. One picture showed two men and a boy sitting in a doctor's waiting room. RBD patients often failed to mention that they were in a waiting room, thus missing the main theme of the picture. Other studies report similar results: when asked to narrate pictorially presented stories, these patients omitted the main theme, mentioned too much unimportant detail, had no overarching organization to determine which information is central, and filled their responses with tangential remarks and embellishments (Joanette et al., 1986; Myers, 1993; Wapner et al., 1981).

Literary Abilities after Frontal Lobe Damage

Patients with dorsolateral frontal lobe damage have language disturbances not unlike those that have been reported for RBD patients (McDonald & Pearce, 1996). Like that of RBD patients, frontal lobe patients' speech is disorganized and filled with confabulations and irrelevancies (Alexander, Benson, & Stuss, 1989; Alexander & Freedman, 1984; Luria, 1976; Stuss, Alexander, Lieberman, & Levine, 1978). Like those with right-sided damage, they are concrete in their interpretations of language and fail to make use of conversational inference (McDonald, 1993b; Novoa & Ardila, 1987). And like those with right-hemisphere damage, they have difficulty producing sustained narratives (Damasio & Van Hoesen, 1983; Rubens, 1976). McDonald (1993b) noted the difficulty of distinguishing clearly between left and right prefrontal lobe functions. She found that in the case of medial frontal damage, it is difficult to pinpoint frontal damage to the left frontal lobe and not to the right as well.

Frontal lobe patients show the same deficit in irony comprehension as do patients with right damage. McDonald and Pearce (1996) asked patients with frontal lobe damage to answer questions about ironic interchanges

such as, "What a great football game. Sorry I made you come." The only way to make sense of this exchange is to interpret one of the sentences as meaning its opposite. Although frontal lobe patients performed as well as non–brain-damaged individuals on literal interchanges, they performed worse than the control group on the ironic ones, even when sarcastic intonation was included as a clue. Their difficulty in making sense of the irony was unrelated to their ability to infer emotion from tone of voice, but it was highly related to their performance on the Wisconsin Card Sort Task (WCST). McDonald and Pearce note that success on the WCST requires that one ignore certain salient attributes and sort on some other dimension. Similarly, they argue, understanding irony requires that one ignore the salient literal meaning and base one's interpretation on the underlying intended meaning. Thus they suggest that the frontal lobes are involved in going beyond the surface, literal meaning (see also McDonald, 1992, 1993a).

The similarity between right-hemisphere and frontal lobe language impairments may be due to an overlap in the patient population (Alexander et al., 1989; McDonald, 1993b). McDonald (1993b) demonstrated that in the majority of studies of RBD patients, patients with right frontal (anterior cortex) lesions were included in the right-damaged group, because the most common cerebrovascular accidents resulting in right-hemisphere damage are in the distribution of the right middle cerebral artery, which affects parietal and temporal lobes, and inferior and anterior aspects of the frontal lobes. In the few studies that have classified right-sided lesions as either purely anterior or purely posterior, it is the frontal patients who have the greatest difficulty (McDonald, 1993b). For example, a preference for literal responses to indirect speech acts was found in studies that included patients with frontal damage (Foldi, 1987; Weylman et al., 1989), but not in studies of patients with damage restricted to temporal parietal areas (Hirst et al., 1984). Wapner et al. (1981) found that frontal damage leads to greater embellishment of responses than does posterior damage, and Benowitz, Moya, and Levine (1990) showed that right frontal damage was associated with greater impairment in inferencing than was right anterior damage.

Although the language deficits seen in RBD patients may in part be caused by the presence of frontal lobe damage, nonfrontal areas of the right hemisphere may still play a role in nonliteral language understanding. Posterior lesions in the right hemisphere also result in language disturbances (Hirst et al., 1984; Tompkins & Mateer, 1985), plus the temporal and parietal areas of the right hemisphere are important in sensitivity to prosody (McDonald, 1993b).

The ability to understand and produce literary language requires both hemispheres. Severe aphasia, of course, severely impairs any kind of liter-

ary ability. Nonetheless, despite the left hemisphere's undisputed dominance for language, the right hemisphere is crucial in determining the intention behind an utterance and in relating an utterance to its linguistic, situational, and narrative context. Because patients with right hemisphere damage are often unable to recognize context and intention, they misinterpret nonliteral language. Moreover, because of the right hemisphere's attention to context and narrative structure, this hemisphere plays a critical role in story understanding. However, whether all of these deficits are specific to any kind of right-hemisphere damage, or whether it is particularly the involvement of the right frontal lobes that leads to these impairments, is not yet clearly established.

Conclusion

Aphasia bears a different relationship to each of the three art forms considered here. The relationship is never a simple one, and the pattern found often differs, depending on whether gifted or average individuals are being discussed. Despite the complexity of the findings, the following generalizations can be offered.

Disorders of language sometimes leave musical skill relatively unaffected. However, aphasia and amusia often co-occur, suggesting either shared or contiguous neural processes. The right hemisphere has a particularly important role to play in music, contributing not only to technical skill but also to the individual's affective relationship to music. Yet, it may well be that in the case of the highly trained individual, left-hemisphere damage impairs the ability to perceive a piece of music in an analytic mode. Furthermore, the representation in the brain of musical abilities seems to differ greatly from one talented musician to another.

In the case of the visual arts, aphasias in the average individual appear to entail some loss of graphic ability. However, in artists, in whom painting and drawing are overlearned skills, graphic ability continues to function largely independently of language and other left-hemisphere abilities. Once again, it is right-hemisphere damage that yields the most potent effects, altering style and emotional tone.

It is with literature that one would expect the strongest relationship between aphasia and artistry. Indeed, left-hemisphere damage affects the language of the writer no less than that of the average individual. Overlearning appears to be no protection against the ravages of aphasia, and the language abilities of the writerare indissolubly tied to linguistic competence per se. The critical role of the left hemisphere in the case of the literary arts does not, however, mean that the right hemisphere is not involved.

The right hemisphere is essential in governing attention to linguistic and extralinguistic context. Lacking such sensitivity, RBD patients often misinterpret nonliteral language and are insensitive to the structural underpinnings of narrative materials such as jokes or stories. RBD patients may well benefit from efforts to sensitize them to figurative or fictive uses of language. Moreover, communication may initially proceed most effectively if their proclivity to take messages literally is kept in mind. Correlatively, even aphasic patients with severe compromise of ordinary language functions may retain basic understanding of verbal humor and narrative constructions. Upon these spared capacities it may be possible to build or to resurrect enhanced understanding of the messages of daily life, as well as to provide an entry to simple works of literature.

Acknowledgments

Preparation of this chapter in earlier editions was supported by the Veterans Administration; the National Institutes of Neurological Diseases, Communication Disorders, and Stroke (MS 11408); and Harvard Project Zero. Preparation of the current version of this chapter was aided by a Boston College Research Expense Grant to the first author (E.W.) to support research on right-hemisphere–damaged patients' difficulties in mental state inferences. We thank Howard Gardner, first author of this chapter in this volume's two earlier editions, and Hiram Brownell for their comments and suggestions.

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12

Aging, Language, and Language Disorders

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Although much of our early knowledge of aphasia was obtained from studies of young men injured in war, research into aphasia in the past 40 years has studied primarily older adults suffering the consequences of strokes or neurodegenerative disorders, such as Alzheimer's disease. In this last half of the century we have gained substantially more knowledge of how the brain itself changes with age and of the pathologies often associated with aging. With respect to language disorders in aging, one question has been whether lesions in young adults result in different clinical patterns of aphasia from those in older adults with comparable lesions, and how brain changes associated with older adulthood influence recovery from aphasia.

To answer these and related questions, we must first determine how language changes with age among normal individuals, as clearly aphasia in older adults overlies those normal patterns. Moreover, because the apparent language changes associated with healthy aging may not be language changes per se, but rather language manifestations of changes in broader cognitive abilities not strictly related to language, such as working memory or speed of cognitive processing, we also address these issues. We include a discussion of what is known of the cellular changes in the brain that presumably underlie the language and cognitive changes associated with normal aging.

After outlining the relationship between language, cognition, and the brain in healthy aging, we can then address how changes in these domains

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interact with aphasia to affect aphasia type and recovery from aphasia, and consider explanations that have been given for the patterns observed. Finally we complement our discussion of aphasia as it relates to aging with a discussion of the language changes of Alzheimer's disease and Dementia with Lewy bodies. These changes are similar to those of aphasia in many ways but there are also important differences. Here again we consider explanations to account for language changes, most crucially, disorders of semantic memory.

Language in Normal Aging

In a chapter devoted to language functioning in an aging brain, it is necessary to review what is known about normal language functioning in older individuals without known brain pathology, that is, what language components change by virtue of the aging process itself in the absence of disease. It was thought in the not too distant past that language was one cognitive domain in which little change was expected with normal aging. For example, several researchers reported that vocabulary did not change with age, or if anything, improved with age (Bayles, Tomoeda, & Boone, 1985; Fox, 1947; Lewinsky, 1948; Owens, 1953; Shakow & Goldman, 1938; Thorndike & Gallup, 1944). Based on research over the last two decades, we now know that several components of language function decline with age and that these components change at different rates. Even in the domain of vocabulary it has been demonstrated that subtle deterioration occurs in the quality of the definitions of words as individuals age (Botwinick & Storandt, 1974). We review what is known about age-related change in language, or the lack thereof, for the components of lexical retrieval, discourse production, and language comprehension. We then discuss age-related declines in working memory, the failure to inhibit irrelevant information, cognitive slowing, and neuroanatomical explanations that link language deficits to age-related changes in the brain.

Lexical Retrieval

The most common language complaint among elderly people is the frequency of word retrieval failures. Many investigations have documented the greater incidence of retrieval failure in older adults in noun picture naming tasks, such as the Boston Naming Test (BNT), in both cross-sectional studies (M. S. Albert, Heller, & Milberg, 1988; Borod, Goodglass, & Kaplan, 1980; Goulet, Ska, & Kahn, 1994; LaBarge, Edwards & Knesevich, 1986; Nicholas, Obler, Albert, & Goodglass, 1985; Van Gorp, Satz, Kiersch, & Henry,

1986) and longitudinal studies (Au et al., 1995). Similarly, verb retrieval failures have also been documented in cross-sectional (Nicholas, Obler, Albert, & Goodglass, 1985) and longitudinal studies (Barth, Nicholas, Au, Obler, & Albert, 1998), as well as retrieval failures associated with producing the referent to a definition (Bowles & Poon, 1985; Burke, MacKay, Worthley, & Wade, 1991) and retrieving proper names (Barresi, 1996; Cohen & Burke, 1993; cf. Maylor, 1996). Lexical retrieval failures begin in individuals as early as in their 50s but are more pronounced in individuals in their 70s. Recent longitudinal data from our own laboratory (Connor, Obler, Albert, & Spiro, 1998) suggest that individuals who have reached their mid-80s have declined more than 2.5 *SD*s relative to individuals in their mid-30s in noun retrieval, and nearly 3.5 *SD*s below the 30-year-olds in verb retrieval.

Discourse Production

Unlike the domain of lexical retrieval, discourse production appears to be largely preserved with advanced age, although there may be some subtle changes. At the least, interpretation of the discourse findings is complicated by several factors. Obler et al. (1994) argue that conflicting reports of whether or not there are age-related declines in discourse may be the result of differences in methodology in the studies attempting to measure changes in discourse processing with age and intersubject variability in communication style. Furthermore, they argue that the lack of differences or the nonlinear differences in discourse may be underreported due to the "uninteresting" nature of null results or to the lack of ability to explain nonlinear results with the current models of language production.

Despite the complexity of interpreting the discourse findings, there are a few reports in which a straightforward age-related decline in some aspects of discourse was found. Kynette and Kemper (1986) found in analyses of free speech that older adults preferred to use simpler, less memory-demanding syntactic constructions and more often made errors in their use of simple syntactic structures than did younger adults. There was, however, no age difference in mean length of utterance, or in the number of sentence fragments or the number of filler words used. S. Kemper (1987), in a longitudinal analysis of diaries, also found that syntactic complexity decreased with age. North, Ulatowska, Macaluso-Haynes, and Bell (1986) and North and Ulatowska (1981) obtained age-related differences in discourse processing and production on a number of tasks, and these differences correlated with declines in other cognitive abilities. In addition, the quality of the older adults' responses to interview questions was rated as being poorer than middle-aged adults' responses. The results of these two studies, however, may be accounted for largely by age-related differences

in the comprehension of language and in the memory demands of comprehension, rather than strictly production of language. Two tasks included in their battery that minimized memory and comprehension demands (telling a story based on a series of pictures and telling the procedures necessary to perform some common tasks) produced only subtle declines for the older adult age group.

Language Comprehension

As mentioned, language comprehension appears to be a domain for which older adults experience a larger amount of decline in performance than for discourse production. Feier and Gerstman (1980) reported deficits in comprehending complex sentences that contained center embedded relative clauses in a task where participants demonstrated the actions conveyed by the test sentences with figurines. These deficits first appeared in the 60-year-olds and were firmly established in the 70-year-olds. Furthermore, the older group made errors with more severe interpretive consequences than did the younger groups.

In a similar vein, Emery (1985, 1986) tested healthy older adults on the Token Test and on a test of syntactic complexity and found older adults performed worse than middle-aged adults. Obler, Nicholas, Albert, and Woodward (1985) reported poorer comprehension for the elderly than for the young when sentences were presented with background speech noise and when semantic predictability was low. Longitudinal and cross-sectional studies by Kemper and colleagues (S. Kemper, 1986, 1987; Kynette & Kemper, 1986) revealed that not only do older adults produce less syntactically complex sentences in spontaneous speech, but they are less able to imitate syntactically complex constructions. The authors argue that imitation is impaired because comprehension and memory decline with age. Furthermore, the ability of older adults to comprehend pronominal references when listening to or reading texts is also reported to be impaired, particularly when subject and pronoun are separated by other material (Light & Capps, 1986).

Two more recent studies manipulated the syntactic construction and semantic plausibility of sentences to determine whether age affected comprehension differentially for these components (G. A. Davis & Ball, 1989; Obler, Fein, Nicholas, & Albert, 1991). The results of both studies indicated that complex syntactic constructions and implausible sentences were more difficult for the older adult groups. The trend was for more errors to occur the older the group tested. The weight of all of the reviewed studies leads to the conclusion that there are fairly large and consistently obtained age-related differences in the ability to comprehend linguistic material.

Theoretical Explanations for Language Changes in Normal Aging

Up to this point, we have reviewed the evidence for age differences in the domains of lexical retrieval, production, and comprehension, but have offered no explanations of these language declines other than to say that "memory" may contribute. The remainder of this review of language change in normal aging focuses on the theoretical explanations that have been offered to account for the age differences that we have reported. We review these types of explanations for each of the language domains. The first type of explanation for age changes in language performance are explanations that root the problem within the linguistic domain itself.

Intralinguistic Explanations

LEXICAL RETRIEVAL

What then are the underlying linguistic mechanisms for lexical retrieval failure in aging? Two possibilities have been suggested. First, the lexical–semantic representation of the word may have deteriorated with aging. This hypothesis is not favored as an explanation for lexical retrieval deficits in healthy older adults, but has found favor in a significant number of investigations as an explanation of lexical retrieval failures in Alzheimer's disease. Second, age-related lexical retrieval failures may result from temporary inaccessibility of the phonological code for the retrieval target. This account is thought to be a more plausible explanation of age-related lexical retrieval failures in healthy adults and is consistent with data indicating that older adults often benefit from phonemic cues for irretrievable targets (Nicholas, Obler, Albert, & Goodglass, 1985).

Consistent with the inaccessibility of phonological codes, Burke et al. (1991) reported that the incidence of tip-of-the-tongue (TOT) phenomena increases with age. They account for these data with a network model that partitions language information into a semantic level, a phonological level, and an articulatory system. In this model, activation accrues at various nodes in the system and priming spreads through connected nodes resulting in accurate speech output. Aging weakens the connections among nodes. Therefore, priming of nodes in the semantic system is unable to spread to the appropriate nodes in the phonological system due to the weakening of the links between these networks; the consequence is a TOT state. This Transmission Deficit Hypothesis accounts for age-related increases in lexical retrieval failure, the increased incidence of TOT states as individuals age, and two results from the cuing data. First, healthy older adults do not benefit from semantic cues and often report information

that allows the inference to be made that the semantic information is available to the individual. Second, cross-sectional research suggests that older adults often benefit from phonemic cueing. Phonemic cues are thought to boost activation at the phonological level and, in the Burke et al. architecture, surpass threshold to enable retrieval of the lexical target.

The idea that healthy older adults have only phonological activation problems and that individuals with Alzheimer's disease experience deterioration of the semantic representation has been a popular one. There are, however, recent data that challenge this interpretation. Au et al. (1995), in a longitudinal analysis of the BNT, found that individuals in their 70s do not benefit from phonemic cues as much as younger individuals. Furthermore, in the same group of individuals, Barresi, Nicholas, Connor, Obler, and Albert (1997) analyzed the pattern of naming responses and responses to cuing for the BNT over three longitudinal test sessions (7 years) to evaluate the degree to which lexical retrieval failures could be classified as consistent with a semantic loss account or an inaccessibility account. They found that for individuals who began the study in their 30s, 50s, or 60s, the pattern of lexical retrieval failure was consistent with the inaccessibility of phonological information. For individuals who began the study in their 70s, however, a more complex pattern emerged. That is, although inaccessibility was largely responsible for retrieval failures for all groups, there was also a significant contribution of semantic loss to the retrieval failures of the oldest group. Thus, it now appears likely that both inaccessibility and semantic deterioration play a role in the lexical retrieval failures of healthy individuals over the age of 70.

DISCOURSE PRODUCTION

Because language production exhibits minimal age changes and because the changes that do occur appear to be influenced by extralinguistic factors (we will return to this issue later), no linguistic deficit hypothesis has been offered to explain age-related changes in production. One exception may be the finding by S. Kemper (1986) that older individuals have difficulty imitating left-branching syntactic constructions, that is, phrases in which modifying clauses precede the modified element. One possible explanation for this result may be difficulty with the syntactic processor itself. As stated earlier, the poor imitation performance of the elderly for complex syntactic constructions may also be the consequence of a problem with the comprehension of the constructions.

LANGUAGE COMPREHENSION

This leads to a discussion of the language comprehension deficit exhibited by older adults. Many researchers have proposed that these deficits

are in fact problems with syntactic processing as people age (G. A. Davis & Ball, 1989; Feier & Gerstman, 1980; S. Kemper, 1986, 1987; Obler et al., 1991). S. Kemper (1986, 1987) proposed that the decrement in the spontaneous use of complex syntactic constructions and the decline in the ability to imitate left-branching sentences were due to an "age-related decline in syntactic processing abilities" (S. Kemper, 1986, p. 277). Similarly, G.A. Davis and Ball (1989) and Obler et al. (1991) reported that the likely cause of lower comprehension scores for complex constructions was attributable to declines in syntactic processing, as opposed to declines in syntactic knowledge.

Extralinguistic Explanations

WORKING MEMORY

What causes the changes in syntactic processing? To determine whether the changes in comprehension are due to changes in syntax itself or to another factor that influences syntactic processing, all of the studies on language comprehension have speculated on the role that extralinguistic factors such as memory load play in successful language comprehension. These studies come to different conclusions regarding the influence of these factors. Although G.A. Davis and Ball (1989) give a nod to the notion that working memory plays a role in the syntactic processing declines evidenced by the older adults in their study, they did not measure working memory span in their subjects, nor did they design their stimuli along a hypothetical dimension of working memory load. In contrast, S. Kemper (1986) hypothesized that memory demands should be particularly acute for older individuals, predicting that older adults would have difficulty imitating sentence-initial embedded clauses—differentially so for longer, sentence-initial clauses. Younger adults, she predicted, would have little difficulty with either sentence-initial or sentence-final embedded clauses and would show no effect of clause length. Kemper obtained the predicted Age Group × Clause Position × Clause Length interaction, therefore supporting her contention that memory load influences the imitation of complex syntactic constructions.

Obler et al. (1991) took an individual differences approach to the question of whether or not extralinguistic processes influence performance on a complex syntactic processing task. They examined correlations and performed analyses of covariance to determine the degree to which neuropsychological performance on measures of attention, short-term memory, and mental control accounted for age differences in comprehension. Measures of speed and capacity for sustained attention produced significant correlations with comprehension performance, but only the speed

measure when entered as a covariate attenuated age group differences in comprehension. Thus, Obler et al. (1991) concluded that memory did not exert an influence on comprehension.

Many of these studies, particularly in the domain of language comprehension, allude to the possibility of working memory deficits in older adults as an explanation for language-related decline. Many of these same authors concluded that working memory did not prove to be an explanatory factor in their experiments. This conclusion may be premature in that these studies often used a digit span or other simple span task as a measure of working memory. Span tests have produced few age-related differences in performance. Thus, span can only minimally, if at all, account for age differences in performance. Current conceptions of working memory have two components, a storage component and a manipulation component (Salthouse & Babcock, 1991; Salthouse, Babcock, & Shaw, 1991). Simple span tests do not evaluate the ability of individuals to manipulate information stored in working memory. They also do not take into account the effects on storage of high demands on manipulation (Perfetti & Lesgold, 1977; see Just & Carpenter, 1992, for a complete account). It is possible that given both storage and manipulation demands in a task, age-related differences will emerge that can account for age differences in language performance. Studies examining individual differences in working memory for younger adults have found predictive power in the working memory span score in explaining individual differences in comprehension (Daneman & Carpenter, 1980, 1983; Daneman & Merikle, 1996; Just & Carpenter, 1992; King & Just, 1991). A few studies have demonstrated a relation between the more restrictive definition of working memory (both storage and manipulation components) and language comprehension in the elderly (Gick, Craik, & Morris, 1988; Morris, Gick, & Craik, 1988; Salthouse, 1990; Stine & Wingfield, 1987; Stine, Wingfield, & Poon, 1986; Tun, Wingfield, & Stine, 1991).

INHIBITION

Intimately tied to working memory accounts of age differences in language performance are accounts of age-related changes in inhibitory processes. Hasher, Zacks, and colleagues (Hamm & Hasher, 1992; Hasher, Stoltzfus, Zacks, & Rypma, 1991; Hasher & Zacks, 1988; Kane, Hasher, Stoltzfus, Zacks, & Connelly, 1994; May, Kane, & Hasher, 1995; Stoltzfus, Hasher, & Zacks, 1996; Stoltzfus, Hasher, Zacks, Ulivi, & Goldstein, 1993; Zacks & Hasher, 1994) have championed the theory that working memory deficits are not the true cause of age changes in performance. They are, instead, a consequence of age changes in the ability to inhibit irrelevant information. If irrelevant information cannot be inhibited, then interfering

thoughts displace the information in working memory that is germane to the problem at hand (Giambra, 1989; Rabbitt, 1965). The end result is that cognitive processes, in our case linguistic processes, suffer because capacity for the storage and manipulation of relevant material is reduced for older adults.

The efficiency of the inhibition mechanism has been measured through negative priming paradigms (Tipper, 1985, 1991; Tipper & Cranston, 1985; Tipper & Driver, 1988). On a particular trial, two overlapping stimuli (e.g., letters) are presented. The subject is instructed to say the stimulus presented in green and to ignore the stimulus presented in red. Response latency to name the green stimulus is recorded. The same procedure holds for each trial. The critical comparison is between two different cases for the second trial. In one case, the green stimulus on the second trial is the red (to-be-ignored) stimulus on the first trial (the ignored repetition condition). In the other case, the green stimulus on the second trial is a stimulus not seen before (control condition). If the subject has good inhibitory control, response latency on the ignored repetition trial will be slowed relative to the control trial. This slowing of response due to suppression of previously presented material is called negative priming. It is represented by the difference in response latency in the ignored repetition and control conditions. Hasher, Zacks, and colleagues (see references cited in previous paragraph) found that in many situations, older adults fail to produce negative priming effects or produce diminished negative priming effects (e.g., Earles et al., 1997; cf. Sullivan & Faust, 1993).

Although a reduction in inhibitory control is an attractive hypothetical mechanism for the explanation of language changes, there are two further requirements to test the plausibility of the concept. First, it is necessary to demonstrate that individual differences in inhibitory control map onto individual differences in working memory capacity. Second, it is necessary to demonstrate that individual differences in inhibitory control are predictive of individual differences in language processes (e.g., comprehension).

For the requirement that individual differences in inhibition map onto individual differences in working memory, Engle and colleagues have established this link (Engle, Conway, Tuholski, & Shisler, 1995). They argue that effective inhibition of irrelevant information is the consequence of efficient allocation of the attentional resources of the central executive in working memory (Baddeley, 1992), which they call the Inhibition-Resource Hypothesis (Engle, 1996). Thus, differences in the capacity of working memory are due to differences in controlled, attentional processes and are not due to differences in automatic activation processes (Engle, Cantor, & Carullo, 1992). Individuals with more attentional resources will have a

greater ability to inhibit irrelevant information and are less susceptible to interference effects.

As for the requirement that individual differences in inhibitory control predict individual differences in language processing, Gernsbacher and colleagues have demonstrated that facility in the suppression of irrelevant meanings of homographs (e.g., *bank*—river or money meanings) is predictive of language comprehension skills in good and poor young adult readers (Gernsbacher & Faust, 1991; Gernsbacher, Varner, & Faust, 1990). Finally, it would be preferable to have a demonstration that older adults show inhibitory processing deficiencies that also correspond to language comprehension problems. However, no studies to our knowledge have yet demonstrated that older individuals with poor inhibitory control also have poor language comprehension for sentences that place a high demand on working memory resources.

COGNITIVE SLOWING

Another resource explanation of cognitive aging that takes an even broader view of the mechanism underlying age-related changes for a wide variety of cognitive processes, including language, is the cognitive slowing view (Birren, 1965; Cerella, 1985, 1990; Hale, 1990; Kail, 1991; Lindenberger, Mayr, & Kliegl, 1993; Rabbitt, 1981; Salthouse, 1991, 1992a, 1992b, 1994a, 1994b). The details of the various versions of the cognitive slowing theories differ, but all claim that changes in cognitive performance are due to changes in the speed of carrying out basic cognitive operations.

The change in cognitive processing speed accounts for cognitive performance at both ends of the developmental continuum. Children are slower than persons in their 20s and 30s (Hail, 1990; Kail, 1991), and the cognitive processing rate slows progressively throughout the adult life span (e.g., Myerson & Hale, 1993; Myerson, Hale, Hirschman, Hansen, & Christiansen, 1989). The mathematical functions that describe the relations between response latencies in younger and older adults often account for more than 95% of the variance in the response latencies of older adults on a wide range of cognitive tasks. Both speeded and unspeeded cognitive performance are influenced by developmental changes in processing rate. This is demonstrated by one of the processing-speed models, the Information Loss Model (Myerson, Hale, Wagstaff, Poon, & Smith, 1990). The Information Loss Model assumes that all cognitive operations are made up of a number of steps; the same number of steps for both young and old. As a cognitive operation unfolds, each cognitive step takes a finite amount of time to complete. In old age, there is information lost at each step. The processing time for each step takes progressively longer for older adults as time on task increases due to the cumulative effect of information loss on

previous steps. Thus, the more steps a cognitive operation takes, the larger are the processing time differences between younger and older adults. The specific nature of the cognitive task being performed is irrelevant. The lawful relation between response latencies of young and old arises because of the number of steps that an operation takes. Because of the information loss, merely providing older adults with extra time to complete a cognitive task does not compensate for the cumulative information loss. In the Information Loss Model, the nature of the function that describes the relation of younger to older adults' latencies differs between tasks requiring the processing of language versus nonlanguage materials (Hale, Lima, & Myerson, 1991; B.M. Lawrence, Myerson, & Hale, 1997; Lima, Hale, & Myerson, 1991). The information loss rate for language tasks is less than for nonlanguage tasks. The division between language and nonlanguage domains conforms to the relative preservation of language functions with age.

Continuing debates in the realm of cognitive slowing include the issue of whether or not developmental change in processing speed can be explained by the aging of peripheral sensory processes such as vision and hearing, as argued by Lindenberger and Baltes (1994). Also at issue is whether or not slowing is truly general, that is, whether any task-specific factors are relevant (Tun & Wingfield, 1993). Specifically, we question whether or not cognitive slowing can explain the age-related differences in processing within the language domain that we reported earlier. For example, can cognitive slowing explain the finding of relatively large changes in lexical retrieval with age, but of relatively small changes in discourse?

Neurological Correlates of Age-Related Changes in Language

Although much is known about the neuroanatomy of language dysfunction in aphasia, surprisingly little is known about the age-linked changes in neuroanatomy that underlie the normal, age-related changes in language. Even less is known about the neurochemical changes that may explain deterioration of language function as people get older. In this section we attempt to expose relevant correlations between documented age-related neurological changes and the age-related changes in language that we have just described.

Characteristic age-related changes in brain anatomy include decreased brain weight, gyral atrophy, ventricular dilatation, and loss of myelin staining of fiber systems (T. L. Kemper, 1994). With regard to this last element, Peters and colleagues (1996) argue that loss of myelin, rather than loss of neurons, is the principal neuroanatomical change of normal aging.

Neuronal cell loss does occur, however, primarily in the hippocampus, amygdala, hypothalamus, brainstem nuclei, and cerebellum. Note that these neuronal changes primarily affect subcortical structures. Neuronal cell loss in the cerebral cortex is variously reported as decreased or unaffected (T. L. Kemper, 1994). Neurofibrillary tangles, however, are widely distributed and, for our purpose, are particularly prominent in the medial portions of the temporal lobes, regions important for memory and language. Age-related neuritic plaques favor frontal and parietal lobes (Heilbroner & Kemper, 1990), especially in regions of gyral atrophy (Sandor, Albert, Stafford, & Kemper, 1990). In normal aging, the major sites of cerebral cortical atrophy are the parasagittal gyri and the adjacent frontal and parietal lobes.

If we consider just these anatomical changes for the moment, and try to relate them to the cognitive changes underlying language dysfunction, we might speculate that what could be called "connectivity," rather than "content," is lost with normal aging. Within the widely distributed lexical and semantic networks that provide the basis of language, if "connectivity" is lost or reduced, it would be more difficult to access lexical items and access meaning. Thus we can propose the beginnings of a neuroanatomical explanation for the most frequently reported age-related changes in language: word finding difficulties and comprehension problems. More specifically within the zone of language, age-related loss of dendritic structure has been demonstrated in neurons of the posterior superior temporal gyrus, with more loss occurring in the left hemisphere than in the right (Anderson & Rutledge, 1996). These anatomical changes could further contribute to the problems of naming and word finding.

To these changes in brain structure we should add the specific changes that occur in the auditory system with aging. It is well known that deterioration of function occurs at all levels of the auditory system from peripheral to central (Olsho, Harkins, & Lenhardt, 1985; Wallace, Hayes, & Jerger, 1994). Thirty to 50% or more of elderly persons have significant hearing loss due to intrinsic aging of the peripheral and central auditory systems, compounded by noise damage. Because so much of normal language activity is dependent on auditory system function, to the extent that the auditory system is progressively impaired in normal aging, comprehension of spoken language will be affected.

Neurochemical changes of normal aging may further exaggerate the language changes. Dopamine receptor density declines as we get older (Wong, Young, Wilson, Meltzer, & Gjedde, 1997). Dopamine influences cognition by stimulating receptors in the prefrontal cortex and mesocortical dopaminergic system (Javoy-Agid & Agid, 1980; Luciana, Depue, Arbisi, & Leon, 1992). The cognitive effect of dopamine is seen in attentional

activity and executive system function (Mimura, Motoichiro, Kashima, & Albert, 1998).

To summarize this brief review, neuroanatomical changes in selected language-related subcortical and cortical areas, deterioration of function and structure in peripheral and central auditory system, and decline in dopaminergic activity are all features of the neurology of normal aging which, when combined, could produce the major age-related changes of language that we have described, in particular impaired lexical retrieval and impaired comprehension of spoken language.

Aging and Aphasia

Aphasia Type and Aging

Twenty years have passed since Obler and colleagues (Obler, Albert, Goodglass, & Benson, 1978) first published their report confirming the observation that people with Broca's aphasia tended, on average, to be significantly younger than the median age for aphasics and people with Wernicke's aphasia tended to be significantly older than the median. In subsequent years, studies from research laboratories across the world have reaffirmed this finding and have offered various theories to explain it. In the original report, several possible explanations were suggested, including (a) that elderly people were more likely to have strokes affecting posterior regions of the brain, (b) that changes in cognitive style with aging interacted with brain damage in a variety of locations to result in aphasia that is most like Wernicke's aphasia, and (c) that the neural substrate for language changed with age to result in more fluent aphasias in elderly people regardless of lesion location (Brown & Jaffee, 1975).

Explanations relating to neuroanatomy predominated at first. In a survey of 64 patients, Eslinger and Damasio (1981) reported that their patients with Broca's aphasia were significantly younger than patients with global aphasia and Wernicke's aphasia. They suggested that younger patients were more likely to have strokes affecting anterior regions of the left hemisphere, and elderly patients were more likely to have strokes affecting posterior regions. Kertesz and Sheppard (1981) found similar results in a study of 192 patients with aphasia. They went a step further in suggesting that the explanation might be related to different underlying etiologies rather than simply to different lesion locations, differentiating between (a) embolic strokes in frontal and frontal–parietal regions arising from cardiac and carotid disease, and (b) thrombotic strokes resulting from diseased posterior branches of the middle cerebral artery. This explanation was premised on the fact that the first type of stroke would be more prevalent

in younger people and the second type of stroke would be more common in elderly people. However, as researchers discovered later, this was not the case (Habib, Ali-Cherif, Poncet, & Salamon, 1987).

Brown and colleagues (Brown & Grober, 1983; Brown & Jaffee, 1975) suggested that age was a predictor of aphasia type because it reflected degree of lateralization for language. These authors hypothesized that across the life span there was a progressive regional specification within the language zone from a diffuse bilateral organization in younger years to a unilateral focal organization in later years. As evidence, they cited statistics that 95% of aphasias in subjects younger than age 30 are nonfluent, whereas only 45% of aphasias are nonfluent in subjects over age 60. This idea was supported in the research of Basso, Bracchi, Capitani, Laiacona, and Zanobio (1987), who reported that some older patients with anterior lesions had fluent aphasias, suggesting "an age dependent extension of the area the lesion of which causes fluent aphasia." The Brown et al. hypothesis was further buttressed by Roch Lecours (1995), who asserted that "aspects of the ontogenesis of the human brain and of language . . . may continue for as long as the brain is disease-free." This explanation is different from the lesion location theory of Eslinger and Damasio (1981). Indeed, Habib et al. (1987) further confirmed, in a study of 200 stroke patients, that locus of lesion did not vary according to age, so it was unlikely that age effects on lesion location could explain the findings.

In their 1987 study, Basso et al. also raised the issue of how age might interact with stroke survival rates to result in the observation that nonfluent aphasia patients were younger than fluent aphasia patients. Perhaps elderly people, due to age-related health factors, were not surviving strokes with large nonfluent-aphasia-producing lesions to the same extent as younger people. Thus, they concluded that what we were observing was, in effect, due to an inadvertent selection bias. Similarly, in a critical review of the hypotheses on the relation between aphasia type and age, Coppens (1991) concluded that selection bias and the effects of cognitive changes in aging were the most reasonable explanations of the data. For example, he regraphed data from some of the earlier studies and found that the increase in frequency of Wernicke's aphasia in elderly people could be due simply to a lower mortality rate.

Effect of Age at Onset on Recovery from Aphasia

Clinicians and researchers have long been interested in discovering how various demographic factors such as age might interact to affect recovery from aphasia. It is often assumed, for example, that younger people with aphasia would show better recovery from aphasia than older people. How-

ever given the "selection bias" explanation for the relation between aphasia type and aging suggested in the last section, one could also make the argument that younger people as a group might show worse recovery because of the nature of their strokes (i.e., more large frontal, frontal-parietal, or frontal-parietal-temporal strokes show up in younger people because of greater mortality from similar strokes in older people).

Recovery from aphasia is a complex process, one that is the result of several interacting factors, including initial severity of language impairment, premorbid level of language functioning, concomitant medical and cognitive disabilities, amount and type of treatment, and other factors such as age. When age is looked at as a predictor of recovery, some studies have found that older subjects do tend to recover less (Holland, Greenhouse, Fromm, & Swindell, 1989; Kertesz & McCabe, 1977; Pashek & Holland, 1988; Sands, Sarno, & Shankweiler, 1969; Vignolo, 1969). However, probably a greater number of studies have found that converse: that age had either no effect or only a minor effect on recovery from aphasia (Basso, 1992; Keenan & Brassell, 1974; Kenin & Swisher, 1972; Lendrem & Lincoln, 1985; Pedersen et al., 1995; Pickersgill & Lincoln, 1983; Sarno, 1980; Sarno & Levita, 1971; Wertz & Dronkers, 1990).

Making sense of these contradictions may require a more sophisticated model of analysis that takes into account the interaction of age with other factors. Some of the studies, for example, that found no effects of age on recovery overall, did report that age interacted with recovery in patients who had severe forms of aphasia. Thus, elderly subjects with severe aphasia tended to show less improvement than younger subjects with comparable levels of severity (Pashek & Holland, 1988; Pickersgill & Lincoln, 1983). Just what the mechanism might be for this effect, however, is unclear. We saw in the first section of this chapter that normal elderly people often demonstrated reduced lexical retrieval skills and reduced working memory capacity. These are key abilities that are prerequisites for a good response to many types of speech and language treatment. Therefore, we might expect age to affect recovery from aphasia indirectly because of its effects on linguistic functioning even in an intact brain.

Language in Dementia of the Alzheimer's Type

Investigations into the language disorder associated with Alzheimer's disease (AD) have mushroomed in the past 20 years. Clinicians who work with both aphasia patients and patients with AD are struck by the similarities and the differences between aphasic language disorders and the

linguistic impairments of patients with Alzheimer's disease. In this section, we review the major *explicit* language findings associated with Alzheimer's disease that are obvious from clinical examination and briefly discuss the language changes in Dementia with Lewy Bodies (DLB). We follow this with a discussion of the research literature investigating "semantic memory" in Alzheimer's dementia patients. Unlike the clinical literature, this research examines the extent to which *implicit* knowledge of language may be preserved in AD patients.

Although AD is thought to be the most common form of dementia, it is also well known that the presentation of AD is not uniform. Several subtypes of AD have been recognized, including subtypes that vary according to the relative prominence of certain initial symptoms (e.g., language disorders or visuospatial disorders) and subgroups that differ according to time of onset (i.e., early-onset or late onset). Some researchers have suggested that patients with early onset AD show faster rates of decline in language functioning than patients with later onset AD (Faber-Langendoen et al., 1988; Raskind, Carta, & Bravi, 1995). However, other researchers have failed to confirm this finding (Bayles, 1991a; Bayles, Tomoeda, & Trosset, 1993). Other subgroups of AD have also been studied, for example, those with accompanying disorders such as Parkinsonism. As a result of this variability in the presentation of the disease, the generalizability of the descriptions that follow is necessarily somewhat limited. The description of the language profiles should be viewed as a composite picture of what is commonly seen in AD; the symptoms seen in any one individual are likely to be different from those described here. Furthermore, because AD can only be definitively diagnosed postmortem, references to AD should be interpreted as references to probable AD (see McKhann et al., 1984).

Language Patterns Associated with AD

The clinical manifestation of the language disorder of AD varies according to the progression of the disease. Obler and Albert (1984) proposed a three-stage model to describe the progression of language symptoms often seen in patients with AD, from a mild or early-stage, to a moderate or mid-stage, and finally to a severe or late-stage. Bayles and Kaszniak (1987) also presented a comprehensive description of the language changes observed over the course of the disease in AD. In subsequent years, Bayles and colleagues published numerous reports on the language impairments associated with AD (Bayles, 1991a, 1991b; Bayles, Caffrey, Tomoeda, & Trosset, 1990; Bayles & Tomoeda, 1991; Bayles, Tomoeda, & Rein, 1996; Bayles, Tomoeda, & Trosset, 1992, 1993; Bayles & Trosset, 1992). Likewise, both Cummings and colleagues (e.g., Cummings, Benson, Hill, & Read,

1985; Cummings et al., 1988; Powell, Cummings, Hill & Benson, 1988) and Kertesz and colleagues (e.g., Appell, Kertesz, & Fisman, 1982; Kertesz & Clydesdale, 1994) have added much to the knowledge base about the language changes associated with AD.

MILD, EARLY-STAGE AD

During the early or mild stage, around the time of first diagnosis, AD patients are described as having mild word-finding problems and as using slightly empty, circumlocutory speech. However, their speech is fluent, well-articulated, normally prosodic, and grammatical. Occasional paraphasias are produced, but frank neologisms are rare at this stage. For example, in a study investigating oral narrative discourse in patients with either AD or fluent aphasia (Wernicke's aphasia, conduction aphasia, or anomic aphasia), Nicholas, Obler, Albert, and Helm-Estabrooks (1985) reported that the narrative descriptions of the patients with AD were most similar to the narratives of the anomic aphasics.

Auditory comprehension problems are found in testing, but appear to be secondary to attentional deficits at this stage. Oral reading is unimpaired, but reading comprehension deficits are evident upon testing (Schwartz, Marin, & Saffran, 1979). Writing shows evidence of preservation of syntax (S. Kemper, LaBarge, Ferraro, Cheung, & Storandt, 1993), but also reflects some of the word-finding problems and emptiness seen in spontaneous oral discourse (Bayles & Kaszniak, 1987).

Some studies of AD patients in the early stage of the disease have stressed the similarity of their language performance to that of normal elderly subjects (LaBarge, Balota, Storandt, & Smith, 1992). We recently reported no differences between subjects with probable AD and elderly controls on a measure of semantic relatedness of naming errors on the Boston Naming Test (BNT; Nicholas, Obler, Au, & Albert, 1996). In this study, error responses produced by younger and older normals and patients with mild and moderate AD were blindly rated for semantic "closeness" to the target word. For example, a response such as "concertina" for the target picture of an *accordion* received a high semantic relatedness score; a response such as "you have to put these on these fellows" for the target picture of a *yoke* received a low semantic relatedness score. Analysis of the relatedness scores indicated that there were no differences between the error responses of the elderly controls and the AD subjects on this measure, although the AD subjects had more naming errors overall and they also produced more no-response errors than normal elderly people.

Although most normal aging subjects do not perform in the range of the "average" subject with mild AD, there is overlap in naming performance in these two groups. For example, in our sample of subjects in the preced-

ing semantic relatedness study (Nicholas et al., 1996), 9 of the 12 subjects with probable mild AD received BNT scores above 45 (out of 60); over one-third of the 30 elderly control subjects performed in the same range ($\geq$45 and $\leq$54). This degree of overlap is even more impressive when we consider that all of the normal elderly subjects received scores above the cut-off score of 27 on the Mini Mental State Examination (Folstein, Folstein, & McHugh, 1975), thus ensuring that they were not demented.

Although there is overlap in the naming performance of some normal aging subject and those with mild AD, no one has claimed that mild naming failures in normal aging are due to impairment or deterioration of information in semantic memory. Instead, as we stated in the first part of this chapter, most authors suggest that the lexical retrieval failure in normal aging is due to breakdown or dysfunction in the transfer of information from a correctly accessed semantic representation to a phonological word form. In contrast, when considering the performance of AD patients, authors have suggested that deterioration of information in semantic memory underlies the naming impairment (Chertkow & Bub, 1990; Chertkow, Bub, & Seidenberg, 1989; Henderson, Mack, Freed, Kempler, & Andersen, 1990; Hodges, Salmon, & Butters, 1992; Huff, Corkin, & Growdon, 1986). We address this issue more fully in the section on semantic memory in AD.

Because of the overlap in language performances seen in some normal elderly subjects and patients with early AD, researchers have attempted to devise tasks that would clearly discriminate the two groups. For example, Monsch and colleagues (1992) showed that a category fluency task (generating a list of names within a given semantic category such as animals) was a good discriminative task, whereas letter fluency (generating a list of words all beginning with the same letter) was not. This finding may be reflective of the semantic memory deficits associated with AD.

Snowdon and colleagues (1996) reported intriguing findings from the Nun Study that relate performance on a writing task in early life to cognitive dysfunction and Alzheimer's disease in later life. One of the advantages of studying a group such as the School Sisters of Notre Dame is that subject selection and environmental factors can be more carefully controlled, resulting in a more homogeneous sample than in many longitudinal studies of normal aging. In this particular study, autobiographical texts written by the sisters upon first entering the convent (mean age 22 years) were examined for density of idea content and grammatical complexity. Cognitive functioning was assessed approximately 58 years later, and the brains of 25 participants who died were examined for Alzheimer pathology. Ten of the 25 nuns had neuropathologically confirmed Alzheimer's disease and 15 did not. Of the 10 participants with Alzheimer's disease, 9 had low idea density in their writing samples obtained in early life; whereas

only 2 of the remaining 15 sisters without Alzheimer pathology had low idea density scores. A similar pattern was not found for grammatical complexity, however. Snowdon and colleagues had hypothesized that sisters with low linguistic ability may have had less "neurocognitive reserve capacity," making them more vulnerable to the consequences of neuropathology in later life. However, because only one sister had Alzheimer's pathological changes and yet had not shown cognitive decline, the authors revised their hypothesis to suggest that low linguistic ability in early life may actually be an early expression of Alzheimer's pathology. This finding, of course, will require confirmation in future studies, but it highlights how studies of cognitive–linguistic abilities may be useful in differentiating normal from pathological changes.

MODERATE, MID-STAGE AD

In the mid-stage of Alzheimer's disease, changes in language performance become more marked and differences from normal elderly performance are obvious. The spontaneous speech of AD patients becomes profoundly anomic, circumlocutory, and tangential. Perseveration of individual phrases as well as of ideas (Tomoeda, Bayles, Trosset, Azuma, & McGeagh, 1996) become prominent. Memory impairment interacts with the linguistic disorder to result in frequent repetition of the same ideas and constant repeating of the same questions and/or requests of others. Impairments in auditory comprehension become apparent in conversational interchanges and written output resembles the patterns seen in spontaneous speech. However, in a study of agraphia in AD, LaBarge, Smith, Dick, and Storandt (1992) found that errors produced in a simple task of writing a sentence were *not* correlated with measures of aphasia or other psychometric measures of language performance. Instead, the authors suggested that writing skill may represent procedural memory and that errors in writing may be due to impairments in long-term memory in subjects with AD.

A popular theory to account for the disturbance in language functioning in AD is that a reduction in cholinergic activity in the brain linked to the pathophysiology of AD underlies the linguistic impairments (Arneric et al., 1995; Christensen, Maltby, Jorm, Creasey, & Broe, 1992; R.E. Davis, Doyle, Carroll, Emmerling, & Jaen, 1995; Eagger & Harvey, 1995; A.D. Lawrence & Sahakian, 1995; Poirier et al., 1995; Raffaele et al., 1996; Whitehouse, 1993). Christensen et al. (1992), for example, found that the pattern of memory and language deficits seen in mild AD resembled the pattern of deficits found in normals who were administered scopolamine as a cholinergic blocker. In support of this idea, researchers in Norway (Aarsland, Larsen, Reinvang, & Aasland, 1994) demonstrated dose-dependent

impairments on tasks of language, including spelling, verbal fluency, and object naming, in a group of healthy young women who were given injections of scopolamine. Research documenting improvement in language functioning in AD patients who have been treated with cholingeric medications such as tacrine has supported this idea, although generally effects have been modest and appear in fewer than half the patients treated (R.E. Davis et al., 1995; Eagger & Harvey, 1995; Poirier et al., 1995). However, Tanaka and colleagues (Tanaka, Minematsu, Hirano, Hayashida, & Yamaguchi, 1994) found that treatment with CDP-choline did *not* have a beneficial effect on language functioning in AD, although improvements were noted in patients with non-AD vascular dementia.

SEVERE, LATE-STAGE AD

During the late stage of AD, all language abilities show severe deficits, and many patients eventually stop talking and become mute. Language testing at this point is impossible, although Hier, Hagenlocker, and Shindler (1985) documented some preservation of repetition abilities in late-stage AD patients. This isolated preservation of repetition, sometimes more aptly identified as echolalia, is not unlike the phenomenon seen in cases of "isolation of the speech area," an aphasic disorder caused by extensive brain damage that results in significant language disorder but spares the auditory-input-to-speech-output loop necessary for repetition.

Language Patterns Associated with Dementia with Lewy Bodies

In recent years, a type of dementia known as dementia with Lewy bodies or DLB (McKeith et al., 1996) has been recognized as the second most common form of dementia following dementia of the Alzheimer type. Lewy bodies have been found in 15 to 25% of the brains of elderly demented subjects at autopsy, with areas of predilection including the brainstem, subcortical nuclei, limbic cortex, and neocortex (affecting temporal lobes more than frontal and parietal lobes, which are affected equally). Alzheimer pathology (plaques and neurofibrillary tangles) is often seen in addition to the Lewy bodies. DLB is characterized by progressive mental impairment with prominent attentional and visuospatial difficulties early in the course of the disease. McKeith et al. (1996) also highlighted as core features of DLB: (a) fluctuations in cognitive function, (b) persistent well-formed visual hallucinations, and (c) spontaneous motor features of Parkinsonism. Reportedly, in later stages of the disease, deficits in memory and language functioning resemble the deficits seen in AD (McKeith et al., 1996).

Detailed profiles of the neuropsychological and language impairments associated with DLB are only just beginning to be available (Kalra, Bergeron, & Lang, 1996; Perkins, Whitworth, & McKeith, 1996; Salmon & Galasko, 1996; Salmon et al., 1996). Salmon et al. (1996) compared neuropsychological test performances of patients with DLB (with little or no Alzheimer pathology) to the performances of patients with "pure" AD (no Lewy body pathology) in order to highlight the differences and/or similarities between these two forms of dementia. The patients were equivalent in terms of severity of dementia. Their results indicated that the patients with DLB had significant deficits in memory, language, attention, and executive functioning, with particularly severe deficits in visuospatial functioning, which were significantly worse than in the subjects with AD. The subjects with AD also displayed impairments across the board, but their memory functioning was worse than the patients with DLB. In other words, although memory and visuospatial functioning may be distinguished in these two groups, there was little in their language profiles to distinguish AD from DLB.

Perkins et al. (1996) reported on a retrospective analysis of case notes on 21 patients with DLB. From the limited language data that were available, comprehension and naming skills were mentioned as areas of preservation; syntactic errors and perseveration were described as contributors to incoherence of discourse production. This profile was not confirmed in the report of Salmon and Galasko (1996), who documented that naming abilities were impaired in DLB. Given that many patients with DLB also show Alzheimer pathology, and that many patients with AD also show Lewy body pathology, it may be quite difficult at the present time to isolate the effects on linguistic functioning that are attributable solely to either of these two types of brain pathologies.

Semantic Memory in AD

We saw in the earlier section that word-finding problems, incoherence of discourse, paraphasic errors, and difficulty with reading and writing have been repeatedly mentioned in the language profiles of patients with AD. Numerous studies have interpreted these difficulties in language processing as impairments arising primarily in the semantic memory system, caused by the brain pathology in the Alzheimer brain. Semantic memory (SM) is a term used for the long-term memory store in which conceptual information is represented, including semantic (meaning) and lexical (word) information, as well as facts about the world (Bayles & Kaszniak, 1987; Tulving, 1972). In making the claim that semantic memory shows impairment in AD, researchers have used as evidence the commonly ob-

served difficult in tasks such as naming pictures, defining semantic attributes of objects, designating superordinate categories of items, and the like (Abeysinghe, Bayles, & Trosset, 1990; Bayles & Kaszniak, 1987; Bayles, Tomoeda, & Trosset, 1990; Grober, Buschke, Kawas, & Fuld, 1985). A controversy has been raging for some years now, however, as to whether the problem in SM would be best characterized as a *loss of access* to semantic knowledge or as a *loss of the semantic knowledge representations* themselves. In this section we review this controversy by examining research in three areas: (a) naming, (b) off-line tasks of semantic knowledge, and (c) semantic priming.

NAMING IN AD

Anomia in spontaneous speech and impaired confrontation naming of pictures are two of the most prominent symptoms observed in most people with AD (Bayles & Tomoeda, 1991; Bayles & Trosset, 1992). However, the cognitive mechanism responsible for word-finding problems in AD is still not clear. As we saw in the first part of this chapter, most researchers have come to the conclusion that the word-finding problem associated with normal aging is not due to a loss of information in SM, but rather to an inaccessibility of the phonological label for a concept that was appropriately accessed. Is the problem in AD similar, differing only in a matter of degree, or is it qualitatively different, arising from a different source? Researchers attempting to tackle this question have not yet come up with a definitive answer.

At first, researchers wondered whether the difficulty in naming could be related to visuo–perceptual disorders (Cormier, Margison, & Fisk, 1991; Kirshner, Webb, & Kelly, 1984). Were people with AD failing to name items because they were misperceiving them? Most investigations into this question have concluded that misperceptions account for only a small proportion of the naming errors made by subjects with AD (Goldstein, Green, Presley, & Green, 1992; Hodges et al. 1992; Martin & Fedio, 1983; Shuttleworth & Huber, 1988; Smith, Murdoch, & Chenery, 1989). In a study from our laboratory, for example (Nicholas et al., 1996), we found that fewer than 20% of the error responses produced on the BNT (Kaplan, Goodglass, & Weintraub, 1983) by subjects with moderate AD could be interpreted as misperceptions of the target. Instead, the majority of naming error responses were semantically related to the target.

Errors described as semantically related can provide a wide range of semantic information about the target, from minimal ("an animal" for the target *beaver*) to fully descriptive ("an animal that builds dams and has a flat tail"). How to interpret these errors has been a matter of debate. Some authors have claimed that the first type of response is indicative of semantic

system disruption, reflecting loss of attribute knowledge and availability of only the most basic semantic information in subjects with AD (Bowles, Obler, & Albert, 1987; Hodges et al., 1992). Normal elderly people also give these types of responses, yet in their case, the error is not interpreted as a loss of semantic information, but rather as a lexical access problem. This dilemma in interpretation arising from the analysis of responses on naming tests has led researchers to develop other ways to assess the status of knowledge in semantic memory.

One avenue of investigation is to evaluate consistency of success or failure on tasks that require semantic processing. On a naming task, for example, if an item is named on one occasion, missed on another, and yet named on a third, we can confidently say that the "miss" was due to a momentary retrieval failure and not to a permanent loss of information. Consistency of naming performance across time in patients with AD has been the focus of several studies (Chertkow & Bub, 1990; Henderson et al., 1990; Huff, Mack, Mahlmann, & Greenberg, 1988). Both Chertkow and Bub (1990) and Henderson et al. (1990) found that performance was consistent across time, indicating a loss of semantic knowledge.

Huff et al. (1988) compared the performance of patients with anomic aphasia (from left hemisphere strokes) to the performance of patients with AD, who were matched for the severity of their naming impairment. Patients were given naming tests on three occasions. Both the aphasic group and the AD group showed consistency of naming performance across time, although the effect was stronger in the AD group. On subsequent tests of semantic knowledge tested in two modalities (verbal and nonverbal), both groups also showed impairments reflecting retrieval deficits. The authors concluded that *both* lexical access deficits and loss of information in semantic memory were operating to affect performance in both groups, but access impairments were more prominent in the aphasic group and loss impairments were more prominent in the AD group.

Laine, Vuorinen, and Rinne (1997) came to a similar conclusion in a study of patients with AD who were compared to a group with vascular dementia. Again, both groups showed a semantic component in their naming performance, but the semantic deficit was more obvious in the AD group. Corina and colleagues (Corina, Pressman, Kempler, Andersen, & Seidenberg, 1995) also found both retrieval deficits and loss of semantic knowledge in their group of 16 subjects with AD. Furthermore, these impairments appeared to fall along a continuum, related to the severity of dementia. Subjects who were still able to respond to phonemic cues ("cuers") on the naming task generally retained more semantic knowledge, assessed with property verification probes (e.g., "true or false, a knife is a type of fruit"), than subjects designated as "noncuers." Cuers were also able to

make semantic judgments about items they were not able to name, whereas noncuers were less able to do this. Thus it appears that both characterizations of the naming impairment in AD may be correct. Retrieval deficits and loss of semantic knowledge both contribute to the naming disorder in AD. The reader may recall that a similar conclusion was reached in the discussion of naming impairments in normal aging, although the balance may be different in that population than in one with AD.

SEMANTIC KNOWLEDGE IN AD

Once it became clear that semantic memory showed impairment in AD, research efforts began to be directed at characterizing the nature of the semantic problem in more detail. A prevalent theory is that the nature of the deterioration is a so-called bottom-up process. This means that semantic information, such as knowledge of attributes (perceptual features, functional features, etc.), may be more vulnerable to loss than information at the "top" of the semantic network for a concept (e.g., superordinate categorical information; Flicker, Ferris, Crook, & Bartus, 1987; Hodges et al., 1992; Martin & Fedio, 1983; Schwartz et al., 1979). As we discussed earlier, when subjects misnamed items by saying their superordinate category labels, these error responses were often interpreted as consistent with the bottom-up deterioration theory. However, studies that have attempted to examine this question more directly have produced mixed results.

Grober et al. (1985) found that subjects with AD were accurately able to select attributes for concepts, but they were not able to rank attributes in terms of their importance to the concept. Thus, the contents of semantic memory appeared not to be lost, but rather to be disorganized in some fashion. Nebes and Brady (1988) found similar results in a study of attribute knowledge in patients with AD. Subjects were required to indicate whether a given attribute presented as a written word was related to a picture. Although the subjects with AD were much slower at responding than the normal elderly controls, they were able to make these judgments accurately for all types of attributes assessed, including superordinate category labels, related action–function words, words describing physical features, and general associates. Nebes and Brady concluded that the field of semantic attributes surrounding a concept was not diminished in AD. As additional evidence confirming this finding, Nebes and Halligan (1996) demonstrated that subjects with AD showed normal activation of semantic attributes by sentence context primes.

Abeysinghe et al. (1990) found that subjects with AD were able to define accurately words that they could not provide meaningful associates for, calling into question the interpretation of a lack of associates as necessari-

ly reflective of semantic memory deterioration. Abeysinghe et al. emphasized that some of the findings of semantic memory impairment in AD may reflect task difficulty effects more than true semantic memory impairments. This has been the theme of several additional studies from the laboratory of Bayles and colleagues (Bayles, Tomoeda, Kaszniak, & Trosset, 1991; Bayles, Tomoeda, & Trosset, 1990; Cox, Bayles, & Trosset, 1996). For example, Bayles et al. (1991) tested a large group of subjects with AD ($n = 69$) on a wide variety of tasks using the same 13 concepts in each task. A subset of patients was also assessed longitudinally. This study was premised on the hypothesis that if semantic information about a given concept were truly lost, then subjects should perform poorly on all tasks dependent on processing of that information. Instead, the results provided little indication of any convincing loss of information. Although patients often performed poorly on many tasks, there was no instance where all tasks assessing knowledge of a given concept were failed.

SEMANTIC PRIMING IN AD

Naming tasks and semantic probe tasks such as those used in the studies mentioned earlier are examples of "off-line" tasks of language processing. These tasks probe for explicit knowledge and require controlled, conscious processing. As such, they are subject to the influences of processing in other domains such as memory and attention, domains that are virtually always adversely affected in AD. Therefore it is difficult to determine the extent to which poor performance on these tasks of explicit knowledge is related to purely linguistic impairments.

Because performance on these off-line measures has been difficult to interpret in patients with AD, researchers have turned to alternative methodologies such as automatic priming to evaluate SM more directly. In a standard associative or semantic priming task, normal subjects respond faster to a target word that follows a semantically or associatively related prime word than they do to a target that follows a neutral or unrelated word (Meyer & Schvaneveldt, 1971). This result is found for tasks of lexical decision (indicating whether the target is a real word or not) and pronunciation (reading the target words aloud).

Priming studies with AD patients have produced conflicting results. A variety of priming effects have been found in patients with AD, including patterns of normal priming (Nebes, Martin, & Horn, 1984; Ober, Shenaut, Jagust, & Stillman, 1991), lack of a priming effect or less than normal priming (Albert & Milberg, 1989; Ober & Shenaut, 1988), and hyperpriming (exaggerated priming; Chertkow, Bub, Bergman, Merling, & Rothfleisch, 1994; Chertkow et al., 1989; Nebes, Brady, & Huff, 1989). When normal

priming effects are demonstrated in AD, authors have interpreted their results as indicating that information in SM is intact (yet perhaps inaccessible for conscious use; e.g., Nebes et al., 1984, 1989). Conversely, when priming effects are absent or are excessive, the results are interpreted as reflecting deterioration of SM (Chertkow et al., 1989, 1994). Even within a single study, subgroups of patients with AD have been identified who show abnormal patterns of priming, whereas other patients show the normal pattern (Albert & Milberg, 1989). Furthermore, there is some suggestion that patients with more severe dementia are more likely to show abnormal priming effects, such as hyperpriming. For example, Chertkow et al. (1989) found that patients were more likely to show hyperpriming on targets that had been determined to be "degraded" in SM based on these patients' performance on "off-line" measures of semantic knowledge, similar to those discussed in the last section.

The type of semantic relation between the prime and target has also been shown to affect priming in both normals (Moss, Ostrin, Tyler, & Marslen-Wilson, 1995) and subjects with dementia (Moss, Tyler, Hodges, & Patterson, 1995; Ober et al., 1991). Most studies of semantic priming in AD have used words that are associatively related, that is, chosen from word-association norms. It has been suggested, however, that an associative relation may lead to priming from activation at a presemantic, intralexical level (Glosser & Friedman, 1991). Thus, as associative priming effect that looks "normal" may be found in a patient whose semantic memory system is nevertheless impaired. Nebes (1994) addressed this criticism in a reexamination of data from his earlier studies using sentence primes (Nebes, Boller, & Holland, 1986; Nebes & Brady, 1991). Items were removed from the analysis that could be construed as containing simple intralexical associations. In this new analysis, however, Nebes still found that normal sentence priming was evident in subjects with AD.

How are we to make sense of the conflicting results seen both in tasks of explicit knowledge and in on-line semantic priming tasks? Milberg and colleagues (Grande, McGlinchey-Berroth, Milberg, & D'Esposito, 1996; Milberg, 1998) have presented a novel theory that attempts to explain a wide variety of findings in the literature on semantic deficits in AD by proposing a "gain–decay hypothesis." According to this hypothesis, patients with AD suffer from a "demodulation" of neural activation. Because of the AD brain pathology, dying cells in an excitotoxic state are intermingled with live cells reacting appropriately. In effect, activation levels get turned up higher, much as the "gain" on an electronic device could be adjusted upward. For example, in semantic priming tasks, activations for highly salient information get turned up and priming is excessive for these; at the same time, the activation curves for weaker associates get dragged

below the threshold and no priming is seen for these. Although the details of this hypothesis are still in development, it is an intriguing theory that seems biologically plausible.

Conclusion

Language changes associated with healthy aging are seen most clearly in the areas of lexical access and comprehension, with some subtle differences in discourse structure as well. These findings may be explained—at least in part—by difficulties with working memory, reduced ability to inhibit irrelevant information, and slowing of cognitive processing. At the brain level, they may be correlated with neuroanatomical changes in cortical and subcortical structures, progressive damage to peripheral and central auditory systems, and reductions in levels of dopaminergic activity.

When aphasia occurs in an older adult, we are more likely to see fluent rather than nonfluent forms, perhaps due to progressive changes in the brain localization for language or to differential morbidity from lesions producing fluent and nonfluent aphasias. Although there is no unequivocal evidence that recovery from aphasia becomes less successful with advancing age, there are hints that age or attendant problems may cause a subtle diminution of expected recovery.

Language changes associated with Alzheimer's disease, and perhaps, with Dementia with Lewy bodies as well, bear some resemblance to those of two populations discussed earlier: healthy elderly individuals, especially with regard to lexical access problems, and patients with fluent aphasia, in this case with special regard to comprehension problems.

In the case of Alzheimer's disease, increasing emptiness of speech with sparing of syntax further mimics fluent aphasia. Deterioration of semantic memory per se and impaired access to semantic memory appear to account for a substantial part of the language decline in Alzheimer's disease. Moreover, the cognitive processing deficits associated with healthy aging, such as memory problems, disinhibition, and cognitive slowing, are likely exacerbated in the dementias and thus contribute as well to the language decline.

Acknowledgments

This work was supported by a Veterans Administration Merit Review Grant and by National Institutes of Health Grant AG-14345 to Martin Albert and Loraine Obler, and the Boston University Aphasia Research Center, DC-00081.

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13

Acquired Aphasia in Children

DOROTHY M. ARAM

Introduction

Children versus Adults

Language disruptions secondary to acquired central nervous system (CNS) lesions differ between children and adults in multiple respects. Chief among these differences are the developmental stage of language acquisition at the time of insult and the developmental stage of the CNS.

In adult aphasia premorbid mastery of language is assumed, at least to the level of the aphasic's intellectual ability and educational opportunities. Acquired aphasia sustained in childhood, however, interferes with the developmental process of language learning and disrupts those aspects of language already mastered. The investigator and clinician thus are faced with sorting which aspects of language have been lost or impaired from those yet to emerge, potentially in an altered manner. Complicating research and clinical practice in this area is the need to account continually for the developmental stage of that aspect of language under consideration for each child. In research, stage-appropriate language tasks must be selected, and comparison must be made to peers of comparable age and language stage. Also, appropriate controls common in adult studies, such as social class and gender, are critical. These requirements present no small challenge, as most studies involve a wide age range of children and adolescents. In clinical practice, the question is whether assessment tools used for developmental language disorders should be used or whether adult aphasia batteries should be adapted for children. The answer typically depends on the age of the child and the availability of age- and stage-appro-

priate measures. With childhood acquired aphasias, the question of how language is altered following a CNS insult is inextricably related to how language learning proceeds.

The second major difference between adults and children sustaining language-disrupting neurological insults is the stage of maturity of the CNS at the time of insult. The controversy relating to the degree of early hemispheric specialization versus equipotentiality for language (e.g., Kinsbourne & Hiscock, 1977; Lenneberg, 1967) has sparked much of the research in this area during the past 20 years. Although most of the data appear to evidence considerable early brain specialization for language and other higher cognitive functions (Best, 1988; Molfese & Segalowitz, 1988), the remarkable capacity of young children to recover from major cortical insults has been repeatedly reported. That children recover much more rapidly and completely from focal brain lesions than do adult with comparable insults has become a truism that is generally, although not universally, supported. The rapidity and level of language recovery in young brain-injured children evidence a degree of functional and presumed neural plasticity far exceeding that in adults (Aram & Eisele, 1992). Most (Bates et al., in press; Eisele & Aram, 1993; Lees, 1993; Vargha-Khadem, Isaacs, van der Werf, Robb, & Wilson, 1992) but not all (Martins & Ferro, 1993; Paquier & Van Dongen, 1993) investigators in this field conclude that the pattern of language loss following lesions in children generally do not correspond to what is observed in adults with similar injuries. Rather, as Bates et al. (1997) recently have suggested, the data thus far suggest that "innate regional biases in style of information processing lead to familiar patterns of brain organization for language under normal conditions and permit alternative patterns to emerge in children with focal brain injury" (abstract). An immature CNS at the time of insult does not necessarily lead, however, to a more favorable outcome. In some instances—for example, conditions resulting in diffuse brain involvement—a brain insult incurred at a young age may be more, rather than less, deleterious, a topic returned to later in this chapter. The essential concern here is that just as the child's language system is in the process of development, so too is the central nervous system, processes that are complete or declining in the adult. Although in many instances the immaturity of the child's CNS supports alternatives for greater functional recovery, at other times early insults appear to interfere with primary skills, thus precluding later achievements.

Terminology

ACQUIRED VERSUS DEVELOPMENTAL APHASIA

The term ACQUIRED is used to modify the term APHASIA in children to distinguish language disorders accompanied by known CNS insults from the

much more common form of DEVELOPMENTAL APHASIA. Developmental aphasia, also referred to as developmental language disorders or specific language impairment, is manifest when a child fails to learn to talk normally, but a frank neurological basis is not apparent. Indeed, the presence of a frank neurological abnormality typically is established as an exclusionary criterion for the diagnosis of developmental aphasia or developmental language disorder (Benton, 1964; Tallal, 1988). Although many researchers assume abnormal neurological functions give rise to the developmental aphasias (e.g., Rapin & Allen, 1988), the search for identifiable brain lesions to account for these developmental disorders has been largely unproductive. Also, the language symptomatology that follows acquired unilateral lesions typically has been found to be less severe and less persistent than is often seen in children with developmental language disorders (Aram & Eisele, 1994). This chapter addresses the language abilities only in children with known brain insults, that is, those with acquired aphasia.

ACQUIRED LANGUAGE LOSS VERSUS ACQUIRED BRAIN LESION

According to the literature on acquired aphasia in children, even though the lesion is generally acquired—through stroke, tumor, trauma, or some other form of CNS insult—the language loss is not necessarily "acquired," as frequently the insult occurs before much language has been acquired and therefore not much can be "lost." Few studies have been confined to older children with relatively well developed language at the time of insult; rather most have involved a wide age range at lesion onset, typically extending from pre- or perinatal insults to those incurred during adolescence. Indeed, many studies compare outcome as a function of whether the lesion was sustained prior to 1 year of age (the somewhat arbitrary demarcation for the onset of language) or after 1 year of age, with a broad range of onset ages included in the latter group. Therefore, acquired aphasia, as used here, refers to language discrepancies or abnormalities accompanied by a known brain lesion, irrespective of when during the course of language development that lesion occurred.

Nature of the Studies Available

Although a more homogeneous age of lesion onset would be desirable in reports of acquired aphasia in children, age is only one of several markedly heterogeneous variables complicating interpretation of the majority of studies addressing this topic. The major methodological problems confounding review of work in this area have been reviewed elsewhere (Aram & Whitaker, 1988). Five variables, typically noncomparable both within and across studies, are of particular note:

1. AGE OF LESION ONSET was discussed earlier and generally involves a broad spectrum of ages.

2. The NATURE OF THE NEUROLOGICAL INSULTS described in studies are typically diverse, and often include such disparate conditions as tumors, head trauma, herpes encephalitis, and cerebral vascular accidents in a single study. This chapter draws not only from studies specifically addressing acquired CNS insults and aphasias in children, but also from studies of infantile or childhood hemiplegias, as the overlap of children included in these studies is considerable. In an effort to reduce the wide diversity of conditions discussed here, this chapter does not address the hemispherectomy or epileptic aphasia literature except when an occasional hemispherectomized or epileptic aphasic child has been included in a group study involving heterogeneous causes.

3. EXTENSIVENESS OF BRAIN INVOLVEMENT also varies widely from circumscribed focal lesions to diffuse white and gray matter involvement. Often specification of the degree of actual brain involvement is lacking and can only be inferred from the etiology or clinical pattern. This topic is discussed more fully later in this chapter.

4. AGE AT FOLLOW-UP varies both with respect to chronological age and with respect to time elapsed since lesion onset. A few studies have described language during the acute period of recovery, although most have assessed language status years after lesion onset.

5. The METHOD OF EVALUATION used in various studies has differed. Until the past 15 years, most statements pertaining to language ability were based on nonsystematic clinical observations or on verbal intelligence scores. More recently, most studies have begun to report standardized language tests, and a few have begun to include a more experimental or hypothesis-testing approach to the study of acquired aphasia in children.

Because very few experimentally rich or methodologically sound studies of relatively homogeneous groups of lesioned children exist, the present review is not restricted to those few studies. Rather, an attempt has been made to cull from the diverse studies available and to interpret contradictory findings in light of the differences among subjects and methodologies.

The Clinical Picture: Language Characteristics

Comprehension

Despite pronouncements that receptive disorders are rare or that beyond the acute period comprehension disorders disappear rapidly and vir-

tually completely (Hécaen, 1976, 1983), until recently few studies provided objective data substantiating or refuting these claims. Guttmann (1942) appears to be one of the few early observers noting long-standing receptive as well as expressive deficits following temporal lobe lesions in children. Alajouanine and Lhermitte (1965), although commenting that receptive disorders were rare, reported that 4 of 32 children with acquired aphasia presented marked comprehension disorders. Several case studies have detailed the recovery of comprehension abilities during the acute period, usually (Aram, Rose, Rekate, & Whitaker, 1983; Ferro, Martins, Pinto, & Castro-Caldas, 1982; Martins, Ferro, & Trindade, 1987; Pohl, 1979), but not always (Dennis, 1980; Oelschlaeger & Scarborough, 1976), demonstrating complete or relatively good recovery of comprehension skills. Except for the exemplary work of Dennis and her colleagues with young hemispherectomy patients, prior to the past 15 years there appear to be no studies other than IQ results in which findings are detailed relative to comprehension abilities among children with acquired aphasia. The studies available have focused predominantly on syntactic and lexical comprehension.

SYNTACTIC COMPREHENSION

Dennis (1980) provided a comprehensive study of the acute language status of a 9-year-old girl with a left temporoparietal infarct at 2 weeks and at 3 months after lesion onset. Drawing from an array of standardized and experimental tasks, Dennis (1980) concluded that, although improvement had been observed, at 3 months after lesion onset the child's comprehension of longer, nonredundant oral commands continued to be impaired, and lower level syntactic structures were better preserved than were more complex structures involving supraordinate schemata such as embeddings. On a metalinguistic judgment task in which the interrelatedness of words was assessed, the child seemed to adopt a simplified surface and linear processing strategy for complex utterances. Although still relatively early in recovery, this case study demonstrated significant disruption in all aspects of language, including comprehension, expression, and communicative intent; unfortunately, language status after 3 months was not reported. Other case studies have reported notable syntactic comprehension deficits acutely but with relatively good recovery within the first several months after lesion onset (Aram et al., 1983; Ferro et al., 1982; Pohl, 1979).

Several group studies of children with brain lesions, studied well beyond the acute period, have assessed syntactic comprehension and generally found subtle yet persistent comprehension deficits. Findings between studies vary somewhat, presumably reflecting differences in subject variables, notably the nature and diffuseness of the lesion, concomitant seizure

disorders, and overall intellectual level. Levine, Huttenlocher, Banich, and Duda (1987) found that well after lesion onset, all four groups of children with left, right, congenital, and acquired hemiplegias performed below average on the Northwestern Syntax Screening Test (Lee, 1969); it should be noted, however, that these children's intelligence was below average and half had ongoing seizure disorders. In contrast, Kiessling, Denckla, and Carlton (1983), studying groups of left- or right-hemiplegic children selected because they were functioning well in school, found a significant correlation between right-hand function on the Annett pegboard (used as a measure of left-hemisphere function) and performance on a syntactic awareness task, thus evidencing poorer performance following left-hemisphere injury. However, in a more recent study of comprehension and imitation of complex coordinate, passive, and relative clause structures examined in a group of left-lesioned children and a group of right-lesioned children in comparison to normal controls, left-lesioned children's comprehension was relatively preserved despite significantly impaired imitation (Eisele & Aram, 1994). Right-lesioned subjects' impairment was less pronounced in both comprehension and imitation compared with the left-lesioned group.

Token Test (de Renzi & Vignolo, 1962) results have been reported by several groups of investigators, with findings that apparently reflect the variable subject groups under test. Woods and Carey (1979) reported that left-lesioned subjects differed from controls on the Token Test if lesion onset occurred after but not before 1 year of age. Vargha-Khadem, O'Gorman, and Watters (1985) reported that irrespective of age of onset, their three left-lesioned subject groups (prenatal, early postnatal, and late postnatal) but not the three respective right-lesioned groups performed more poorly than control subjects. Similarly, Aram and Ekelman (1987) found performance of left- but not right-lesioned subjects to be significantly lower than that of controls, and identified no relationship between age of lesion onset and revised Token Test (McNeil & Prescott, 1978) performance among lesioned subjects. Variable performance depending on the Token Test subtest has been noted. Left-hemisphere–lesioned children have been reported to have particular difficulty with subtests that assess syntactic components (Aram & Ekelman, 1987; Cooper & Flowers, 1987; Riva, Cazzaniga, Pantaleoni, Milani, & Fedrizzi, 1986) or that tax verbal memory (Aram & Ekelman, 1987; Rankin, Aram, & Horwitz, 1981), whereas right-hemisphere–lesioned subjects have been reported to do more poorly on items requiring spatial skills (Aram & Ekelman, 1987; Riva et al., 1986). Van Dongen and Loonen (1977), studying a group of acquired aphasic children with mixed etiologies, reported that comprehension deficits on the Token Test during the acute stage were associated with a poor prognosis for recovery.

In summary, the data suggest that syntactic comprehension often is impaired following left brain involvement. Poor performance by right-hemisphere–lesioned subjects appears to be related to spatial demands of the task and/or to more generalized brain involvement. Beyond the acute period, most but not all syntactic comprehension deficits are mild. Poor syntactic comprehension in the acute period appears to be related to a poor prognosis for recovery. Age of lesion onset has not been found to have a consistent relationship to syntactic comprehension.

LEXICAL COMPREHENSION

Until recently, except for a few detailed case studies, lexical comprehension skills among children with acquired aphasia have been studied little beyond the administration of the Peabody Picture Vocabulary Test (PPVT, Dunn, 1965). In one case study of a 10-year-old girl who sustained diffuse traumatic brain involvement after a fall from a horse, Oelschlaeger and Scarborough (1976) documented significant limitations in lexical comprehension 1 year after the trauma was sustained. Most group reports do not indicate deficits as pronounced as that observed by Oelschaeger and Scarborough (1976), but do evidence mild long-standing deficits in lexical comprehension. Using the PPVT, Aram, Ekelman, Rose, and Whitaker (1985), Cooper and Flowers (1987), Levine et al. (1987), and Riva et al. (1986) all reported lower performance by the lesioned than the control subjects. Although Kiessling et al. (1983) reported a correlation between right-hand function and PPVT performance, most studies (Aram et al., 1985; Levine et al., 1987; Riva et al., 1986) have failed to find a lateralized left-hemisphere effect on PPVT performance. In fact, Eisele and Aram (1993) found that although both right- and left-lesioned subjects scored more poorly than controls on the PPVT-R, the right-lesioned children presented the greatest impairments, leading these investigators to suggest that the right hemisphere may have a specialized role in mediating the acquisition of word meaning. Similarly, Bates and her colleagues (1997), using a parent informant inventory, the MacArthur Communicative Development Inventories (Fenson et al., 1993), also found children with right lesions between 10 and 17 months of age to present greater delays in word comprehension than children with left-hemisphere lesions, contradicting what would be hypothesized from the adult literature.

Finally, one of the few studies (Cooper & Flowers, 1987) to examine lexical comprehension beyond single-word representation assessed meaning in context with the Processing Spoken Paragraphs subtest of the Clinical Evaluation of Language Functions (Semel-Mintz & Wiig, 1982). On this test, significant deficits among the group of chronic brain-injured children were reported. However, because of the diffuse nature of brain involve-

ment for most of the subjects, no attempt was made to relate findings to laterality of brain involvement.

In summary, based on studies predominantly assessing single-word comprehension, at least mild lexical deficits have been noted in most groups of children with acquired aphasia. Lexical comprehension deficits generally have been found in children with either left or right hemisphere damage, and recent studies suggest greater impairment in the latter group of children.

Language Production

Language production is the most extensively studied aspect of acquired childhood aphasia. Although descriptions tend to center on reduced verbal output, descriptions of more fluent-type aphasia and paraphasic behaviors have been reported. Some limited work has also addressed lexical retrieval and phonological production.

REDUCED VERBAL PRODUCTION

Most early reports of acquired aphasia focused on the diminished speech output and telegraphic speech thought to characterize acquired aphasia in children (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Early descriptions emphasized the striking feature of reduced verbal output, ranging from mutism to a reluctance to speak, and stated that syntax was simplified rather than erroneous (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Equally impressive to many early observers was the rapid recovery of expressive abilities, the absence of fluent Wernicke-type aphasias, and the infrequency of paraphasias (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Although fluent aphasias with neologistic, semantic, and phonemic paraphasias have since been described (see the next section), reduced output continues to be regarded as the dominant feature of acquired aphasia in children (Cranberg, Filley, Hart, & Alexander, 1987; Martins & Ferro, 1987, 1993).

Woods and Carey (1979) provided what appears to be the first experimental study of productive syntax in left-hemisphere–lesioned patients. By using a series of syntactic production tasks, including identifying and correcting anomalous sentences with "that" clauses, ask–tell distinctions, and the sentence completion task from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983), they found that left-lesioned childhood aphasics, sustaining lesions after but not before 1 year of age, differed significantly from control subjects.

Most subsequent studies of children with acquired lesions have found

syntactic limitations to be associated predominantly with left but not right lesions. For example, both Kiessling et al. (1983), in a study of hemiplegic children, and Riva et al. (1986), in a study of acquired lesions of diverse etiologies, found left-hemisphere–lesioned children to perform more poorly than right-lesioned or control subjects on sentence repetition tasks. Similarly, Carlsson et al. (1994) found right-hemiplegic (left hemispheric involvement) children to be more impaired than left-hemiplegic children on several verbal tasks. Also, in an analysis of the spontaneous conversational language of left- and right-lesioned children studied well beyond the acute period, Aram, Ekelman, and Whitaker (1986) found left- but not right-lesioned children to perform less well on a range of measures of simple and complex sentence structure. In a more recent study assessing imitation of complex coordinate, passive, and relative clause structures, Eisele and Aram (1994) again found the residual deficits in syntactic ability to be more pronounced as a result of early left in comparison to right brain lesions. This study also compared imitative abilities to comprehension of the same structures, demonstrating a marked dissociation of comprehension and production of syntax among the left-lesioned children.

Significant deficits in syntactic production rarely are reported following right lesions except in cases thought to represent crossed aphasia (Martins et al., 1987). An exception has been a study that used a speech shadowing task (Woods, 1987) in which sentences were to be repeated in correct and reversed word order. On this task both left- and right-lesioned subjects performed more poorly than controls, despite the right-lesioned subjects' normal performance on other language measures. Woods (1987) explained his findings by suggesting that the shadowing task tapped skills dependent on global cerebral function, rather than on more narrowly defined language functions.

Thus it appears that reduced output and simplified syntax are the typical presentation following left-hemisphere lesions. In addition, syntactic deficits may persist for years.

FLUENT VERBAL PRODUCTION

Many of the early descriptions of acquired aphasia in children stated that fluent aphasias did not occur or were extremely rare in children younger than 10 years of age (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976). The absence of fluent aphasia in young children was ascribed to an underdeveloped or underautomatized Broca's area controlling expressive language which was incapable of "running on" in the absence of appropriate input from posterior language areas. More recently, however, several examples of fluent aphasias with jargon or logorrhea have been described in young children. Woods and Teuber (1978) appear

to be one of the first to have described jargon aphasia, defined as jabbering away with unintelligible sounds as though understood, in a single 5-year-old among 65 children studied. Additional case studies of fluent aphasias have been reported by Martins and Ferro (1993), Van Hout and Lyon (1986), and Van Dongen, Loonen, and Van Dongen (1985). These studies detailed young children with logorrheic speech, a high proportion of neologisms, and significant comprehension disorders. Van Dongen et al.'s (1985) three cases also presented a phrase length of at least seven words, a speaking rate of more than 90 words per minute, and normal prosody, articulation, pauses, and effort. These patients also exhibited frequent paraphasias.

PARAPHASIAS

Just as fluent aphasia was thought to be rare or absent among childhood acquired aphasics, paraphasias also were believed to be exceptional (Alajouanine & Lhermitte, 1965; Hécaen, 1976). Their occurrence, however, has been described among children with reduced verbal productions, particularly among the few with more fluent verbal production. Paraphasias appear to be more common during the acute period following acquired brain lesions, and have been described as especially notable and persistent among children with diffuse brain involvement as opposed to more focal or lateralized brain lesions. Visch-Brink and Sandt-Koenderman (1984) and Van Dongen and Visch-Brink (1988) have provided detailed descriptions of neologisms, literal paraphasias, and verbal paraphasias in spontaneous speech and naming tasks in first two and then six children who were 5 years and older at the age of insult. In addition, they presented a single case of phonemic jargon aphasia. These case studies exemplify the early appearance of neologisms and paraphasias after lesion onset and their rapid disappearance, usually within 2 or 3 weeks of lesion onset, at least among head-injured patients.

Van Hout, Evrard, and Lyon (1985) also summarized the verbal and paraphasic errors among 11 children with acquired aphasia. They divided their patients into three groups according to the evolution of their paraphasias, which also coincided with the severity of associated problems. For Groups I and II, the paraphasias resolved in a matter of days and over a few months, respectively; for Group III, the paraphasias persisted for more than a year. Particularly notable among Van Hout's patients was the severity of associated problems in all but Group I and the diffuse nature of brain involvement in all patients. Three of the four patients in Group III, the most severely impaired group, had herpes encephalitis, and the fourth incurred cerebral trauma followed by a Stage III coma for 1 month with decerebrate posturing. All had significant associated problems and appeared to be

grossly demented. Although paraphasic errors may occur in the acute period following acquired focal lesions in young children with either reduced or fluent verbal outputs, in general, their persistence appears to be associated with more pervasive cognitive disorders as a consequence of diffuse brain involvement.

NAMING AND LEXICAL RETRIEVAL

Although a few studies (Van Dongen & Visch-Brink, 1988) have described paraphasic errors on naming tasks, the majority of studies addressing naming and lexical retrieval have simply recorded the correctness of response. Most have reported reduced naming abilities among children with left- but not right-brain lesions. For example, Hécaen (1976, 1983) reported that 44% of left-lesioned but none of the right-lesioned children had naming disorders that he described as being impoverished. Similarly, Van Dongen and Visch-Brink (1988) reported no naming problems in spontaneous speech or on naming tasks among right-lesioned children. Among those with left lesions, they differentiated between children with head injury who demonstrated a successive decrease in neologisms and recovery of all naming errors by 6 months of age, and non–head-injured children (cerebrovascular accident, subdural empyema, encephalitis) who presented more severe and persistent aphasic symptoms with irregular distribution of types of naming errors during recovery. Yet Eisele and Aram (1993) reported that left-lesioned children performed comparably to controls on the Expressive One-Word Picture Vocabulary Test (Gardner, 1979). Riva et al. (1986) and Thal et al. (1991) found deficits among right- as well as left-lesioned subjects, although both Thal et al. (1991) and Bates et al. (1997) found deficits to be the greatest after insult to the left posterior hemisphere.

Aram, Ekelman, and Whitaker (1987) administered the Word Finding Test (Wiegel-Crump & Dennis, 1984) and the Rapid Automatized Naming Test (RAN; Denckla & Rudel, 1976) to left-lesioned, right-lesioned, and control subjects. On the Word Finding Test, left-lesioned subjects were slower than other subjects in latency of response when given semantic or visual cues, and they made more errors when given rhyming cues. On the RAN, left-lesioned subjects were significantly slower than controls in naming all semantic categories (colors, numbers, objects, and letters). In contrast, right-lesioned subjects responded as quickly as or more quickly than control subjects in all access and semantic category conditions, yet produced more errors than controls, suggesting a speed–accuracy trade-off. The overwhelming error type was "no response," and few error types among the lesioned subjects differed appreciably from those of controls. Lesioned subjects, however, were assessed at least 1 year and often sever-

al years after lesion onset and thus the assessments did not capture any naming errors that may have occurred acutely.

Age of lesion onset has been equivocally related to naming deficits among left-lesioned subjects. Woods and Carey (1979) found left-lesioned subjects who sustained lesions after but not before 1 year of age to be impaired on naming tasks. However, Vargha-Khadem et al. (1985) found left-lesioned subjects, irrespective of the age when the lesion was sustained (congenital, early, or late acquired), to perform more poorly than right-lesioned subjects on the Oldfield–Wingfield Object Naming Task (Oldfield & Wingfield, 1964). Similarly, Aram et al. (1987) found no relationship between age of lesion onset and lexical retrieval abilities among either left- or right-lesioned children.

In summary, it appears that naming and lexical retrieval deficits are common after left, but also may be observed after right hemisphere lesions and for the most part they do not appear to be related to age of lesion onset. Paraphasic errors have been described especially in the acute period after lesion onset. Although paraphasic errors may persist for some aphasic children, they do not appear to be a common feature later in recovery.

PHONOLOGICAL AND ARTICULATORY PRODUCTION

Aside from the few case reports of phonemic paraphasias (Van Hout et al., 1985; Visch-Brink & Sandt-Koenderman, 1984), very little detail has been offered relative to phonological production or articulatory abilities among children with acquired aphasia. No consensus appears regarding the occurrence of phonological and articulatory problems. Early studies suggested that articulatory disturbances were common. Alajouanine and Lhermitte (1965) stated that if the lesion occurred before 10 years of age, disorders of articulation were always present; these disorders were described as a phonetic disintegration no different from those observed in adults, if the stage of development was taken into account. Hécaen (1976) suggested that articulatory disturbances occurred after either left or right hemisphere lesions and were frequent in children younger than 10 years of age at lesion onset. Hécaen (1983) also reported that articulatory problems occurred 81% of the time after left anterior lesions versus 20% after left posterior lesions. Such a high occurrence of articulatory disorders, as suggested by Hécaen, has not been substantiated in most recent reports or reported after the acute period. For example, Cranberg et al. (1987) reported that only one in eight children with acquired aphasia exhibited dysarthria at follow-up; Dennis (1980) noted dramatic improvement in articulation by 3 months following lesion onset in her 9-year-old child with a left posterior infarct; and Kershner and King (1974) found articulation er-

rors to be no more common among left- or right-hemiplegic children than among controls.

The only detailed study of early phonological ability in children with focal lesions is that of Marchman, Miller, and Bates (1991), who reported a longitudinal study of the phonology observed in babbling and in first words. These investigators noted a small proportion of "true" consonants for the infants with focal injury, along with idiosyncratic patterns of consonant development. In contrast to Hécaen (1983), Marchman et al. (1991) reported most improvement among children with anterior as opposed to posterior brain damage. The limited work on productive phonology is inconclusive. Aram et al. (1987) reported that in response to rhyming (phonemic) cues, left- but not right-lesioned children made more errors in word retrieval than did control children; however, Woods and Carey (1979) reported that left-lesioned children did not differ from controls on a task requiring rhyming and completing nursery rhymes.

Finally, Trauner, Ballantyne, Friedland, and Chase (1996) report what appears to be the only study of affective and linguistic prosody in children after focal brain damage. In this study both right- and left-hemisphere–lesioned groups were deficient on tasks involving expression of affective prosody and linguistic prosody, whereas only right-hemisphere–lesioned children were impaired on tasks of affective comprehension, suggesting no clear brain lateralization for prosody early in development.

In summary, very little systematic investigation of phonology or articulation has been pursued among children with acquired aphasia; the data are not sufficient to generalize as to conditions that may relate to the presence or absence of phonological and articulatory problems.

Reading

Despite relatively good recovery for spoken language, long-term reading and writing problems are often, although not always, reported to persist. Alajouanine and Lhermitte (1965) reported that none of their 32 children with acquired aphasia were able to follow normal progress at school; although they were able to regain what they had previously learned, they had difficulty learning new information. Eighteen of the 32 experienced persistent reading problems, 9 were totally unable to read; 5 had a severe alexia for letters, syllables, and words; and 4 had alexia for letters and somewhat better reading of words. Similarly, both Cranberg et al. (1987) and Cooper and Flowers (1987) found sizable numbers of their acquired aphasic children to have long-term word recognition or reading comprehension deficits.

Although reading usually is reported as more impaired than spoken language among children with acquired aphasia, this is not always the case at least acutely. For example, Dennis (1980) reported that at 2 weeks after lesion onset for a 9-year-old child, reading was higher than oral language, at 3 months, reading but not oral language was age-appropriate. This case demonstrated that, even among children with acquired aphasia, a dissociation between auditory and reading comprehension may exist. Hécaen (1976, 1983) is one of the few to suggest that, although reading problems may occur in the acute period, especially following left-hemisphere insults, reading problems usually disappear rapidly and completely.

Findings relating reading deficits to lesion laterality and age of onset are somewhat equivocal. Several investigators have compared reading in children with predominantly left or right hemisphere lesions. Although some researchers (Kershner & King, 1974; Reed & Reitan, 1969) reported no difference between left or right congenitally hemiplegic children on the Wide Range Achievement Test (WRAT; Jastak & Jastak, 1978), a test of single-word recognition, others found WRAT word recognition to be related to adequacy of right-hand performance, reflecting left hemisphere functioning (Kiessling et al., 1983). Vargha-Khadem, Frith, O'Gorman, and Watters (1983) reported that children with either left or right lesions tended to have more difficulty than control subjects on measures of reading speed and reading comprehension, whereas those with left-hemisphere lesions acquired postnatally were most impaired in their reading skills. Aram, Gillespie, and Yamashita (1990) reported few significant mean group differences between left-lesioned children and their controls or between right-lesioned children and their controls on a battery of phonetic analysis, word recognition, and reading comprehension tests, although mean performance of the lesioned subjects was consistently below that of controls. However, notable individual differences were present within the lesioned subject groups, with 5 of 20 left-lesioned and 2 of 10 right-lesioned children presenting marked reading deficits, in contrast to only 1 of 30 control subjects. Age of lesion onset was not found to differentiate those with and without reading problems, although a family history for reading disorders or involvement of specific subcortical structures was present for all subjects with reading problems.

Overall, it appears that long-standing reading problems involving phonetic analysis, decoding, and comprehension may occur for a sizable proportion of children with acquired aphasia. Although reading problems have been reported following a variety of acquired lesions, they appear to be most common among the children with postnatally acquired left lesions.

Writing and Spelling

Although it has been suggested that written language skills are particularly impaired among children with acquired aphasia, there appear to be no detailed reports of these children's writing ability. Alajouanine and Lhermitte (1965) reported that written language was always more severely disturbed than oral language. Among their 32 children with acquired aphasia, severe alterations in writing were noted in 19, 8 of whom could only copy words and 5 were said to be "dysorthographic" in their spontaneous writing. Even Hécaen (1976, 1983), who considered oral language and reading problems to disappear "rapidly and completely," noted that writing problems among children with acquired aphasia tended to persist and may even become permanent. Beyond a few case study examples in which writing skills were only one aspect described (e.g., Dennis, 1980; Ferro et al., 1982), apparently no studies have explored the nature of these written language impairments.

Spelling deficits, reported to be relatively common among children with acquired aphasia, also have not been described extensively. Cranberg et al. (1987) and Cooper and Flowers (1987) reported spelling problems in 3 of 8 and in 8 of 15, respectively, of their children with acquired aphasia. Woods and Carey (1979) reported that children with left-hemisphere lesions before as well as after 1 year of age were significantly poorer than controls in spelling a series of eight words. Vargha-Khadem et al. (1983) also found that children with left-hemisphere lesions performed more poorly on spelling tasks than those with right lesions or control subjects, especially children with postnatally acquired left lesions. This group had notable difficulty with infrequently occurring words; findings for frequently occurring words were less clear-cut. Vargha-Khadem et al. (1983) appear to be among the few who have provided qualitative data describing the spelling errors made by aphasic children, which were categorized as morphophonemic, orthographic, or preservation of the sound frame.

Thus, despite the observation that writing and spelling problems are relatively frequent and persistent among children with acquired aphasia, especially in those with postnatally sustained left-hemisphere lesions, the problems presented have not been detailed.

Summary of Language Characteristics

Although typically less severe than the deficits observed among adults with acquired aphasia, a range of language deficits have been described in both the acute period and in the long term among children with a variety

of acquired brain lesions. Syntactic comprehension disorders, although more pronounced in the acute phase, have been shown to persist long-term following predominantly left-hemisphere lesions. Lexical comprehension deficits also tend to persist, and may be especially pronounced after right hemisphere lesions. Reduced, syntactically simplified language output is the most commonly described expressive language characteristic observed among children with acquired aphasia, particularly subsequent to predominantly left hemisphere involvement. Paraphasias and more fluent-type aphasias, however, have been described usually during the acute phase of recovery or following diffuse brain involvement. Impaired naming and lexical retrieval exemplified by paraphasias, impoverished vocabularies, and slow rate of retrieval have been reported both in the acute and long-term periods, especially after left-hemisphere lesions. Data addressing phonological production and articulation are equivocal, with insufficient detail available to resolve the contradictory reports. Reading, writing, and spelling deficits are the most frequent, persistent, and significant sequelae among children with acquired aphasia. Reading deficits involving phonetic analysis, word recognition, and reading comprehension have been described, typically in association with left-hemisphere lesions. Writing and spelling limitations also often appear to be long-standing problems, predominantly associated with left-hemisphere lesions, yet data detailing either limitation are sparse.

Factors Related to Recovery of Language Abilities

Clearly, recovery of language skills among children with acquired lesions is variable, both within and across studies. The primary factors thought to be associated with how well a child recovers involve the nature of the neurological insult and the age of the child at lesion onset.

Nature of the Neurological Insult

The nature of the neurological insult appears to account for much of the variability in outcome reported for children with acquired aphasias. Several aspects relating to the neurological insult are examined here: the degree of brain involvement, the etiology of the lesion, lesion laterality (i.e., involvement of the left or right hemisphere), the specific site of lesion (i.e., the actual structures involved within a hemisphere), the size or extensiveness of the lesion within a hemisphere, and the presence and severity of an accompanying seizure disorder.

DEGREE OF BRAIN INVOLVEMENT

Unlike most of the adult aphasia literature, the majority of studies addressing acquired aphasia in children are not confined to focal, more circumscribed lesions. Rather, most have included at least some children with known or presumed diffuse brain involvement, for example, secondary to asphyxia, head trauma, infectious processes, or cranial radiation and/or chemotherapy for the treatment of tumors. Often, however, the degree of more generalized involvement is not addressed; instead, lesions are treated as if the effect were confined solely to a focal area, for example, the location of a subdural hematoma after head trauma, or of a tumor with no mention of cranial radiation or chemotherapy. When results in these studies vary from findings in more circumscribed lesions, the findings need to be interpreted in light of some degree of probable diffuse brain involvement.

The few studies that have explicitly contrasted the effects of diffuse versus focal brain involvement on children's cognitive abilities, have consistently identified bilateral and/or diffuse hemispheric involvement as a poor prognostic sign (Hécaen, 1976; Janowsky & Nass, 1987; Loonen & Van Dongen, 1990; Van Hout et al., 1985). For example, as noted earlier, most of the children with severe comprehension disorders and persistent paraphasias (Van Hout et al., 1985) typically incurred diffuse brain involvement. Annett (1973) apparently was one of the first to study the relationship between the degree of more diffuse brain involvement and higher cognitive functions, including language. She reported an association between a decline in intelligence scores and increased impairment of the nonhemiplegic hand, thus demonstrating involvement of both hemispheres, not merely the more apparent side of hemiplegia. More recently, Loonen and Van Dongen (1990) reported an inverse relationship between recovery of spontaneous language and auditory comprehension skills and the degree of bilateral brain involvement. In general, then, it appears that lesions involving focal areas of the brain are associated with better recovery than are lesions involving diffuse areas of the brain.

ETIOLOGY

Closely related to the degree of brain involvement is the etiology of the brain lesion. In a review of 47 cases of acquired aphasia, Martins and Ferro (1987) reported that the prognosis for vascular and traumatic lesions was better than for encephalitis and tumors. Similarly, Van Hout et al. (1985) found a high incidence of infections, in particular herpes encephalitis, among her patients with the most severe and persistent language deficits. Many investigators have documented the deleterious effects on

higher cognitive function of cranial radiation and chemotherapy for CNS tumors in children owing to the diffuse effects of these treatments (e.g., Fletcher & Copeland, 1988). Guttmann (1942), Van Dongen and Loonen (1977), and Van Dongen and Visch-Brink (1988) suggested that children with aphasic symptoms recover more rapidly from head trauma than from vascular lesions; however, one might question the severity of their patients' head trauma, as the severity of head trauma is thought to be the single most important variable in determining recovery (Fletcher & Levin, 1988). When considering prognosis as it relates to etiology, severity of the injury or disease process must be considered, as well as the concomitant treatment. Thus, in general, etiologies implicating more diffuse brain involvement, including infectious processes, tumors treated with cranial radiation therapy and chemotherapy, and severe head injury are related to poorer outcome than more focal lesions, for example, as a result of vascular problems. Even within groups of children with focal lesions, however, outcome is variable. In these cases, the additional variables of lesion laterality, size, site, and presence of seizures need to be considered.

LESION LATERALITY

Woods and Teuber (1978) reported what is probably the landmark paper addressing lesion laterality and acquired aphasia in children. These investigators noted that since the introduction of antibiotics and mass immunization programs in the 1930s and 1940s, stemming previously common forms of diffuse brain involvement in children, the incidence of aphasia arising from right-hemisphere lesions is no higher than that reported in adults. Excluding earlier studies in which reports of diffuse brain involvement were frequent, these investigators reported that the incidence of aphasia associated with right-hemisphere lesions was less than 10%. If left-handers were excluded, the incidence dropped to 5%. Others have since substantiated that the incidence of aphasia after left lesions is comparable for right-handed children and adults, and the risk is substantially greater after left than right lesions at any age (Carter, Hohenegger, & Satz, 1982; Satz & Bullard-Bates, 1981). Such data prompted Hécaen to change his view regarding the lack of language lateralization in children and state, "One could reasonably conclude, therefore, that studies of acquired aphasia in children support the notion of early cerebral lateralization and even innate cerebral organization for the presentation of language" (1983, p. 586). In this chapter, deficits in syntactic comprehension, syntactic production, and naming and lexical retrieval were all associated with left- and not right-hemisphere lesions. Thus it appears that when lesions in children are confined to one hemisphere, just as in adults, aphasia is associated pre-

dominantly with left hemisphere involvement. Yet, as detailed earlier, right-hemisphere lesions may also have a subtle impact on many aspects of language, in particular, on lexical comprehension where deficits have been more marked after right-hemisphere than left-hemisphere lesions.

SITE OF LESION WITHIN A HEMISPHERE

Unlike most studies of adult aphasics, until very recently few studies with children provided sufficient evidence of lesion localization. In the past 15 years, studies have begun reporting computerized tomographic (CT) scan or magnetic resonance imaging (MRI) data for the majority of subjects, yet even these reports fail to define adequately the involvement of subcortical areas or to address actual areas of brain dysfunction. Nonetheless, some preliminary attempts toward localization have been offered.

Before the availability of CT scans and other noninvasive radiological evidence, findings were based on an array of laboratory (e.g., pneumocephalograms, arteriograms, surgery) and clinical (e.g., sensory and motor abnormalities) findings, with little consensus among reports. For example, Guttmann (1942) suggested that posterior left lesions resulted in more pronounced language deficits, whereas Hécaen (1983) concluded the converse, that anterior lesions produced more severe language deficits than did posterior lesions. The availability of CT scan data has not appreciably clarified the relationship between lesion location and language symptomatology. For example, Cranberg et al. (1987) found nonfluent aphasias to occur following either anterior or posterior lesions, and Visch-Brink and Sandt-Koenderman (1984) were unable to identify a relationship between lesion location as determined by CT scans and the occurrence of paraphasias. Similarly, in our series of studies assessing aspects of language among children with unilateral brain lesions (reviewed in Aram, 1988), we have been unable to identify involvement of localized cortical areas associated with specific language symptomatology beyond a slight trend for somewhat greater deficits following posterior left lesions, although this finding may interact with lesion size. In a series of studies (Aram, Ekelman, & Gillespie, 1989; Aram et al., 1990), however, we presented evidence of particularly pronounced language deficits for a small group of children with involvement of specific subcortical nuclei (the head of the caudate and the putamen) and the adjacent white matter tracts, suggesting that children with involvement of these structures may not recover as well as those with involvement of other portions of the left hemisphere. Whether these findings will be substantiated in work with larger groups of children remains to be seen.

LESION SIZE WITHIN A HEMISPHERE

Several studies have quantified lesion size based on CT scans and have attempted to draw relationships between lesion size and language sequelae, but with contradictory results. Several have suggested that the larger the lesion, the poorer the cognitive performance (Cohen & Duffner, 1981; Levine et al., 1987; Riva et al., 1986), whereas others have found little relationship between lesion size and recovery (Loonen & Van Dongen, 1990; Thal et al., 1991; Vargha-Khadem et al., 1985). Some investigators have suggested that lesion size may be important in older but not in younger age groups (Kornhuber, Bechinger, Jung, & Sauer, 1985). At this point the role lesion size plays in determining recovery is not clear, although it may be that size interacts with site and possibly age of onset.

PRESENCE OF SEIZURES

Most studies demonstrate poorer outcomes when seizures accompany acquired brain injury than when seizures are not present (e.g., Aicardi, Amsili, & Chevrie, 1969; Annett, 1973; Solomon, Hilal, Gold, & Carter, 1970; Van Dongen & Loonen, 1977). Levine et al. (1987) appear to be among the few whose findings do not support such a relationship. These investigators reported that IQ deficits correlated with the presence of electroencephalographic (EEG) abnormalities; however, when lesion size was entered as a covariate, the relationship between IQ and EEG abnormalities no longer maintained, suggesting that lesion size rather than EEG abnormalities was a more powerful predictor of cognitive recovery.

Most clinicians and investigators, however, present evidence to the contrary, viewing the presence of seizures as a negative factor affecting outcome. Isaacs, Christie, Vargha-Khadem, and Mishkin (1996) reported that brain-lesioned children with a history of seizures were impaired on measures of dichotic listening, manual functions, and IQ, whereas those with no seizures were impaired only on manual functions. As well, Vargha-Khadem (1993) found that children with lateralized brain lesions with an accompanying seizure disorder were more impaired on an array of language and memory tasks than were lesioned children without seizures. Finally, Sussova, Seidl, and Faber (1990) found hemiparetic children with an accompanying seizure disorder to have lower IQs than those without seizures. Taken as a whole, there is a sizeable body of data demonstrating that the concomitant presence of seizure disorders appears to have an adverse effect on cognitive and language outcome, which most probably can be related to the spread of abnormal electrical activity, thus implicating more diffuse brain involvement than the more circumscribed effects of the original lesion. Furthermore, as pointed out by Isaacs et al. (1996), the pres-

ence of seizures also apparently interferes with compensatory mechanisms.

Although the overall prognosis for language recovery in children with seizures in general is poorer for children with seizures than without, one also needs to take into account the frequency, severity, and type of seizures, as well as the effectiveness, dosage, duration, and number of anticonvulsants used.

Age of Insult

Because children with acquired aphasia usually recover more rapidly and completely than adults, age is assumed to be an important factor in determining outcome. The presumed plasticity possible in the immature brain has typically been identified as the mechanism responsible for age-dependent recovery (e.g., Lenneberg, 1967). Also, much of the earlier work with animals suggested better recovery of functions following early rather than later lesions (Kennard, 1936), a position often referred to as the "Kennard principle."

Evidence from children with acquired aphasia, however, is highly contradictory, and no single relationship between age at lesion onset and outcome is supported. Some researchers have suggested that lesions sustained before the onset of language do not have as significant an effect on language as those sustained after 1 year of age (e.g., Woods & Carey, 1979). Others have suggested that earlier lesions have more pervasive effects on development of higher cognitive functions than do later lesions (Basser, 1962). Most studies have failed to identify a clear relationship between age of lesion onset and recovery (Aram, 1988; Hécaen, 1976; Loonen & Van Dongen, 1990; Woods & Teuber, 1978). Some have suggested that the variable effect of age on outcome may be explained by other factors, for example, different etiologies among the age groups (Martins & Ferro, 1987). Finally, some studies suggest that the importance of age at lesion onset is not for predicting the rapidity or completeness of recovery, but for understanding the specificity of the lesion's effect, dependent on the stage of language development at the time. For example, Alajouanine and Lhermitte (1965) contrasted language characteristics of children with lesions before and after 10 years of age, stating that reduced verbal expression, disordered articulation, and comprehension deficits are characteristic of the effects of lesions before 10 years of age, whereas paraphasias and written language disturbances are common when lesions are sustained after 10 years of age.

Thus, it does not appear that a single relationship between age of lesion onset and prognosis for language recovery holds; rather, age may interact

with other variables, such as etiology, and may exert a variable effect on language depending on the stage at which language is disrupted.

How Language Recovers

Although considerable theorizing has been offered to explain language recovery among children with acquired aphasia, how and where language recovers are still largely speculative issues. Several mechanisms have been suggested, such as FUNCTIONAL SUBSTITUTION of "uncommitted" portions of the same or the opposite hemisphere, and REDUNDANT NEURAL REPRESENTATIONS that make possible the release of existing but previously suppressed pathways (see Aram & Eisele, 1992, for a review of mechanisms proposed for recovery). Much of the evidence for functional reorganization following brain lesions has been derived from work with animals. The limited data available pertaining to reorganization of language functions among children with acquired aphasia has addressed only which hemisphere continues to be active during language tasks as opposed to where within a hemisphere language functions occur. The few studies available on individuals with early lateralized lesions have used dichotic listening, sodium amytal, or electrophysiological procedures.

Evidence from Dichotic Listening Tests

Several investigators have used dichotic listening tasks to infer hemispheric laterality following early lateralized lesions. The dichotic paradigm presents different stimuli independently but simultaneously to each ear; the hemisphere contralateral to the ear through which the higher score of accurate recognition is obtained is considered to be dominant for that aspect of language (Berlin & McNeil, 1976). By presenting two-digit dichotic pairs to children and adolescents with lateralized seizures and hemiplegia, Goodglass (1967) found a dramatic inferiority of report from the ear opposite the injured hemisphere in most cases, with several instances of total suppression. Distinguishing between a "cerebral dominance effect," which usually favored one ear by only a small difference, and "a lesion effect," where difference ranged up to 100%, Goodglass suggested a parallel between lateralized suppression of auditory input and visual or tactile neglect, extinction, and displacement.

Subsequently, several other investigators have reported similar findings of inferiority of verbal recognition contralateral to the lesioned hemisphere and instances of total auditory suppression (Ferro et al., 1982; Isaccs et al., 1996; Martins et al., 1987; Pohl, 1979; Yeni-Komshian, 1977). Both Pohl (1979)

and Yeni-Komshian (1977) administered dichotic tests during the course of recovery and related dichotic findings to language improvement. Pohl (1979) studied a 6-year-old boy with a left middle cerebral artery occlusion at 8 months and again at 13 months after lesion onset. Under dichotic testing conditions, total right-ear extinction was found at both times and was not modified through verbal training. At 13 months, however, the right-ear extinction disappeared if words were presented monaurally to the right ear with white noise presented on the left. Pohl interpreted the right-ear extinction as signaling a switch in hemisphere dominance for speech from the left to the right hemisphere. Similarly, Yeni-Komshian (1977) described dichotic findings and language skills over time for four children with acquired brain damage. The three children with bilateral brain involvement all showed marked right-ear advantage initially and were unable to process competing stimuli, although they regained some capacity to do so over time. A relationship between the degree of language loss and the ability to process two competing stimuli was noted. Yeni-Komshian interpreted the pronounced right-ear advantage, which coincided with significant recovery of language, as an indication that language was originally represented in the left hemisphere and that recovery also took place in the damaged left hemisphere. In contrast, her fourth child, an 11-year-old boy with a total destruction of the left hemisphere, persisted in demonstrating a marked left ear advantage, and, despite intensive therapy, a severe aphasia remained at 14 months following lesion onset. Yeni-Komshian proposed that the strong left-ear advantage was suggestive that language recovery, although impaired, was taking place in the right hemisphere. More recently, Isaacs et al. (1996), in a large-scale study of 62 children with left hemisphere injury and 53 with right injury reported a left-ear disadvantage for dichotic digits for 60% of children with left congenitally acquired lesions compared with 26% of control children, suggesting that for these children language representation had shifted to the right hemisphere.

Finally, Nass, Sadler, and Sidtis (1992) studied dichotic speech and complex pitch discrimination in children with congenital left or right hemisphere lesions. In contrast to adults with similar lesions, neither left- nor right-lesioned congenital groups were significantly impaired on speech discrimination, although both performed more poorly than controls in complex pitch discrimination. Nass et al. interpreted their findings as evidence for the relative sparing of function following congenitally acquired lesions. This also was evidence for the earlier maturation of the left hemisphere, rendering it less capable of assuming functions from a damaged right hemisphere, and for "crowding" of functions transferred to the right hemisphere, thus accounting for the proper performance of both groups on complex pitch discrimination.

These studies demonstrate the utility of dichotic listening tasks in providing one approach for identifying which hemisphere assumes language functions following acquired lesions.

Evidence from Sodium Amytal Studies

Probably the strongest evidence of hemispheric dominance for language comes from sodium amytal studies, also referred to as the "Wada procedure" after the neurologist who developed the technique. This technique uses a short-acting barbiturate injected into either the left or the right internal carotid artery, which is repeated on a separate occasion in the alternate carotid artery. During the short period in which the drug circulates through the hemisphere, functions normally sustained by that hemisphere are significantly impaired, thus permitting determination of that hemisphere's role in a specific function. Because of the invasive nature of the technique, sodium amytal studies are usually restricted to preliminary assessment before surgical resection of the brain to relieve intractable seizure disorders to determine the effect of the surgery on language and memory functions. Thus, the patients for whom these data are available consist predominantly of persons with severe and often long-standing seizure disorders; nonetheless, for these patients a direct indication of hemisphere dominance for language can be obtained.

Rasmussen and Milner (1977) provided one of the most extensive summaries of sodium amytal findings as they pertain to lateralization of language functions following left hemisphere lesions sustained early in life. In a review of 134 patients in whom the epileptogenic lesions all occurred before 6 years of age, and in most instances from the prenatal period, the following data were reported. First, 81% of their left-lesioned patients who remained right-handed were also left hemisphere dominant for speech, suggesting that an early left-hemisphere lesion that does not modify hand preference is unlikely to change hemispheric dominance for language. In contrast, among the non–right-handed subjects with left-hemisphere lesions, 53% had right-hemisphere language representation and 19% had evidence of bilateral representation. Second, Rasmussen and Milner reported that speech could be mediated asymmetrically in the two hemispheres, with the anterior speech areas in one hemisphere and the posterior areas in the other hemisphere. Third, even gross lesions that did not involve the primary speech zone (the inferior frontal and posterior temporoparietal regions of the left hemisphere) rarely altered speech lateralization, whereas damage to either of these critical areas usually resulted in right or bilateral speech representation. Finally, they speculated that after 5 years of age recovery is achieved by intrahemispheric reorganization rather than by a

shift of hemispheric dominance, which suggested that upward displacement of the posterior speech zone to include more of the parietal cortex may provide such a compensatory mechanism.

In a more recent review, Mateer and Dodrill (1983) likewise found that left-hemisphere lesions involving the inferior frontal and posterior temporoparietal regions usually resulted in either right-hemisphere or, more rarely, bilateral hemispheric representation for language. In addition, they reported that for their group of patients with bilateral speech representation, the early brain injuries appeared to be diffuse and not lateralized to a single hemisphere. However, they pointed out that all instances of bilateral damage did not necessarily result in bilateral language representation. Mateer and Dodrill suggested that early diffuse injury may either actively inhibit language lateralization or possibly require contributions from both hemispheres for the support of language development.

In general, the data from sodium amytal studies do provide evidence for right or bilateral representation of language functions for some left-lesioned subjects, particularly when the patient is left-handed, when the primary speech zones are involved, and when injury occurs at a young age. Yet these data are derived from a small subgroup of individuals with intractable seizure disorders who require an invasive procedure for determining lateralization. Whether or not these findings generalize to other groups of brain-injured children is unclear.

Evidence from Electrophysiological Findings

Electrophysiological procedures such as those involving auditory evoked potential provide a noninvasive means of determining brain activity in response to language stimuli. As of yet, however, it appears that few investigators have applied these techniques to the study of brain organization for language of children with acquired aphasia. Papanicolaou, DiScenna, Gillespie, and Aram (1990) reported the use of the probe evoked potential paradigm (Papanicolaou & Johnstone, 1984) with a group of children with unilateral left lesions in the absence of seizure disorders. The left-lesioned children in this study displayed the normal pattern of predominantly left hemisphere engagement in a language task and right hemisphere engagement in a visuospatial task. Thus, among this group of left-lesioned subjects it appeared that language restitution and development involved intra- rather than interhemispheric functional reorganization. That 9 of the 14 children in this study were left-handed at the time of study is not consistent with Rasmussen and Milner's (1977) findings reported with the sodium amytal procedure; however, differences may relate to the fact that in Papanicolaou et al.'s patients, lesions were focal and

not accompanied by seizures, compared with Rasmussen and Milner's patients who had intractable seizure disorders. Also, in the evoked potential study, the language tasks involved only a phonological target detection task, signaled by raising the index fingers, whereas the tasks used in Rasmussen and Milner's sodium amytal study involved naming and sequential speech. Thus the task used in the evoked potential study did not require any language production and may not have tapped more anterior speech areas. Therefore, the Papanicolaou et al. findings cannot preclude the possibility, suggested by Rasmussen and Milner (1977), that speech functions among early lesioned subjects may be mediated asymmetrically by the hemispheres. Had a language production task been included, it is possible that results for that task may have been different from those for the phonological detection task.

Clearly, much remains to be learned about how language recovers among children with acquired aphasia. The few studies available suggest that both intra- and interhemispheric reorganizations occur. With the application of noninvasive techniques, such as evoked potentials, and more dynamic imaging techniques, such as positron emission topography and functional magnetic resonance imaging, greater understanding of factors related to the process of recovery from acquired aphasia in childhood hopefully will be forthcoming.

Acknowledgment

The studies by the author reported in this chapter were supported by National Institutes of Health Grant NS17366.

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14

Aphasia after Traumatic Brain Injury

HARVEY S. LEVIN and SANDRA BOND CHAPMAN

Introduction

In recent years, a major paradigm shift has occurred in the way that aphasia in traumatic brain injury (TBI) is considered. Previously, there was a bias toward defining the communicative impairments in both adult and pediatric populations with TBI within the narrow context of specific language disturbances. As such, language impairments were evaluated in the domains of phonology, semantics, morphology, and syntax. Disturbances in these specific language areas were identified by relying on structured language measures that had been developed for the purposes of either identifying the linguistic sequelae in adults with aphasia after stroke or determining the presence of developmental language problems in children. Current evidence indicates that the cognitive–communicative deficits in TBI are not readily detectable by structured language measures (Chapman, 1997; Hartley & Jensen, 1991; MacDonald, 1993).

As evident from the literature review that follows, the language–brain framework established by classic aphasia theory has contributed to our understanding of language deficits in TBI. Moreover, recent developments have illustrated that discourse measures provide valuable indices of communicative competence in patients with stroke-induced aphasia in addition to the performance data derived from aphasia batteries (Chapman & Ulatowska, 1994; Ulatowska & Chapman, 1994). Such developments have paved the way for using discourse procedures to characterize the communicative ability or disability in TBI. Growing evidence indicates that discourse measures are more sensitive to the cognitive–linguistic sequelae associated with brain injury in both adult and pediatric populations than are

ACQUIRED APHASIA, THIRD EDITION

the structured measures of linguistic abilities (Chapman, 1997; Coelho, Liles, & Duffy, 1995; Hartley, 1995).

It is important to recognize that the cognitive–linguistic profiles in patients with TBI are distinct from the classic patterns seen in adult patients with stroke-induced aphasia or in children with developmental language delay. The distinctions between patients with TBI and adults with stroke-induced aphasia or children with developmental language problems arise largely from differences in the pathophysiology of the mechanisms of brain injury and from the diverse concomitant neurobehavioral disturbances. The management of the communicative disturbances in TBI requires an understanding of the neurological aspects of the injury; of the complex interactions between the injury mechanism, extent of brain injury, and disturbances to cognitive and linguistic–social systems; and of the clinical utility of discourse methods in elucidating the rich interplay between cognitive and linguistic abilities.

This chapter reviews the extant literature that has contributed to the current empirical and theoretical understanding of the cognitive–linguistic abilities in TBI populations. The specific issues address are

1. The pathophysiological profile of TBI.
2. The nature of the specific language disturbances identified in TBI.
3. The dilemmas in sorting out linguistic disturbances from confusional states.
4. Additional concomitant disturbances of speech and language.
5. The theoretical and clinical reasons for a shift in focus to discourse measures.
6. Critical issues related to recovery in communicative competence including age at injury, severity of injury, focality of lesion, and concomitant cognitive and behavioral disturbances.
7. Special considerations for management of cognitive–communicative behaviors.

Epidemiology and Mechanisms of Injury

Definitions

In contrast to the frequent occurrence of open head injuries from penetrating missile wounds (e.g., bullets, shell fragments) in casualties of war, closed head injury (CHI) predominates in civilian head trauma. The term CHI is used here to refer to head trauma in which the primary mechanism of injury is a sudden acceleration–deceleration imparted to the freely mov-

ing head. Impact of a blunt object is another common mechanism of CHI. The primary cause of CHI in many areas of the United States is vehicular accident (Kraus et al., 1984), whereas assault is a more frequent mechanism in some urban areas. Falls also are a common cause of head injury in young children.

Epidemiology

Kraus and coworkers (1984) reported incidence data based on all hospital admissions for traumatic brain injury in San Diego County. Using case ascertainment criteria, such as acute impairment of consciousness, Kraus et al. found an overall incidence of 180/100,000 population, which closely approximates previous findings reported for Olmstead County, Minnesota, over the period from 1935 to 1974 (Annegers, Grabow, Kurland, & Laws, 1980). As shown in the age- and sex-specific incidence curve (Figure 14.1), the incidence of head injury rises sharply in late childhood and reaches a peak exceeding 400/100,000 population in adolescent and young adult males. A second peak in incidence is seen in older adults, which could have an impact on rehabilitation services because of the shift in the age distribution of the general population. Although males predominate during most of the age span in hospital admissions of adults with CHI, the male–female disparity in head injury is low in young children and in adults over 70 years of age (Kraus et al., 1984).

Epidemiologic statistics vary depending on the source of information. An important epidemiologic finding in the San Diego study was that mild to moderate head injury accounted for about three-fourths of all admissions of acute head trauma (Kraus et al., 1984). In contrast, the impressions gained from rehabilitation studies about the frequency of aphasia probably reflect selection of more severely injured patients. At the other end of the continuum, available data probably underestimate the incidence of mildly injured patients, as many are treated and then released from emergency rooms.

Mechanisms of Injury

Neuropathologic investigation of the traumatized human brain (see Adams, Mitchell, Graham, & Doyle, 1977) and studies using experimental models of head injury in animals (see Ommaya & Gennarelli, 1974) have suggested that a primary mechanism of CHI is rotational acceleration of the skull, which produces shear strains within the intracranial contents. Histological study of the brains of patients dying soon after CHI has disclosed diffuse injury to the cerebral white matter, which apparently results

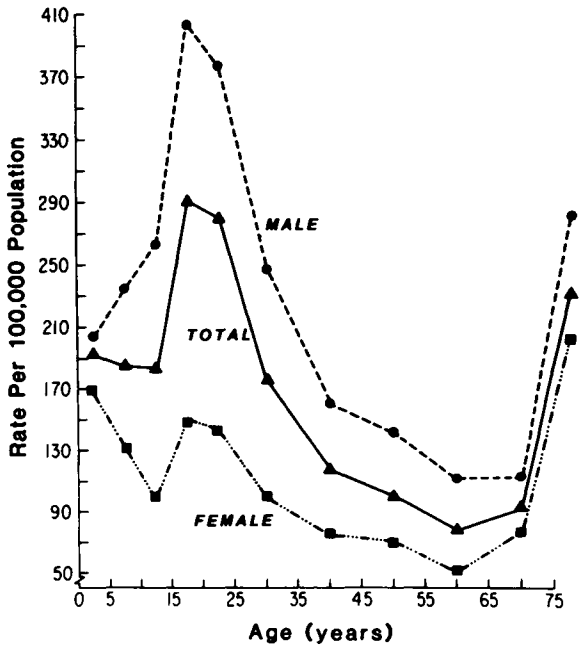


FIGURE 14.1. *Age- and sex-specific incidence rates of brain injury per 100,000 population, San Diego County, California, 1981. From Kraus et al. (1984). Reproduced with permission of the author and publisher.*

from shearing and stretching of nerve fibers at the moment of impact (Adams et al., 1977). Pertinent to the development of hemispheric disconnection, the corpus callosum is especially vulnerable to diffuse, mechanically induced shear strains. Ommaya and Gennarelli (1974) postulated that the severity of diffuse CHI follows a centripetal gradient; that is, the injury extends to the rostral brain stem only in cases with severe diffuse hemispheric injury. The bulk of cerebral white matter may be reduced further by delayed degeneration, which results in ventricular enlargement. Complications contributing to the severity of generalized CHI include brain swelling, increased intracranial pressure, hypoxia, and infection.

Focal lesions after CHI result from contusion of the brain surface by transient in-bending of the skull or by penetration of bone fragment in cases of depressed skull fracture, which may also produce brain laceration (Gurdjian & Gurdjian, 1976). Focal areas of ischemia are frequently present in the neocortex and basal ganglia (Graham & Adams, 1971). Stresses of the impact may cause arterial and venous tears resulting in intracerebral (see

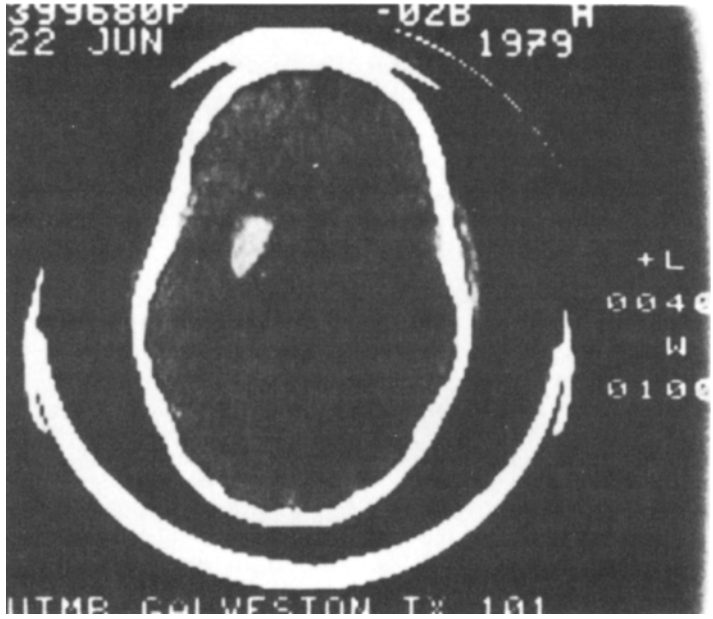


FIGURE 14.2. *Computerized tomographic scan obtained on the day of severe closed head injury in a 12-year-old girl struck by a car. The scan shows a left-hemisphere intracerebral hematoma in the putamen and anterior limb of the internal capsule. This left-handed patient had a right hemiplegia and was mute for 6 weeks after regaining consciousness.*

Figure 14.2) or extracerebral hematomas. The orbital surfaces of the frontal and temporal lobes are particularly vulnerable to contusion by impaction against the bony sphenoid wing. Formation of hematomas is also common in this area. Large mass lesions may produce contralateral shift of midline structures and tentorial herniation of the temporal lobe, possibly involving the uncus and hippocampus.

Assessment of Initial Injury

Closed Head Injury

Closed head injury often produces a period of amnesia, if not loss of consciousness, immediately after impact. The acute severity of diffuse CHI is measured by the degree and duration of altered consciousness. Teasdale and Jennett (1974) developed the Glasgow Coma Scale (GCS; see Table

TABLE 14.1
The Glasgow Coma Scale

Best eye opening	Best motor response	Best verbal response
4 Spontaneous	6 Obeys Commands	5 Oriented
3 To Speech	5 Localizes to Pain	4 Confused
2 To Pain	4 Flexion-Withdrawal to Pain	3 Inappropriate Words
1 None	3 Abnormal Flexion to Pain	2 Incomprehensible
	2 Extension to Pain	1 None
	1 None	

14.1) for the assessment of coma. This scale consists of three components: the minimal stimulus necessary to elicit eye opening, the best motor response to command or to painful stimulation, and the best verbal response. Summation of the component scores of the GCS yields a total score, which can range from 3 to 13. Jennett et al. (1977) defined a severe acute CHI as one that results in no eye opening, inability to obey commands, and no comprehensible speech, that is, a GCS score of 8 or less for a period of at least 6 hours. Recent outcome research has typically defined a moderate CHI primarily by impaired consciousness (i.e., a GCS score from 9 to 12) which does not produce coma, whereas a mild CHI is reflected by confusion and disorientation (a GCS score from 13 to 15) with negative findings on brain-imaging studies (e.g., computerized tomography, magnetic resonance imaging).

Recent evidence raises a question concerning the contribution of acute linguistic disturbance to duration of impaired consciousness. To investigate the relationship between lateralization of focal parenchymal lesion and impaired consciousness. Levin, Gary, and Eisenberg (1989) serially assessed selected patients from the Traumatic Coma Data Bank who had unilateral intracerebral lesions of at least 15 cc until they recovered from coma. When the criterion for resolution of coma was the return of the ability to obey simple commands, patients with left-hemisphere lesions were found to have a more prolonged period (mean = 32.8 days) of impaired consciousness than had patients who sustained focal right-hemisphere insults (mean = 8.8 days). In contrast, lateralization of lesion had no effect when localization of a painful stimulus (e.g., moving an arm toward the site of supraorbital pressure) was the criterion for improved consciousness in patients who were initially comatose. The investigators interpreted these findings as evidence for the contribution of acute disturbance of receptive language to the impression of more prolonged impaired consciousness in

CHI patients with left-hemisphere lesions. An implication is that nonverbal modes of response and processing are necessary to evaluate recovery of consciousness in head-injured patients with left-hemisphere lesions.

Confusion and anterograde amnesia (i.e., the inability to consolidate information about ongoing events) usually persist for a varying duration after the patient emerges from coma (Russell & Smith, 1961). The duration of posttraumatic amnesia (PTA) may range from a few minutes after mild CHI that produces no coma to several months following severe CHI. The duration of PTA is assessed directly by questioning the patient concerning orientation and recent events (Levin, O'Donnell, & Grossman, 1979) and is estimated retrospectively by inquiring about the period for which the patient has no remembrance (Russell & Smith, 1961). Focal brain lesions (e.g., hematomas) may occur in the presence of relatively mild or moderate diffuse CHI, as reflected by the period of coma and PTA.

Missile injury causes tearing of the scalp, depression or fracture of the skull, and possibly wounding of brain tissue in the track of the foreign body (see Figure 14.3). A small shower of bone fragments is often project-

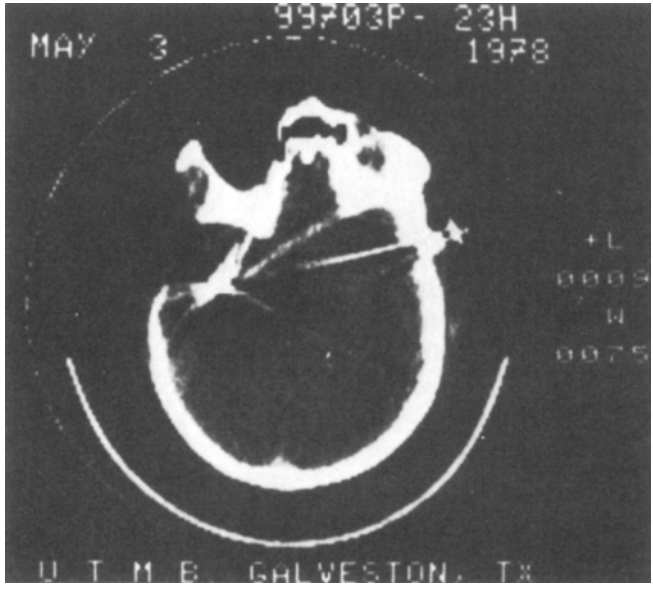


FIGURE 14.3. *Gunshot wound of the right frontotemporal region visualized by computerized tomography. Note the path of the bullet and bone fragments which traversed to the left temporal area. Wernicke's aphasia with jargon persisted for 18 months postinjury in this woman.*

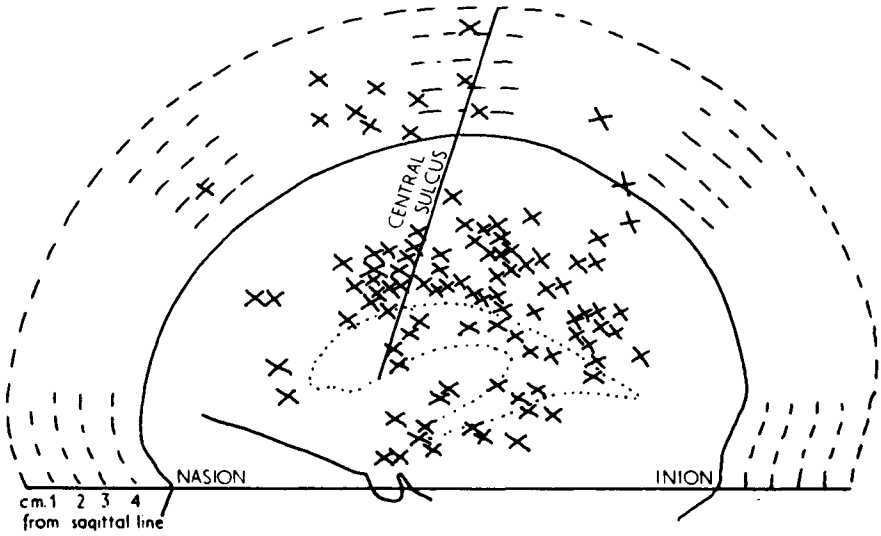


FIGURE 14.4. *Chart indicating the center of injury for missile wounds to the left hemisphere that caused aphasia. Cases with foreign bodies in remote regions of the brain were excluded from this map. Localization of missile wounds on the outline of a normal lateral skull was based on skull x-rays and surgical findings. From Russell and Espir (1961).*

ed into the brain from the point of impact; the extent of dural penetration and loss of brain tissue are indexes of injury severity (Newcombe, 1969). As a consequence of dural penetration, posttraumatic seizure disorder is more strongly associated with aphasia secondary to missile wounds than in cases of CHI (Russell & Espir, 1961).

To determine the locus of lesion, Russell and Espir (1961) used surgical findings and lateral and anteroposterior skull x-rays to chart the entry wound and missile track on a lateral sagittal diagram of the hemisphere (see Figure 14.4). Verification of lesion localization by postmortem data suggested that this was a fairly accurate method. Although missile wounds tend to be more circumscribed than diffuse CHI and produce little or no coma, metal fragments can spread far from the primary locus of injury. Furthermore, Mohr et al. (1980) found that missile injury that produced a language disorder was frequently associated with a period of unconsciousness, suggesting that diffuse effects were contributory. Missile injury that results in aphasia also commonly produces motor and/or sensory deficit contralateral to the dominant hemisphere; this association is stronger than in the case of CHI (Levin, Grossman, & Kelly, 1976).

From this summary of the pathophysiology of head injury, we may in-

fer that clinical data concerning the extent of focal brain injury and the severity of diffuse cerebral disturbance are pertinent to the assessment of posttraumatic aphasia.

SPECIFIC LANGUAGE DISTURBANCES IN TBI

One distinctive feature of acute aphasia after CHI is the predominance of anomia (Heilman, Safran, & Geschwind, 1971). Fluent speech is often associated with verbal paraphasia and circumlocution; comprehension and repetition are relatively spared, whereas naming is markedly defective, especially to confrontation. Anomic errors include semantic approximation (e.g., "snout" for tusks of an elephant), circumlocution (e.g., "to make music" for pedals of a piano), and concrete representation (e.g., "orange" for a circle).

Wernicke's aphasia is the second most common language disorder after CHI. Although an acute picture of fluent paraphasic speech, poor comprehension for oral and written language, and impaired repetition has been described in CHI cases with left temporal lesions (Heilman et al., 1971; Stone, Lopes, & Moody, 1978; Thomsen, 1976), restoration of comprehension may be rapid after a hematoma resolves or is surgically removed (cf. Stone et al, 1978).

We describe a case of transient Wernicke's aphasia that occurred after a relatively mild diffuse injury (as reflected by the GCS) concomitant with a suspected left-hemisphere mass lesion that was not directly visualized by computerized tomography (CT). The patient was a 17-year-old right-handed student who sustained a closed head injury in a motorcycle accident. When admitted to the neurosurgery service on the day of injury, he had a GCS score of 11 and no focal motor or sensory deficit. CT showed compression of the left lateral ventricle, which resolved during the course of hospitalization. The patient's speech was fluent at a rate faster than normal and was contaminated by jargon (e.g., "ruby baby"). Comprehension was grossly impaired, and the patient's mood was characterized by excitement and agitation. Throughout the first 2 weeks of his hospitalization, he was grossly disoriented and continued to exhibit Wernicke's aphasia. Stereotyped phrases and expletives represented the primary verbal output. The patient's orientation began to improve and reached a normal level by 3 weeks postinjury. Although a clinical interview showed substantial improvement in his comprehension; the Multilingual Aphasia Examination (MAE) given the fourth week postinjury disclosed defective visual naming (e.g., he described a rectangle as a "long square"), inability to repeat sentences presented orally, and decreased word finding. Follow-up assessment 6 months postinjury revealed total recovery of language (see Figure 14.5).

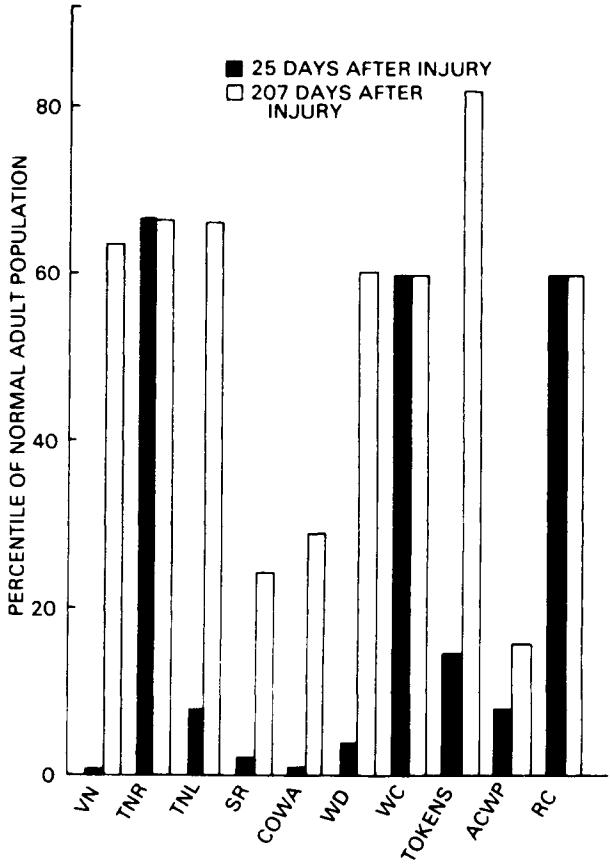


FIGURE 14.5. *Profile of language test scores obtained 1 and 6 months postinjury in a 17-year-old student who had a Wernicke's aphasia that completely resolved. VN, Visual Naming; TNR, Tactile Naming, Right Hand; TNL, Tactile Naming, Left Hand; SR, Sentence Repetition; COWA, Controlled Word Association; WD, Writing to Dictation; WC, Writing–Copying; TOKENS, Token Test; ACWP, Auditory Comprehension of Words and Phrases; RC, Reading Comprehension.*

Open Head Injury

Most published studies of aphasia after missile wounds to the brain are based on detailed observations of servicemen who were treated at the Military Hospital for Head Injuries in Oxford during and after World War II (Newcombe, 1969; Russell & Espir, 1961; Schiller, 1947). Mohr et al. (1980) and Ludlow and coworkers (1986) extended this research to include servicemen who sustained penetrating head injuries in Viet Nam. These au-

thors have frequently described linguistic disturbance characteristic of Broca's aphasia, which is typically seen after occlusion of the left middle cerebral artery.

Russell and Espir (1961) obtained information on localization of injury by separately studying aphasics who had circumscribed left-hemisphere wounds without foreign bodies in remote areas of the brain (see Figure 14.4). In contrast to the rare occurrence of nonfluent agrammatic language disturbance after CHI, Russell and Espir (1961) reported that 12% of aphasics with missile wounds had Broca's aphasia, which was typically associated with right-sided weakness and a focal injury to the frontal or Rolandic area. In a related study, Schiller (1947) linked a disturbance of articulation, inflection, and rate of speech with a wound at the foot of the precentral convolution. He observed that agrammatism, disturbed prosody, and perseveration were present in patients with left frontotemporal missile wounds. Russell and Espir found focal missile wounds in the dominant parietal lobe to result frequently in a global aphasia, although small posterior parietal lesions resulted in specific anomia, alexia, and agraphia. Similarly, Mohr et al. (1980) noted that parietal injury was more likely to produce aphasia than was a focal wound of any other lobe. Global aphasia with jargon, prolonged posttraumatic amnesia, and residual memory deficit have been observed during the early stage of recovery from penetrating injury of the left temporal lobe (Russell & Espir, 1961). Focal temporal wounds damaging the optic radiations resulted in a visual field defect in addition to global aphasia. Impairment of reading was common in these patients.

Russell and Espir (1961) analyzed the occurrence of aphasia after unilateral brain wounds separately for right- and left-handers. The authors defined handedness in terms of preference for a majority of motor skills. As anticipated from other sources of data concerning cerebral dominance, only 1% of right-handers became aphasic after right-hemisphere wounds, whereas 17% of left-handers became aphasic after right-hemisphere wounds. Also consistent with other lines of evidence for cerebral dominance, unilateral left-hemisphere wounds more frequently produced aphasia in right-handers (65%) than in sinistrals (38%). The overall figures for aphasia after unilateral injury of either hemisphere were 37% for right-handers and 27% for left-handers. Although these figures are incompatible with the concept that sinistrals are more likely to become aphasic, the comparative data presented by Russell and Espir included right-handed patients with lesions outside the speech territory. Of the right-hemisphere wounds that resulted in aphasia in sinistrals, the frontal or parietal lobes were involved in all cases.

In summary, the pattern of aphasia observed after missile wounds to the brain conforms fairly well to the localization of language in patients with

cerebrovascular disease (see Chapter 3 for background). The localizing significance of missile wounds is greatly enhanced by identifying patients in whom there is no evidence of additional brain penetration by fragments in remote areas (see Figure 14.4).

Language Disturbances versus Confusional States

PERIOD OF POSTTRAUMATIC AMNESIA

As mentioned in the review of mechanisms of injury, the early postcomatose stage of recovery from CHI is typically characterized by an amnesic condition during which the patient is confused. Reduplicative paramnesia (Benson, Gardner & Meadows, 1976)—that is, the mistaken identification of a person, place, or event for one previously experienced—confabulation, and profound impairment of memory may be misinterpreted as signs of language disorder. The distinction may be particularly difficult in a patient whose fluent speech is disconnected and perseverative. Confused, nonaphasic speech after CHI was evident in a patient studied in Galveston.

A 24-year-old, right-handed woman was transferred from a community hospital to the University of Texas Medical Branch 3 hr after she sustained a CHI in a motor vehicle accident on 28 January. The GCS score was 8 when she was initially examined. The cerebral ventricles and cisterns were poorly visualized on CT, suggesting the presence of diffuse cerebral swelling. Although she obeyed commands after 4 days, her disorientation persisted until 5 March. Spontaneous speech during the confusional period was continuous, rambling, and disorganized in this fearful, agitated woman. When queried on 27 February regarding the reason for her hospitalization, she responded, "Something that came up natural being born somewhere born somewhere in here." Later in the day the patient elaborated that she was in the hospital "to have a baby" and that the year was 1952 (she was born in 1953). Reminiscent of the patients described by Weinstein and Kahn (1955), during examination the following day, she commented that she was hospitalized because of "being stabbed." This statement was interpreted as a reference to her intravenous tubes. Assessment of language on 19 April, however, disclosed completely normal findings on the MAE.

Analysis of this patient's utterances provided little evidence of consistent paraphasic errors, particularly when she was asked structured questions that focused on specific objects rather than on expository material. Repeated questioning within the limits of her short attention span disclosed no evidence of receptive impairment similar to that found in patients with Wernicke's aphasia. Administration of tests of naming and

word finding to this patient during PTA would likely have yielded defective scores. Weinstein and Kahn (1955) described patients with brain damage of diverse etiologies, including diffuse cerebral disturbance, who exhibited misnaming that was qualitatively atypical for aphasia. Anomic errors were frequently associated with objects that bore a relation to the patient's illness and frequently occurred during a period of disorientation, confabulation, and denial of illness. The authors observed that, in contrast to patients with classical anomic aphasia, patients exhibiting nonaphasic misnaming frequently showed no evidence of groping for words in their spontaneous speech nor did their naming necessarily improve when correction was offered.

Conversely, the presence of paraphasic errors in conversational speech after CHI may be misinterpreted as evidence for disorientation and confusion. This condition is likely to be found in CHI patients with mass lesion or depressed skull fracture involving the left hemisphere. In such cases, a multiple-choice format of testing orientation may be useful, as well as relatively nonverbal tests during the early stages of recovery.

POSTCONFUSIONAL STAGE OF RECOVERY

Few studies of aphasia during the early stages of recovery from CHI have concurrently assessed orientation. Consequently, there is a possibility that PTA had not completely resolved at the time language was evaluated.

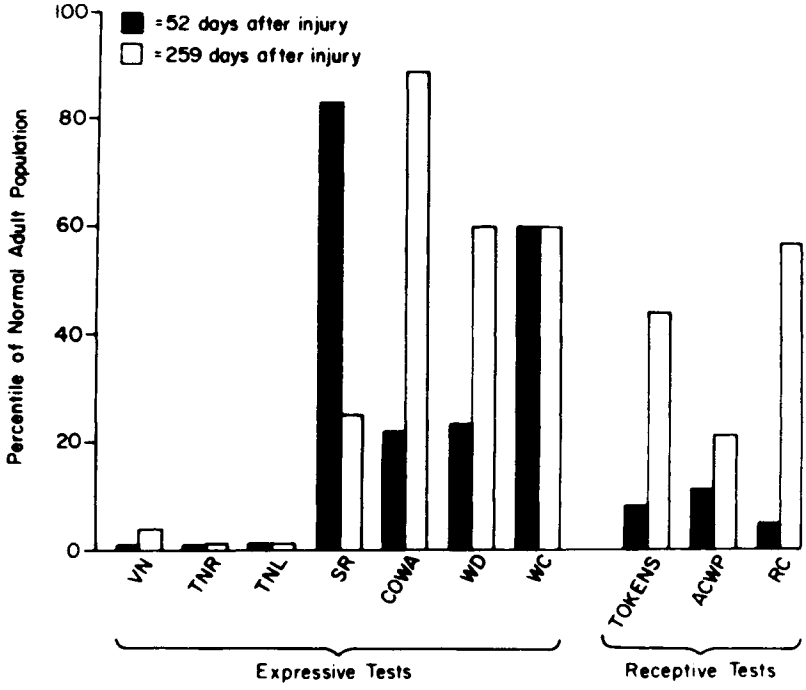
Clinical examination of language in consecutive CHI admissions at the Boston City Hospital by Heilman et al. (1971) yielded 13 cases of aphasia, including 9 patients with anomic aphasia and 4 cases of Wernicke's aphasia. Aphasics accounted for 2% of the Boston series, a base rate close to that obtained in a previous study of consecutive CHI admissions (Arseni, Constantinovici, Iliescu, Dobrota, & Gagea, 1970). In the Boston study, the authors defined anomic aphasia as a fluent aphasia in which the patient demonstrates verbal paraphasia for all kinds of material, especially to confrontation. Wernicke's aphasia was defined as a fluent aphasia with paraphasia, impaired comprehension for spoken and written language, and poor repetition. Broca's aphasia was defined as nonfluent aphasia with relatively intact comprehension. No patient had a Broca's aphasia or exhibited a total disruption of language. Heilman et al. (1971) excluded patients with intracranial surgery (other than evacuation of subdural hematoma). This strategy of patient selection, combined with the relatively high proportion of falls relative to motor vehicle accidents, may have restricted patients with mass lesions, thereby resulting in fewer aphasic cases compared with those of other neurotrauma centers. Heilman et al. (1971) distinguished the anomia in their CHI patients from nonaphasic misnaming

(Weinstein & Kahn, 1955). In contrast to the narrow range of anomic errors (e.g., related to illness) in cases of nonaphasic misnaming, the anomic CHI patients described by Heilman et al. exhibited diverse naming defects in spontaneous speech and writing.

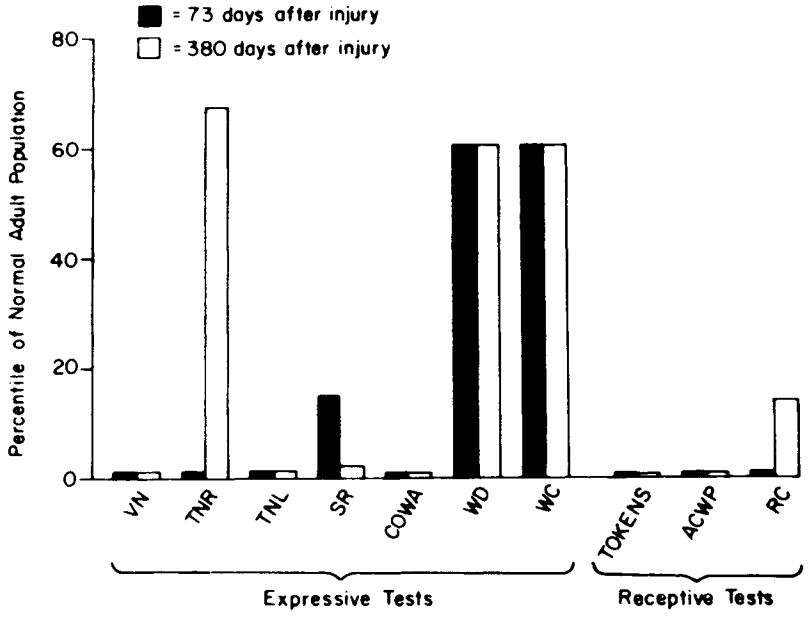
As shown in the following case, we have also been able to distinguish anomic aphasia from nonaphasic misnaming by delaying evaluation of language until the injured patient recovers to a normal level of orientation. This case was a 17-year-old student who was transferred to the University of Texas Medical Branch 3 hours after a motor vehicle accident. The GCS score on admission was 8. Although the patient obeyed commands on the day of admission, delayed neurological deterioration was reflected by the development of a right hemiparesis and evolution of a Wernicke's-type aphasia. Three days postinjury a partial left temporal lobectomy was performed with evacuation of an intracerebral hematoma. The patient remained confused for a month after injury but exhibited gradual improvement of receptive language. Administration of the MAE two months after injury disclosed findings consistent with an anomic aphasia. There were frequent errors of circumlocution (e.g., she described an island as "a place where you fish"), semantic approximation (e.g., she described the trunk of an elephant as a "nose"), and a tendency to substitute names of concrete objects for geometric designs (e.g., she described a triangle as "the thing you use when you play pool"). As shown in Figure 14.6A, the patient's long-term recovery of language was complete, except for a subtle residual anomic disturbance that was evident only under testing conditions.

Anomic aphasia may also persist after resolution of PTA in patients with severe diffuse CHI who evidence no other focal neurologic signs. In a study of 26 CHI patients without mass lesions who had been in a coma for a least 24 hours, Thomsen (1975) found that aphasic symptoms were present during the first 2 or 3 weeks after injury in 12 cases. Verbal paraphasia (i.e., substitution of inappropriate words) and anomia were the most common defects; receptive impairment and dysgraphia were also frequently observed, whereas agrammatisms and other symptoms suggestive of Broca's aphasia were rarely seen.

FIGURE 14.6. *(A) Baseline and follow-up language profiles of a 17-year-old student with residual anomia that was initially accompanied by a receptive impairment after surgical evacuation of a left temporal intracerebral hematoma. (B) Baseline and follow-up findings in a 19-year-old student which show persistent impairment of expressive and receptive language associated with cognitive deficit and progressive ventricular enlargement. He sustained a severe diffuse injury (coma = 21 days) complicated by bifrontal subdural hematomas. VN, Visual Naming; TNR, Tactile Naming, Right Hand; TNL, Tactile Naming, Left Hand; SR, Sentence Repetition; COWA, Controlled Word Association; WD, Writing to Dictation; WC, Writing–Copying; TOKENS, Token Test; ACWP, Auditory Comprehension of Words and Phrases; RC, Reading Comprehension. From Levin, Grossman, Sarwar, and Meyers (1981). Reproduced with permission by the publisher.*


100
80
60
40
20
0
= 52 days after injury
= 259 days after injury
Percentile of Normal Adult Population
VN
TNR
TNL
SR
COWA
WD
WC
TOKENS
ACWP
RC
Expressive Tests
Receptive Tests

A


80
60
40
20
0
= 73 days after injury
= 380 days after injury
Percentile of Normal Adult Population
VN
TNR
TNL
SR
COWA
WD
WC
TOKENS
ACWP
RC
Expressive Tests
Receptive Tests

B

The following is a case that closely resembled the series of diffuse head injuries described by Thomsen. A 20-year-old, right-handed man was admitted to the hospital with a servere CHI (GCS score = 5) but no focal motor deficit. The CT scan suggested generalized brain swelling without a mass lesion. Baseline assessment 4 months after injury disclosed anomic errors that deteriorated into jargon (e.g., the handle of a fork was described as a "forkline" and the posterior aspect of the leg was described as a "negline"). Circumlocution was evidenced by his response when the examiner pointed to the pedals of a piano: "If you want a different sound, push them down." Word finding was defective on a test of letter–word association.

Clinical examination of language has disclosed a broad range of language defects in CHI patients. Thomsen (1976) characterized the findings in a series of patients with left-hemisphere mass lesions as "multisymptomatic aphasia." She used this term to describe patients who exhibited anomia, agraphia, and impaired comprehension; one-third of the patients in her series had global or receptive aphasia. Anomic aphasia was less common, and there were no cases of Broca's aphasia. Posttraumatic dyslexia and dysgraphia have also been reported by other authors (de Morsier, 1973).

Additional Concomitant Disorders of Speech and Language

Posttraumatic disorders of speech and language may also include mutism, stuttering, echolalia, palilalia, and dysarthria.

MUTISM

Total abolition of speech and language may occur after termination of coma in patients capable of following commands during the transition between spontaneous eye opening and recovery of orientation. As previously described, transient mutism is characteristic of aphasia after head injury in children.

Prolonged if not permanent speechlessness is observed in adults who are persistently vegetative or exhibit akinetic mutism (Cairns, 1942; Plum & Posner, 1980). The akinetic type is a form of subacute or persistent mutism with little or no vocalization. Behaviorally this condition is distinguished from the vegetative state by its immobility. The features common to both conditions include apparent wakefulness with restoration of the sleep–wake cycle and inability to demonstrate cognitive function through interaction with the environment. When akinetic mutism is a sequel to CHI, diffuse cerebral injury is to be suspected.

Geschwind (1974) distinguished between nonaphasic and aphasic

mutism. The aphasic type, which was thought to occur rarely in adults with CHI (cf. de Morsier, 1973), is accompanied by linguistic errors in writing. Nonaphasic mutism is associated with acute onset of right hemiplegia; writing is normal and there are no signs of aphasia when speech is restored. Following Bastian (1898), Geschwind referred to this condition as aphemia rather than aphasia. In such cases mutism may arise from focal lesions, often involving the basal ganglia. In the following paragraph, we describe a case of subcortical mutism.

In a prospective study of patients admitted to neurosurgery services in Houston and Galveston, posttraumatic mutism was present in nine patients (nearly 3% of the series) despite recovery of consciousness and communication through a nonspeech channel (Levin et al., 1983). CT scans revealed subcortical lesions situated primarily in the putamen and internal capsule of four patients, and four of the remaining five without subcortical lesions had left-hemisphere cortical injury. The patients without subcortical injury visualized by CT exhibited a longer duration of impaired consciousness consistent with diffuse brain injury and showed more long-term linguistic deficits. The four patients with basal ganglia lesions included two children and an adolescent, a finding consistent with other evidence that basal ganglia lesions in CHI may be more common in the pediatric age range.

STUTTERING

Published studies suggest that stuttering is a more common sequel of penetrating missile wound than of CHI (Peacher, 1945). De Morsier (1973), however, noted a fluency disorder in more than half of his series of CHI patients, including four cases with posttraumatic stuttering. Helm, Butler, and Benson (1978) implicated bilateral injury in patients with acquired stuttering after CHI.

ECHOLALIA AND PALILALIA

Echolalia is the repetition of words spoken by others, whereas palilalia is the automatic repetition of one's own words. Echolalia may follow a period of mutism in cases with diffuse cerebral dysfunction (CHI) or may occur in patients with transcortical motor aphasia, that is, disturbed expressive and receptive language with preserved repetition. Apart from generalized cerebral disturbance, these disorders have been associated with large frontal lesions. According to Geschwind (1974), echolalia and palilalia are uncommon in patients with lesions primarily involving the perisylvian region of the dominant hemisphere.

Stengel (1947) distinguished between the automatic and mitigated forms of echolalia. The former is parrot-like, with no elaboration of the in-

put. Mitigated echolalia is the questioning repetition of words spoken by others, often with a change of personal pronoun. Stengel postulated that mitigated echolalia may facilitate comprehension in patients with receptive language disturbance. Accordingly, the transition from automatic to mitigated echolalia may be a sign of clinical improvement that parallels the developmental sequence in children. Stengel also observed that the mitigated type may be confined to social conversation and less evident when the patient is directly questioned by an unfamiliar speaker.

Thomsen (1976) reported three cases of echolalia in a series of 50 patients with severe CHI. Of the two echolalic patients with left-hemisphere mass lesions, one initially had a global aphasia and the other had minimal spontaneous speech. The third patient, who sustained a severe diffuse CHI with residual hydrocephalus, evidenced echolalia and palilalia. In contrast to the general association of echolalia with impoverished spontaneous speech (see Geschwind, 1974), Thomsen commented that the patient with diffuse CHI talked incessantly without monitoring the amount of output. We also studied a patient who developed a similar echolalia after CHI.

An 18-year-old, right-handed student was brought to the emergency room of the University of Texas Medical Branch shortly after an automobile accident on 11 May. Initial examination disclosed a GCS score of 6, fixed and dilated pupils, and a right hemiparesis. A CT scan on the day of injury was normal. She slowly improved and eventually followed commands on 9 June. After transfer to the Del Oro Rehabilitation Hospital in Houston on 16 June she remained confused and disoriented until 26 June. During this period, the patient's spontaneous speech changed from an overall impoverishment to a greater-than-normal flow in which automatic echolalia was prominent. Observations by neuropsychologists during the course of rehabilitation showed a transition to mitigated echolalia which resolved by the middle of July. Repetition was most evident in the presence of persons familiar to the patient. An aphasia examination on 16 July disclosed intact spontaneous speech and relatively normal naming. Echolalia had resolved, but repetition of sentences and verbal associative fluency were markedly impaired. Comprehension of complex commands on the Token Test was also defective, although the patient could read and comprehend single words and phrases. Further progress in rehabilitation was complicated by her disinhibited behavior, a finding in agreement with Stengel's (1947) interpretation of echolalia as a failure of inhibitory control.

In summary, echolalia and palilalia are infrequent sequelae of CHI that are found in cases with severe diffuse CHI or with large mass lesions in the dominant hemisphere. The absence of any reference to echolalia and palilalia in several studies supports the contention that they rarely occur

after CHI (Levin et al., 1976; Najenson, Sazbon, Fiselzon, Becker, & Schechter, 1978; Sarno, 1980).

DYSARTHRIA

Sarno (1980) defined dysarthria as a speech disorder arising from pathology in the motor speech system that is evident in defects of the acoustic aspects of the speech stream (i.e., articulation, resonance, stress, and intonation). The severity of dysarthria varies from articulatory imprecision to completely unintelligible speech. Dysarthria may be caused by a lesion of either the central or the peripheral nervous system. Peacher (1945) reviewed the cases of dysarthria recorded by U.S. Army hospitals during World War II. Of the injuries producing dysarthria, which were primarily missile wound, 69% involved a lesion of the peripheral nerves. Trauma to the facial nerve was the most common site of lesion, although Peacher did not distinguish between central and peripheral facial nerve injuries.

Investigators of speech disorder after CHI have frequently reported dysarthria in patients with focal mass lesion of the left hemisphere (Alajouanine, Castaigne, Lhermitte, Escourolle, & De Ribaucourt, 1957; de Morsier, 1973; Thomsen, 1975) and in cases of diffuse cerebral injury (Sarno, 1980, 1984; Sarno, Buonaguro, & Levita, 1986; Thomsen, 1976). Dysarthric patients are frequently hemiparetic or may be quadraplegic. Serial assessment of language after severe CHI has suggested that dysarthria often accompanies aphasia during the early stage of recovery from CHI, and may persist after restoration of language. This dissociation is illustrated in a patient who was admitted to the University of Texas Medical Branch.

A 33-year-old, right-handed carpenter sustained a severe CHI in a motorcycle accident on 17 December. Evaluation in the emergency room shortly after injury disclosed a GCS score of 4. A CT scan showed a large left parietotemporal epidural hematoma, which was evacuated on the day of admission. Although he progressively improved and followed commands on 20 December, a left facial palsy and right hemiparesis remained. The combined aphasia and severe dysarthria rendered his speech unintelligible. By the first week in January, the patient's language and speech disorder partially resolved, although he continued to evidence anomia and impaired comprehension. A CT scan 10 months postinjury disclosed a large hypodense area at the site of the operated hematoma and a small hypodense area in the genu of the left internal capsule which was interpreted as a small lacunar infarct. He was transferred to a rehabilitation center prior to neuropsychological evaluation but returned a year later for testing. Despite frequent articulatory defects, expressive and receptive lan-

guage skills had uniformly recovered, as reflected by normal scores on all subtests of the MAE.

In contrast to this patient's case, Sarno (1980, 1984) and Sarno et al. (1986) reported that subclinical language deficit (e.g., decreased word fluency) was present in all dysarthric patients in a series of CHI cases. The findings in the Galveston patients suggest that the correspondence between language skills and motor speech varies depending on the interval between injury and assessment.

We may conclude from these studies that assessment of communicative disorder after CHI should include evaluation of dysarthria. The tests for articulatory agility and rating speech characteristics that are included in the Boston Diagnostic Aphasia Battery (Goodglass & Kaplan, 1983) are brief and useful for this purpose.

Cognitive–Communicative Deficits beyond Aphasia

The administration of standardized examinations for aphasia has yielded a characteristic profile of language disturbance in adults after CHI. This strategy has disclosed that language processing deficits on testing in the absence of clinical manifestations of classical aphasia is a common finding in CHI (Sarno, 1980, 1984; Sarno et al., 1986). Moreover, quantitative and qualitative assessment of discourse has facilitated the study of long-term recovery in CHI (cf. Levin, Grossman, Sarwar & Meyers, 1981).

Structured Language Measurement

Profiles of language disorder after CHI have been developed using the MAE (Benton, 1967; Benton & Hamsher, 1978) and the Neurosensory Center Comprehensive Exam for Aphasia (NCCEA; Spreen & Benton, 1969). The MAE evaluates expressive language on subtests of naming pictures of objects (Visual Naming), Sentence Repetition, Digit Repetition, and retrieving words beginning with a designated letter (Controlled Word Association). Benton and Hamsher (1978) included a spelling test in their 1978 revision of the MAE. Comprehension of oral language is evaluated by the Token Test and by a receptive test in which the patient points to the picture corresponding to a word or phrase presented orally (Aural Comprehension of Words and Phrases). Reading comprehension is tested using a similar format. The NCCEA (Spreen & Benton, 1969) includes similar tests, in addition to tests of naming objects presented tactually (Tactile Naming),

construction of sentences (Sentence Construction), identification of objects by name, oral reading, writing names, writing to dictation and copying, and articulation. Both examinations yield a percentile score based on normative data for each subtest; the manual for the NCCEA also provides percentile scores based on the performance of aphasics. Gaddes and Crockett (1973) published normative data for children on the NCCEA. The Boston Diagnostic Aphasia Test (Goodglass & Kaplan, 1983) also provides a profile of language abilities. It incorporates tests for articulation, repetition of automatized sequences, and rating of spontaneous speech.

We administered portions of the MAE and NCCEA to a consecutive series of patients with CHIs of varying severity (Levin et al., 1976). In this study, injury that produced no neurological deficit or loss of consciousness longer than a few minutes was designated as Grade I; Grade II referred to an injury producing a coma of not longer than 24 hr; and Grade III designated an injury that resulted in a period of coma exceeding 24 hr. A language subtest score that fell below the second percentile of the normative population was considered defective. Whereas clinical examination of spontaneous speech disclosed evidence of aphasia in only eight patients (16% of the series), nearly one-half of the patients were impaired in naming objects (Figure 14.7). Word-finding difficulty (Controlled Word Association) and impaired writing to dictation were also common expressive defects in this series. In contrast, Figure 14.7 shows that repetition of sentences was well preserved. Nearly one-third of the patients had difficulty in comprehending complex oral commands on the Token Test. The results provided strong support for the presence of higher-level deficits in manipulating the system in apparently nonaphasic CHI patients, including cases with injuries of moderate severity.

Sarno et al. (1986) elucidated the characteristics of language and speech disorder after CHI in a study of 125 CHI patients who were rendered comatose for periods ranging from 15 min to 6 months. These patients were referred to the Rusk Institute for Rehabilitation Medicine in New York. On the basis of clinical evaluation and administration of subtests of the NCCEA (median injury–test interval of 45 weeks), the authors classified the patients into categories of grossly obvious aphasia, dysarthria with language deficit reflected by test scores ("subclinical" aphasia), and language deficit without dysarthria. They found that the proportion of patients with each category of language disturbance was approximately equal. The aphasic group ($n = 37$) consisted of 19 patients (51%) with fluent aphasia, 13 (35%) with nonfluent aphasia, and 5 (14%) with global aphasia. As depicted in Figure 14.8, aphasic patients had linguistic test scores that fell below the other groups. Most aphasics had defective scores on all four lan-

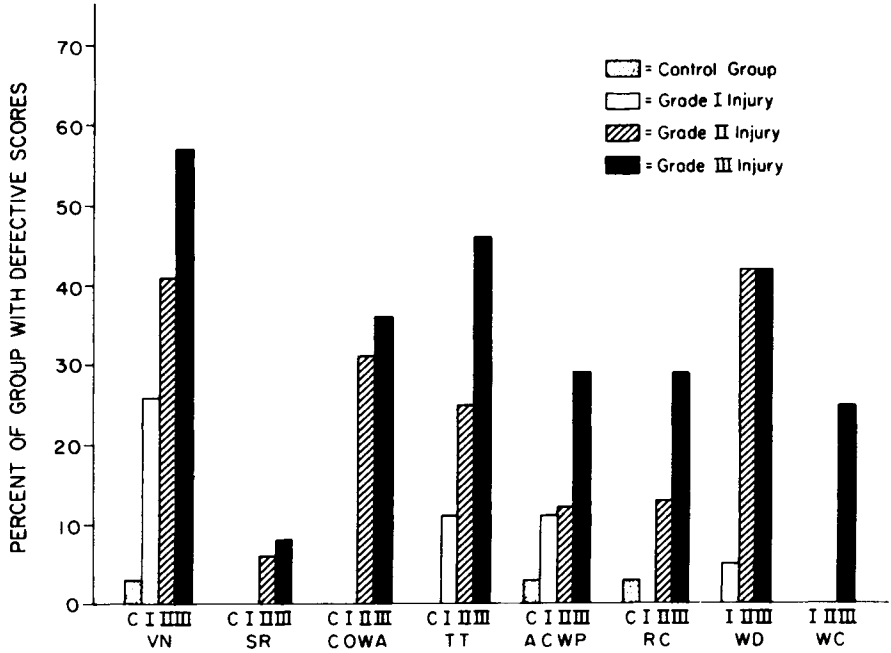


FIGURE 14.7. *Percentage of head-injured and control patients with defective scores in the Multilingual Aphasia Examination. VN, Visual Naming; SR, Sentence Repetition; COWA, Controlled Word Association; TT, Token Test; ACWP, Auditory Comprehension of Words and Phrases; RC, Reading Comprehension; WD, Writing to Dictation; WC, Writing–Copying. From Levin, Grossman, and Kelly (1976). Reproduced with permission of the publisher.*

guage subtests that were administered, whereas 56% of the subclinical patients failed at least two of the four tests. The patients with dysarthria had word fluency and sentence repetition scores that fell below the pure subclinical group. No CHI patients in this series, however, obtained completely normal scores.

Figure 14.8 shows the test results of the CHI patients in Sarno et al.'s (1986) study transformed into percentile scores for an aphasic population. Accordingly, any score below the 90th percentile is impaired in relation to normal subjects. The mean scores indicated reduced word fluency in the subclinical groups, although visual naming is also compromised. Consistent with the results of the Galveston study, Sarno's subclinical patients without dysarthria had adequate sentence repetition, whereas the dysarthric patients exhibited difficulty on this task. Impaired comprehension of complex oral commands was also found in patients without obvious

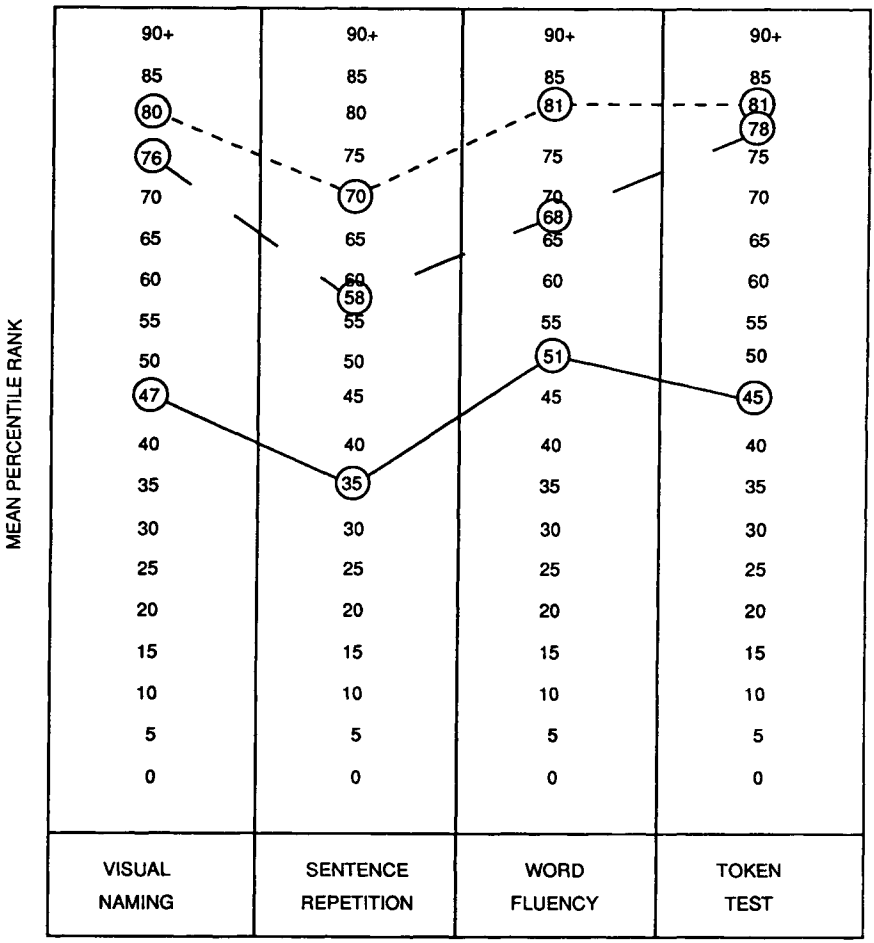


FIGURE 14.8. *Mean percentile rank of aphasic and subclinical language disorder groups on subtests of the Neurosensory Center Comprehensive Examination of Aphasia.* ——, *Aphasic;* – –, *dysarthric and subclinical;* - - -, *subclinical. Note that the percentile scores are based on results obtained in an aphasic population. From Sarno, Buonaguro, and Levita (1986). Reproduced with permission by the author and publisher.*

aphasia. In a discussion of the results obtained in the Galveston study and in her own investigation of language disturbance after CHI, Sarno and colleagues (1986) concluded that sophisticated, neurolinguistic measures were necessary to detect the nature of the deficits in this population to guide effective clinical judgment.

Disparity between Discourse and Results on Structured Tests

Recent studies have shown that head injury and other etiologies of acquired brain damage can produce a residual impairment of discourse ability despite recovery of performance on structured language tests as described in the previous section. Consistent with description of residual speech following severe head injury as being tangential, if not confabulatory, several studies have reported diminished efficiency of communication in the discourse of CHI patients (Chapman 1995; Ehrlich, 1988; Mentis & Prutting, 1987; Novoa & Ardila, 1987; Penn & Cleary, 1988; Wyckoff, 1984). The studies by Chapman (1995), Ehrlich (1988), Mentis and Prutting (1987), and Wyckoff (1984) were confined to CHI patients, whereas the other investigations included other etiologies. Techniques used in studies of discourse include asking patients to generate stories in response to a series of pictures (e.g., comic strip), to relate a personal story, to retell a story, and to generate a procedural narrative such as how to buy groceries (Biddle, McCabe, & Bliss, 1996; Coelho, et al., 1995; Wyckoff, 1984).

An important methodological aspect of discourse approaches is that they offer ways to evaluate the ability to manipulate larger units of language than can be realized through examining competence at the single-word or sentence level. It is now widely recognized that the ability to utilize the formal aspects of the language system (e.g., semantics, syntax) does not necessarily correspond with the ability to process discourse-level information (Chapman, Watkins, et al., 1997).

Discourse in Adults with TBI

In a study that analyzed the discourse responses for two narratives (in response to a comic strip and retelling a story) and one procedure (explaining how to buy groceries), Wyckoff (1984) compared the findings obtained in 11 survivors of severe CHI (including five patients who were considered to be aphasic according to a standardized battery of language tests) with a group of normal subjects. The discourse of the head-injured patients was characterized by slow speech, in which a greater percentage of syllabic utterances were dysfluencies, and by generally diminished productivity. The head-injured patients produced about one-half to two-thirds of the amount of accurate content relative to the discourse of normal speakers. Moreover, the discourse of head-injured patients contained fewer cohesive ties between utterances, a feature exemplified by failure to provide a ref-

erent. Wyckoff concluded that discourse analysis could potentially identify difficulty in functional communication, which would otherwise be overlooked by traditional, standardized language tests. At the same time, she suggested that further study utilizing spontaneous conversation with another speaker could potentially identify additional deficits, such as difficulty in topic maintenance.

In a study of 10 nonaphasic CHI patients who returned for linguistic examination at least 6 months posttrauma, Ehrlich (1988) analyzed the stories that they recited in response to the "Cookie Theft" picture from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). The only difference that emerged in the comparison of narratives by these patients with those of normal adults was a reduction in the amount of information per unit of time. This pattern reflected the tendency of CHI patients to utter lengthier, slower spoken language.

More detailed analysis of discourse in CHI adults has involved assessment of cohesion using the technique developed by Halliday and Hassan (1976): two items are cohesively related (i.e., a cohesive tie) if the interpretation of one of the elements in a text depends on reference to another (based on grammar and vocabulary). This methodology has shown that head-injured survivors frequently fail to provide the speaker with a referent. Fewer cohesive ties per communication were found in the discourse of survivors of severe CHI compared with that of normal subjects (Hartley & Jensen, 1991; Liles, Coelho, Duffy, Robert, & Zalageus, 1989; Wyckoff, 1984). Mentis and Prutting (1987) studied cohesion in the discourse of three patients who had sustained a severe CHI at least 1 year earlier. In common with the patients studied by Ehrlich (1988), these CHI survivors were nonaphasic according to clinical examination and to their scores on a conventional aphasia test battery. A conversation and narrative language sample (e.g., description of the patient's work) with a familiar partner were videotaped. Although the number of cohesive ties was similar in the conversation of the two groups, the normal subjects had a greater number of cohesive ties in their narratives compared with the CHI patients, who exhibited no variation under the two conditions. The investigators postulated that word retrieval problems contributed to the reduced number of cohesive ties in the narratives of the head-injured patients.

In addition to cohesive analysis, Liles and colleagues examined complete episodic structure in the narrative discourse of adults with TBI. These researchers found that adults with TBI produced fewer complete episodes compared with normal control subjects. Other studies have verified the finding of incomplete narrative structure in adults with TBI manifested by omission of critical information (Biddle, et al., 1996; Glosser & Deser, 1990; Hartley & Jensen, 1992).

Relationship of Impaired Discourse to Frontal Lobe Lesions

Of the few published studies of discourse analysis in brain-damaged patients, only sparse data have been reported on localization of lesion. This issue is relevant to linguistic deficit in head-injured patients because the frontal region is the most common site of focal lesions. To this end, Novoa and Ardila (1987) tested 21 patients with prefrontal lesions resulting from several etiologies, including head trauma. Administration of various linguistic tasks and open-ended questions disclosed that patients with left frontal lesions had diminished verbal output and exhibited a tendency toward perseveration, confabulation, and disorganized ideas. The investigators also noted that patients with prefrontal injuries had difficulty in responding with interpretative statements, a finding similar to the problem in abstraction that was reported by Ulatowska, Freedman-Stern, Doyel, and Macaluso-Haynes (1983).

The relationship of localization of lesion within the frontal region to discourse was studied by Kaczmarek (1984). The adult patients were asked to repeat stories that had been told by the examiner, describe a situation depicted by pictures, and generate stories about specific topics, some familiar to the patient and others more abstract. Although Kaczmarek studied brain tumor patients, the study is described here because of its relevance to head-injured patients. The most prominent finding in the patients' discourse was their inability to organize information, as reflected by their excessive perseverations, digressions, confabulations, and use of stereotyped phrases. The perseverations, which were especially frequent in the patients with left dorsolateral frontal lesions, typically occurred when the patients were initiating their narratives, and they often failed to continue.

Implications of Discourse Studies

The recent trend toward quantitative analysis of discourse extends the concept of subclinical language disorder to patients who are not obviously aphasic by clinical examination. As pointed out earlier in this chapter, the communication problems of CHI patients often reflect the combined effects of impaired cognition and language. Moreover, the tangential, perseverative, and slow discourse of many survivors of severe CHI contributes to their disability and social isolation. Future studies could utilize neuroimaging to analyze the relationship of lesions in specific frontal lobe sites to various abnormalities in discourse. Inclusion of appropriate comparison groups, such as CHI patients with extrafrontal lesions, and assessment of cognitive functions (e.g., planning, flexibility in reasoning) purportedly

subserved by the frontal lobes would be informative. In this connection, it remains to be seen whether patients with frontal lesions exhibit deficits analogous to inefficient discourse on nonverbal tasks that involve organization of patterns or designs.

Prognosis for Recovery

Reports based on large numbers of servicemen with penetrating missile wounds suggest a favorable prognosis for recovery from aphasia (Mohr et al., 1980; Russell & Espir, 1961). Although the results of these studies suggest more rapid restitution of language than in aphasia secondary to cerebral vascular disease, the young age of the brain-injured servicemen may also be a contributing factor. Recovery of language after missile wounds has been especially rapid and complete in cases of nonfluent, expressive aphasia ("motor aphasia") produced by focal left frontal injury or by lesions situated in the lower part of the rolandic area (Mohr et al., 1980; Russell & Espir, 1961). Although resolution of right-sided weakness generally parallels the recovery of language in dysfluent aphasics, more deficit may persist after restitution of language. Russell and Espir noted that rolandic lesions also frequently resulted in residual sensory defect.

Mohr et al. (1980) observed that over the course of at least 1 year the aphasia produced by left parietal injury evolved into a residual expressive disorder characterized by reduced fluency. In patients with left posterior parietal lesions, Russell and Espir (1961) observed that anomia, alexia, and impaired spelling were the characteristic sequelae. Mohr et al.'s follow-up findings in patients with acute Wernicke's aphasia secondary to left temporal missile injury showed, however, that impaired comprehension persisted in more than three-fourths of these patients.

Studies reporting on the rate of recovery from aphasia after left-hemisphere missile wounds have not been in close agreement. This disparity may reflect differences in the site of injury, selection criteria, assessment techniques, and follow-up interval across the various series. Whereas Mohr et al. contended that most recovery of language occurs within 1 year after injury, Walker and Jablon (1961) observed recovery of language within 9 months after injury in nearly one-third of their cases. By 7 to 8 years postinjury, however, more than one-half of the servicemen who had been aphasic continued to exhibit language disturbance.

The 15-year outcome of nonfluent aphasia resulting from penetrating missile wounds of the left hemisphere was elucidated in a detailed study by Ludlow et al. (1986). The selection criteria included persistence of nonfluent aphasia 6 months after injury and absence of confounding compli-

cations such as bilateral hearing loss. Of the 39 servicemen who returned for follow-up, two-thirds had fully recovered, whereas one-third had residual nonfluent aphasia according to the results of standardized tests, including the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). The conversation of the nonrecovered men was characterized by simple sentences, a high proportion of automatic phrases, and relatively well-preserved retrieval of vocabulary. Comparison of the follow-up CT scans in these groups disclosed that left-hemisphere lesions encroaching on posterior cortex (Wernicke's area and supramarginal gyrus) most clearly separated the nonrecovered (92%) from the recovered (42%) patients. Other lesion sites that distinguished the residual aphasics included the posterior white matter and basal ganglia. In contrast, 77% of both groups had anterior cortical lesions involving Broca's area. Consistent with this lesion localization, right hemiparesis was present in 75% of the nonrecovered patients compared with 36% of the recovered group. Interestingly, 19% of the recovered patients were left-handed prior to injury, whereas none of the patients with residual aphasia were sinistrals. Ludlow and coworkers (1986) postulated that bilateral speech representation in the left-handers enhanced their recovery from nonfluent aphasia. In summary, the findings of these investigators suggest that lesions restricted to the left anterior region portend recovery from nonfluent aphasia (especially in left-handers) following left-hemisphere missile injury.

In a broader study of long-term outcome after missile injury, Newcombe (1969) found that nearly one-third of the Oxford patients with left-hemisphere wounds continued to evidence aphasic symptoms when examined at the time of follow-up. Word fluency, measured by retrieval of items from a given category (e.g., colors), was reduced in the total series of left-hemisphere–injured patients, although the deficit was more severe in patients with residual aphasic symptoms. Defects in vocabulary, reading, spelling, and writing were confined to patients considered to be aphasic. In contrast, more than one-half of the total series (including nonaphasic patients with left-hemisphere wounds) continued to complain of "being at a loss for a word." Residual impairment of verbal memory was found in patients with left-hemisphere injury, including those who were nonaphasic at the time of follow-up. In summary, most patients with left-hemisphere wounds were either rendered aphasic or showed subtle anomic and/or verbal memory impairments.

Investigators only recently have studied the early recovery of communicative skills after prolonged coma in patients with severe CHI. Najenson et al. (1978) plotted recovery curves for communicative skills during the first 18 months after severe CHI in 15 patients with prolonged coma (undefined) who were referred to a rehabilitation unit. The authors developed

TABLE 14.2

Assessment of Communication Functions after Prolonged Coma[a]

Auditory comprehension	*Oral Expression*
Awareness of gross environmental sounds	Voicing
Awareness of speech	Saying vowels
Ability to indicate yes and no	Saying consonants
Understanding own name	Saying own name
Recognition of family names	Saying nouns
Understanding simple verbal orders	Saying verbs
Recognition of names of familiar objects	Saying noun–verb combinations
Recognition of colors	Saying short sentences (automatic)
Recognition of forms	Saying short sentences (nonautomatic)
Understanding use of familiar objects	Conversational speech
Visual comprehension	*Reading*
Awareness of visual stimulation	Reading own name
Understanding gesture direction	Reading family names
Association of identical objects	Reading single words
Association of identical forms	Reading simple sentences
Association fo similar objects	Reading newspaper headlines
Categorization	Reading newspaper articles
Speech	*Writing*
Articulation	Writing own name
Respiration	Writing family names
Voice	Wriring words
	Writing simple sentences
	Writing a letter

[a]From Najenson, Sazbon, Fiselzon, Becker, and Schechter (1978). Reproduced with permission of the authors and publisher.

a scale, shown in Table 14.2, to rate expressive and receptive functions because the patients were unable to cooperate with the standardized tests usually used during later stages of recovery. As shown in Table 14.2, this scale consists of six major functions, and each is divided into specific communicative skills. At evaluation, each specific test is assigned a score ranging from 0 to 4. The total score for each major function is summed and expressed as a percentage of the maximum possible score. Najenson et al. plotted the percentage scores monthly to depict the course of recovery. Six patients in this study remained in a vegetative state, and nine cases had partial or full restitution of language. The authors observed a consistent sequence of recovery. Comprehension of gestures and oral language appeared first, usually between 3 weeks and 5 months after trauma. Oral expression, reading, and writing were slower to recover, and motor defects

in speech (e.g., articulation, respiratory control, and phonation) were often persistently impaired. Of the nine patients who recovered communication, eight had dysarthric speech. The authors observed that the recovery of communicative ability corresponded to progressive improvement in locomotion.

In a study of outcome after severe diffuse CHI (coma longer than 24 hr), Thomsen (1975) administered a follow-up (mean interval = 31 to 33 months after injury) language examination of her own design to 12 patients who had been acutely aphasic. Four patients, including two sinistrals, had no signs of aphasia. In the others, amnestic aphasia (slow rate of speech, slow repetition of words or phrases, verbal paraphasia, and perseveration) was frequently present. Thomsen noted a residual decline in complex verbal skills, such as detailed verbal description and the use of antonyms, synonyms, and metaphors. Impaired reading was found in four cases, but no patient was totally alexic. Although these findings agree with the view that aphasia secondary to CHI has a good prognosis, Thomsen emphasized that residual linguistic defects and dysarthria were present. Moreover, she pointed out that the manifestations of "subclinical" language problems depend on the recovery of memory and general cognitive function.

In a second investigation, Thomsen (1976) reexamined the language of 15 patients who had focal mass lesions (in which temporal lobe damage predominated) or extensive destruction of the left hemisphere and who had been aphasic during the initial hospitalization. When tested at least 1 year after injury (mean interval = 29 months), an overall trend of improvement was evident, although all patients exhibited residual language deficits. The course of recovery was characterized by improved comprehension of oral and written language and less severe agraphia. As in Thomsen's (1975) study of diffuse CHI patients, amnestic aphasia and perseveration persisted in nearly all patients. Global aphasia with gross impairment of all language functions typically evolved into receptive aphasia, whereas patients who initially had a receptive aphasia frequently evidenced improvement in comprehension despite residual anomia. Although Thomsen concluded that nearly all patients with focal left-hemisphere lesions made some improvement, she noted that "half the patients had severe or moderate aphasia two and a half years after the trauma and a few had not been able to pass the level of automatic language (e.g., expletives, stereotyped phrases)" (p. 376).

Groher (1977) administered the Porch Index of Communicative Ability to 14 consecutively admitted, comatose CHI patients at 1-month intervals beginning shortly after termination of coma (mean duration = 17 days). He reported progressive improvement in expressive and receptive skills

over a 4-month period. Naming to confrontation recovered in all patients, whereas spelling errors, incomplete sentence construction, and syntax errors persisted. The degree of recovery suggested by this study appears to be greater than the impression conveyed by other investigators of aphasia after severe CHI. This finding may be attributed to Groher's inclusion of a consecutive series of comatose patients instead of confining the study to acutely aphasic cases.

When consecutive referrals of CHI patients to a rehabilitation program are considered, a different view of outcome emerges which may reflect the severity of injuries requiring intensive retraining. Sarno et al. (1986) described the results of administering the NCCEA to 125 patients after an average postinjury interval of 45 weeks. The series, which included a wide range of coma duration (15 min to 6 months), consisted of CHI produced by motor vehicle accidents in about one-half of the cases.

Consistent with earlier findings (Sarno, 1980, 1984), Sarno et al. reported that language disorder and/or dysarthria were present in all patients. Of interest were the distinctions among classic aphasia ($n = 37$), dysarthria with subclinical aphasia ($n = 43$), and subclinical aphasia ($n = 45$). Sarno et al. diagnosed dysarthria and classic aphasia on the basis of spontaneous speech and comprehension during an interview. The investigators used the term SUBCLINICAL APHASIA DISORDER to refer to evidence of linguistic processing deficits on testing in the absence of clinical manifestations of linguistic impairment. Applying this broad classification, Sarno et al. found that all patients in the series had residual speech or language defects. The aphasic group was older (mean age = 38 years) than the dysarthric (mean = 24 years) and subclinical (mean = 28 years) groups. Right hemiplegia was more common among aphasics, whereas quadriplegia was more frequent in dysarthrics. The quantitative test results obtained by Sarno et al. are illustrated in Figure 14.8, which shows percentile scores based on an aphasic population. The subclinical linguistic deficits involved visual naming, word fluency, and comprehension of multistage commands (Token Test; see Figure 14.8). The findings obtained by Sarno et al. confirmed Thomsen's (1975) findings in showing that subtle language disturbance is common after CHI even in the absence of unequivocal aphasia.

In the course of a long-term study of patients with severe CHI who had been acutely aphasic, Levin et al. (1981) assessed recovery from aphasia in 21 patients. The results of initial CT scans and findings from surgery disclosed evidence of primary left-hemisphere injury in eight cases, focal lesions of the right hemisphere in four cases, bilateral injury in two patients, and diffuse CHI in the remaining patients. The MAE (Benton, 1967) and portions of the aphasia battery of Spreen and Benton (1969) were administered at the time of follow-up. Nine patients fully recovered from acute

aphasia as reflected by uniformly normal scores and intact conversational speech. The 12 patients with residual language deficit (indicated by at least one grossly defective score) were equally divided between cases with a persistent impairment of both expressive and receptive abilities and cases of specific language deficit. Anomia and decreased word finding were the most common isolated defects. Patients who fully recovered from acute aphasia or who exhibited a specific language deficit. Anomia and decreased word finding were the most common isolated defects. Patients who fully recovered from acute aphasia or who exhibited a specific language disturbance at the time of follow-up were generally functioning within the average range of intelligence, whereas patients with generalized language impairment evidenced intellectual deficit on both the Verbal and the Performance scales of the Wechsler Adult Intelligence Scale (WAIS).

In accord with the course of recovery observed in an earlier study by Thomsen (1975), acute global aphasia in this series of patients frequently evolved into a specific anomia. This pattern is illustrated by the serial findings in a 17-year-old student (see Figure 14.6A) who had a left temporal mass lesion and a diffuse injury of mild to moderate severity. She initially exhibited impairment of naming, word association, and alexia, whereas residual language deficit was confined to anomia. In contrast, Figure 14.6B depicts persistent, generalized language defects in an 18-year-old patient who sustained a severe CHI (coma = 21 days) complicated by bilateral frontoparietal subdural hematomas. He evidenced a concomitant decline in cognitive ability, as reflected by a disparity between follow-up results on the WAIS and his high school test scores.

To summarize, the studies of long-term recovery of language after CHI show an overall trend of improvement that may eventuate in restoration of language or in specific defects ("subclinical" language disorder) in naming or word finding in about two-thirds of the patients who are acutely aphasic. Generalized language deficit, which is associated with global cognitive impairment, persists in patients who sustain severe CHI.

Aphasia in Children after Closed-Head Injury

Cerebral Plasticity and Recovery of Function

Investigations of early insult to the human brain have suggested that it possesses considerable plasticity for language development. Lenneberg (1967) proposed that recovery from aphasia parallels the degree to which

specialization for language has become lateralized, that is, the commitment of the left hemisphere. From observations of aphasia in children, he postulated that cerebral dominance is established from 2 to 10 years of age. Young children would thus be able to displace language to the right hemisphere after injury to the left hemisphere. This postulation is also consistent with the observation that a right-hemisphere lesion more frequently produces aphasia in young children than in older patients. In contrast to evidence that the development of hemispheric dominance may be a gradual process beginning at age 2 (Woods & Carey, 1979), more recent research has documented that left-hemisphere lesions in the postnatal period result in subtle, persisting deficits in object naming and auditory language comprehension (Vargha-Khadem, O'Gorman, & Watters, 1985). In addition to interhemispheric plasticity, Hécaen (1976) suggested that intrahemispheric reorganization may subserve the rapid recovery from expressive aphasia in children. Studies of infantile hemiplegia have shown no evidence of residual aphasia and only transient or mild aphasia when hemispherectomy is performed on young children with hemiplegia who have acquired language prior to surgery (Basser, 1962; Teuber, 1978). The integrity of language after early brain injury may be achieved, however, at the expense of functions primarily subserved by the right hemisphere (Teuber, 1978). Chapter 13 contains a more detailed discussion of the relationship between brain maturation and recovery from aphasia.

The impression of greater capacity for recovery from CHI in children has received some support from a study at the Children's Hospital in Philadelphia that showed a mortality of only 6% in cases with a severe injury defined by an initial GCS score of 8 or less (Bruce et al., 1979). Although Bruce et al. reported that 90% of the children achieved a good recovery or were only moderately disabled, more detailed studies employing psychological assessment have documented permanent cognitive deficit in more than one-third of children who sustain severe head injuries (Brink, Garrett, Hale, Woo-Sam, & Nickel, 1970). Thus, it may be anticipated that cognitive impairment contributes to the clinical picture of children who are referred for assessment of aphasia after severe CHI.

Description and Clinical Course of Aphasia after Brain Injury in Children

Guttmann (1942) published one of the earliest systematic studies of aphasia in children. In his review of previous descriptions, he commented that childhood aphasia was historically viewed as a congenital disorder, whereas acquired aphasia was less well known. The author attributed the prevailing concept of acquired aphasia as a rare disorder in children to a

lack of familiarity with its distinctive features. In a series of 30 children (2 to 14 years of age) that included nine cases of head injury, Guttmann pointed out that his patients generally did not exhibit obvious paraphasic errors and were less likely than adults to complain of difficulty in speech. Consequently, Guttmann suggested that acquired aphasia in children may be overlooked or misinterpreted as an unwillingness to speak. Fourteen of the 16 children with left-hemisphere lesions, 1 of the 13 with right-hemisphere lesions, and 1 child with bilateral injury were aphasic. The author characterized the children's initial language disturbance as an absence of spontaneous speech, followed by a poverty of expression, hesitancy, and dysarthria when speech returned. In view of incomplete follow-up data and technical limitations, Guttmann refrained from definitive statements regarding prognosis or localization of lesion.

Alajouanine and Lhermitte (1965) studied 32 aphasic children with left-hemisphere lesions, including 13 patients with head injury who ranged in age from 6 to 15 years of age. Consistent with Guttmann's (1942) earlier observations, the most striking feature of the aphasic disorder was poverty of expression, including oral and written language and gestures. Dysarthria, which was present in two-thirds of the children, was associated with hemiplegia. The authors found that, in contrast to aphasia in adults, fluent paraphasic speech was rare and perseveration was absent even in children with temporal lesions. The seven children with paraphasic errors were older than 10 years of age. In agreement with Guttmann's findings, only one-third of the children had a receptive deficit for oral speech, although nearly two-thirds were alexic. Comparison of the aphasic children who were under 10 years of age with those from 10 to 15 years of age disclosed that the younger patients had a more profound reduction of verbal expression and more consistent disruption of articulation. Follow-up examinations at 6 and 12 months after the appearance of aphasia disclosed normal or nearly normal language in 22 and 24 children, respectively. The authors observed subtle alterations of language in 14 children on tests of narration, construction of sentences, and definition of words.

Hécaen (1976) described 26 children ($3\frac{1}{2}$ to 15 years of age) with focal cortical lesions of whom 16 (7 left, 6 right, 3 bilateral) sustained a CHI. Nineteen children were aphasic, including 88% of those with left-hemisphere lesions and 33% of those with right-hemisphere lesions. Consistent with previous reports of acquired aphasia in children, Hécaen considered the two essential features of language disorder in his patients to be the loss of initiation of speech (if not mutism) and the absence of paraphasia. Articulation disorder was also common. Impaired auditory verbal comprehension was found only in children with temporal lobe lesions. Alexia resolved rapidly, whereas disturbance of writing was the most common

symptom during the acute period and the least likely to resolve. Acalculia was also frequently present in the aphasic children. Although Hécaen's findings lend support to the impression of more rapid resolution of aphasia in children, only one-third of his cases recovered fully. Although Hécaen's series confirmed the widely held view that aphasia in children is nonfluent, recent case reports have documented fluent aphasia when the focal lesion encroaches on Wernicke's area (van Dongen, Loonen, & van Dongen, 1985).

Insofar as Hécaen investigated acquired aphasia, he confined his study to children who had developed language prior to injury (the youngest aphasic in Hécaen's series was $3\frac{1}{2}$ years of age). We had the opportunity, however, to study serially the recovery of a $2\frac{1}{2}$-year-old girl in whom language development was advanced (e.g., she spoke in sentences, recited the alphabet, and wrote her name prior to injury) and a decided right-hand preference had been established. The child sustained a CHI in a motor vehicle accident on 20 March. When examined in the emergency room shortly after injury, the GCS score was 6; she had a right hemiparesis and a third-nerve palsy. An angiogram showed no evidence of mass lesion. She followed commands after 10 days, but remained mute. The patient gradually vocalized and uttered the *b* sound on 9 April when shown the alphabet. She verbalized single words and brief phrases and joined in singing but failed to initiate speech. Clinic visits over the subsequent 5 years disclosed progressive improvement in language to a nearly normal level within 1 year after injury. During the 6 months after injury, the only residual noted in a speech evaluation was a slight hesitancy. The patient shifted her manual preference to the left hand and left foot, although strength on her right side was greatly improved.

Studies of pediatric head trauma have focused on quantitative assessment of language (Ewing-Cobbs, Levin, Eisenberg, & Fletcher, 1987; Levin & Eisenberg, 1979). Levin and Eisenberg administered the NCCEA to a consecutive series of children (6 to 12 years of age) and 42 adolescents (13 to 18 years of age) after they regained orientation. No child was mute or unresponsive at the time of testing. The series comprised 60% mild injuries (no coma) and 40% with more severe CHI (coma duration from 1 hr to more than 3 weeks). The results showed that residual language defect was present in about one-third of the patients. Consistent with findings in adults with CHI, anomia was the most prominent deficit and verbal repetition was least affected. Comprehension of oral language was impaired in 11% of the children studied. More recently, Ewing-Cobbs et al. (1987) reported evidence for greater vulnerability of linguistic functions that were in a phase of rapid development at the time of injury. Findings obtained about 1 month postinjury disclosed that writing to dictation and copying were

more impaired in children than in adolescents who had sustained CHI of comparable severity. In general, naming, other expressive functions, and writing were more sensitive to severity of injury than was receptive ability. Together these results suggest that subclinical language disorder occurs after CHI in children with a frequency comparable to that found in adults.

Discourse in Children with TBI

As in adult TBI populations, discourse measures are more sensitive to the long-term sequelae in pediatric populations than are traditional measures of the formal linguisitic domains (Biddle et al., 1996; Chapman, 1997; Chapman, Levin & Lawyer, 1998; Ylvisaker, 1993). The majority of studies have identified significant discourse impairments in children with severe brain injuries compared with normal control children or even children with mild brain injuries (Biddle et al., 1996; Chapman et al., 1992; Chapman, Levin, Matejka, Harward, & Kufera, 1995; Chapman, Watkins et al., 1997). Although there is considerable individual variation in discourse disability among children after TBI, depending on variables such as age at time of injury or severity of injury, to mention a few, studies have identified certain common disruptions. The most consistent finding is that measures of amount and organization of information are more likely to reveal the specific difficulties associated with TBI than are measures of amount and complexity of language comprising the discourse sample (Chapman et al., 1995; Chapman, Watkins et al., 1997). The most commonly occurring performance deficits revealed by discourse information measures include (a) omission of important information, (b) disorganized structure, and (c) inability to paraphrase the information at a higher level of interpretation (Biddle et al., 1996; Chapman et al., 1992, 1995; Chapman, Watkins et al., 1997). Narrative discourse is more problematic than conversational discourse for pediatric brain-injured populations, probably because of the larger units of information that must be manipulated in narrative discourse.

As one might predict, written discourse is even more vulnerable than oral discourse to the long-term effects of pediatric TBI (Chapman & Lawyer, 1997; Ewing-Cobbs et al., 1987; Yorkston, Jaffe, Polissar, Liao, & Fay, in press). Children who sustain a severe TBI omit important core information and fail to produce complete episodes compared with their verbal discourse (Chapman & Lawyer, 1997). The differences between the oral and written modalities are notable, given that the written stories were always elicited after the oral version which placed the burden of formulation constraints on the oral version.

The converging evidence indicates that discourse deficits are likely to

persist for years after sustaining a severe TBI despite the relatively favorable prognosis for recovery of specific linguistic functions. Nonetheless, it is important not to ignore the possibility of specific language deficits in pediatric brain-injured children. Clearly, some children with moderate to severe TBI can exhibit deficits in the lexical and grammatical domains of language. Chapman, Watkins, and coworkers (1997) found that 75% of children with moderate to severe brain injury experience discourse disability. One-third of the children in this study showed significant deficits in the structured language measures.

The relationship between frontal injuries and discourse impairments in pediatric brain-injured populations has been examined by our research group. Frontal lobe injuries were found to contribute to the severity of discourse impairment and to the nature of the discourse disturbance (Chapman, 1995; Chapman et al., 1992; Chapman, Levin, Wanek, Weyrauch, & Kufera, 1998). Children with relatively large frontal lobe injuries after CHI manifested greater discourse impairments than did a group with comparable injury severity without injuries to the frontal regions. With regard to nature of impairment, children with left frontal lesions showed a divergent pattern from children with primarily right frontal lesions. Whereas children with left frontal lesions produced simplified narratives at both sentential and discourse levels, children with right frontal lesions showed a reduction at a discourse level only.

In summary, the results verify that traditional language measures are insufficient to characterize the full range of cognitive–communication deficits common to both adult and pediatric TBI populations. In contrast, discourse is likely to remain impaired after severe TBI. Longitudinal studies verify the persistence of discourse disturbances (Chapman, Levin, & Harward, 1996; Chapman et al., 1995). In children, the discourse deficits may directly contribute to academic failure and poor peer reintegration (Chapman, Levin, & Lawyer, 1998; Chapman et al., 1995).

Effects of Severity of Injury

Severity of Diffuse Injury

The duration of coma and persistence of PTA are widely viewed as indices of diffuse injury severity. The divergence in findings on the effects of duration of coma on language may reflect differences in both the definition of coma and the interval between injury and assessment. Brooks, Aughton, Bond, Jones, and Rizvi (1980) defined duration of coma in reference to the

GCS: no eye opening, failure to obey commands, and absence of comprehensible speech. In contrast, we used the interval during which the patient was unable to follow commands (Levin et al., 1976).

Most investigators have reported no relationship between duration of coma and severity or persistence of language disturbance (Brooks et al., 1980; de Morsier, 1973; Groher, 1977; Sarno, 1980, 1984; Sarno et al., 1986). The data of patients with mass lesions, however, are typically merged with data of diffuse CHI cases. We reported significant correlations between duration of coma and test scores on visual naming, word fluency, comprehension of aural language, and reading comprehension. Review of individual scores on scatter plots disclosed cases with brief coma and a patient with a left-hemisphere mass lesion who evidenced language disturbance. The correlations were nonsignificant on other subtests of the MAE. In a follow-up study of long-term recovery (median interval, 1 year) of language, we confirmed that persistent impairment of both expressive and receptive abilities was related to a prolonged period of coma (Levin et al., 1981). Brooks et al. (1980) found that residual word fluency was correlated with the duration of PTA, which was estimated by retrospectively questioning the patient.

Severe diffuse CHI is frequently presumed in patients with a long period of coma in the face of a normal CT scan. Severity of generalized injury may also be reflected by an acute CT scan showing compression of the ventricles and cisterns by cerebral swelling. The possibility of differential recovery of language in patients with this CT pattern has not been studied. In summary, we may conclude that prolonged coma is neither a necessary nor a sufficient condition for residual aphasia.

Focal Brain Lesions

Localization of lesion in aphasia is reviewed by Damasio in Chapter 3. This section provides a cursory summary of findings in patients with head injury. In acute CHI the presence of a focal mass lesion (i.e., hematoma or contusion) is visualized in most cases by a CT scan. Figure 14.2, a CT scan obtained on the day of injury, shows an area of increased density consistent with an intracerebral hematoma. Smaller lesions, such as hemorrhagic contusions, are also identified by CT. Although magnetic resonance imaging can detect intracranial lesions not seen on CT, it frequently requires a longer scanning interval and is more sensitive to artifact produced by the patient's movement. Moreover, bringing conventional life support equipment into a magnetic field can pose problems.

In general, the type of aphasia associated with a specific locus of lesion corresponds to the language disorder produced by nontraumatic vascular

lesions in the same region (Alajouanine et al., 1957). This generalization has received support from studies of adults with left temporal intracerebral hematoma who exhibit fluent, paraphasic speech and impaired comprehension (Debray-Ritzen, Hirsch, Pierre-Kahn, Bursztejn, & Labbé, 1977; Stone et al., 1978). Injury to the dominant temporal lobe or extensive left-hemisphere damage accounted for most of the aphasic CHI patients with focal lesions described by Thomsen (1976). The presence of a focal lesion concomitant with diffuse injury may result in an apparent "crossed aphasia." From neurologic findings, CT results, and observations made during surgery, we recorded six patients with predominant right-hemisphere lesions who exhibited a linguistic defect on at least one subtest of the MAE (Levin et al., 1976). In contrast to aphasics with major involvement of the left hemisphere, contralateral hemiparesis was not present in these patients. We also observed that clinical evidence of injury to the rostral brain stem was more closely associated with linguistic disturbance than were signs of hemispheric injury, a finding consistent with Ommaya and Gennarelli's (1974) model of CHI discussed earlier in this chapter.

Subcortical Lesions

Evidence for the participation of subcortical structures in language is reviewed in Chapter 3. We have been impressed by the long periods of mutism in three patients, two adolescents and an adult, in whom CT disclosed a subcortical intracerebral hematoma. The findings in one patient are summarized (see Figure 14.2) here.

A 12-year-old, left-handed girl (inconsistent familial sinistrality) was evaluated 20 min after sustaining a CHI on 22 June when she was struck by a car. Examination in the emergency room disclosed a GCS of 6. She began to obey commands on 30 June but had a hemiplegia and uttered no words or sounds. The CT scan disclosed a hemorrhagic contusion of the left putamen and anterior limb of the internal capsule (Figure 14.2). She remained mute until 15 July. Throughout the mute period she was dysgraphic, her spelling was impaired, and she wrote in block letters. A repeat CT scan showed density changes that suggested resolution of the hemorrhagic lesion in the basal ganglia. Detailed assessment of language on 13 August when she was fully oriented disclosed an impoverished lexical stock with infrequent initiation of spontaneous speech. The MAE showed a decrement in verbal associative fluency and defective comprehension (Token Test). Spontaneous speech was grossly intact when she was examined 1 year later. The follow-up tests disclosed a residual impairment in word fluency and impaired comprehension of complex commands (Token Test), although there was a trend of improvement.

Concomitant Neuropsychological Deficits

Hemispheric Disconnection Syndrome

In view of the shearing and stretching of axons and resultant injury to the corpus callosum, hemispheric disconnection syndrome would appear to be a likely consequence of severe CHI. The first description of a hemispheric disconnection after CHI was the case reported by Lhermitte, de Massary, and Huguenin (1929) of a jockey who developed alexia without agraphia after falling off a horse. He had a right homonymous hemianopsia and a left inferior quadrantopsia. The patient could not read words but was capable of reading single letters. The second case study of hemispheric disconnection after CHI was published 40 years later (Schott, Michel, Michel, & Dumas, 1969), but there was no anatomic confirmation of injury to the callosum. The authors described tactile anomia, ideomotor apraxia, and agraphia confined to the left hand. A case of posttraumatic disconnection syndrome with neuropathologic verification was found to evidence ideomotor apraxia and agraphia confined to the left hand (Rubens, Geschwind, Mahowald, & Mastri, 1977). Neuropathologic findings showed marked thinning of the corpus callosum with demyelination and loss of axons.

We tested interhemispheric transfer of information by comparing naming of objects placed in the left or the right hand in a study of long-term recovery from acute aphasia following CHI (Levin et al., 1981). There was disproportionate impairment of naming objects placed in the left hand, a finding consistent with callosal dysfunction. Two cases with residual expressive and receptive deficits also exhibited ideomotor apraxia, as the patients could not do better than approximating familiar gestures when requested orally to perform them. We postulated that ideomotor apraxia in these patients may have resulted from interruption of intrahemispheric connections.

The presence of hemispheric disconnection can be clinically investigated by detailed assessment of ideomotor apraxia, testing writing in both hands, and examining the patient's ability to name objects placed in either hand. It is necessary to exclude the possibility of a primary sensory defect by determining whether the patient can visually match objects that are incorrectly named.

To investigate posttraumatic hemisphere disconnection effects, Levin et al. (1989) administered dichotic listening and intermanual tests to 69 patients who had sustained CHIs of varying severity. Manual tests included naming objects palpated in either hand, transfer of postures from one hand to the other, and writing. Consistent with a prediction of decreased effi-

ciency of interhemispheric transfer of information, the degree of ear asymmetry in dichotic listening performance was directly related to the severity of head injury as reflected by impaired consciousness. Suppression of responses to left-ear input was the most characteristic abnormality in severely injured patients, despite preservation of hearing. Depth and localization of parenchymal lesions characterized by magnetic resonance imaging were also related to the degree of ear asymmetry. Lesions situated in sites that could potentially interfere with callosal auditory or geniculocortical pathways produced a greater disparity in response to left- versus right-ear input than did parenchymal lesions in areas such as the frontal lobes. Striking dissociation between marked asymmetry in dichotic listening performance and unilateral deficits on the intermanual tests was exhibited by individual patients. These results provide further evidence for the effects of multifocal brain lesions involving the white matter on tests that require intra- and/or interhemispheric integration.

Disturbances in Cognitive Abilities of Executive Function

Disturbances in cognitive abilities of executive function are common sequelae of TBI in both adult and pediatric populations, particularly when the injury compromises the prefrontal network (Coelho et al., 1995; Levin, Goldstein, Williams, & Eisenberg, 1991; Sohlberg & Mateer, 1989; Ylvisaker & Fenney, 1996). These executive functions include cognitive capacities involved in (a) attainment of a future goal such as the goal setting, planning and problem solving necessary for completing a project, (b) self-awareness and self-regulation, and (c) self-inhibition (Pennington, 1991). These cognitive abilities may be associated with or contribute to the deficits in higher-level discourse processing in children (Chapman, Levin & Lawyer, 1998; Ylvisaker & Fenney, 1996) and in adults (Coelho et al., 1995; Hartley, 1995; MacDonald & Johnson, 1996). It is necessary to consider the relationship between discourse function and cognitive abilities of executive control to manage effectively the cognitive–communicative disability manifested in TBI.

Other Neuropsychological Deficits

Descriptive studies after CHI have emphasized the frequent finding of concomitant neuropsychological deficits. Associated verbal impairment, which may be viewed as an integral aspect of aphasia, includes alexia, agraphia, and acalculia (de Morsier, 1973; Heilman et al., 1971). Heilman et al. observed that anomic patients frequently exhibit right–left confusion,

finger agnosia, and difficulties in calculation, writing, and reading. The authors also found reversible amnesic disorder in four of the nine anomic patients.

Thomsen (1977) compared verbal learning and memory for words, sentences, and numbers in CHI patients with persistent aphasic symptoms and in nonaphasic CHI cases who had comparable duration of PTA. This study, which tested patients more than 2 years postinjury, disclosed that both groups had impaired verbal memory for information beyond immediate span when their performance was compared with that of a control group. Residual impairment of immediate memory (e.g., digit span) was confined to the aphasic CHI patients, whose learning and retention of unrelated words were inferior to those of nonaphasic head-injured patients.

In a quantitative study of language defects after CHI, we considered related deficits on visuoperceptive and visuomotor tasks (Levin et al., 1976). Although we found a trend suggesting an association, it was not significant when these patients were compared with other CHI patients who were spared linguistic disturbance.

Historically, the relationship between aphasia, of any etiology, and intellectual function has been a controversial issue. We approached the issue by differentiating acutely aphasic CHI patients whose language fully recovered from patients with specific residual defects or chronic impairment of both expressive and receptive abilities (Levin et al., 1981). Patients with persistent aphasic disorder had marked cognitive deficit on both verbal and visuospatial subtests of the WAIS, whereas the other patients recovered to a low normal or average intellectual level.

Behavioral Disturbances

The sequelae of CHI frequently include alterations in behavior (see Levin & Grossman, 1978). Severe head injury may result in thinking disturbances reflected by intrusion of irrelevant material into spontaneous speech. Patients disabled by CHI often lack insight into the severity of their deficits and the inappropriateness of their verbalizations. Motor retardation and withdrawal to isolated activities are also common in these patients. To a lesser degree, depression and anxiety may be present and affect the course of speech therapy, although these sequelae are not closely related to the severity of initial injury. Studies that have employed CT to localize focal brain lesions secondary to vascular disease or trauma have indicated that depression is more severe following left-hemisphere insult (Robinson & Szetela, 1981). The program of research by Robinson and his coworkers has included CHI and stroke patients. Analysis of the relationship between type of aphasia and affective change has shown that nonfluent aphasics are more depressed than fluent and global aphasics, accord-

ing to self-report and ratings by clinicians (Robinson & Benson, 1981). Whether this vulnerability to depression in nonfluent aphasics is attributable to closer proximity of their left-hemisphere brain lesions to the frontal pole, interruption of catecholaminergic pathways, or greater self-awareness of deficit in these patients awaits further study.

Special Aspects of Speech–Language Management

The general topic of speech-language management for aphasia is discussed by M. T. Sarno in Chapter 16. Although the techniques for remediation of linguistic disturbances after head injury may not differ fundamentally from the methods used for aphasics with cerebral vascular disease, the speech–language pathologist should be particularly sensitive to the unique disturbances in communicative function following TBI.

With regard to assessment, a comprehensive evaluation should incorporate a battery of standardized language measures as well as measures of discourse function. Traditional language measures will allow identification of disturbances in the formal linguistic domains of phonology, semantics, and syntax, although these disturbances represent only a small proportion of the more pervasive cognitive–communicative disturbances. It is now widely recognized that the cognitive–communicative deficits associated with TBI are not readily detectable by structured language measures (Chapman, 1997; Hartley & Jensen, 1991; Ylvisaker & Szekeres, 1989). The application of discourse methods in both the oral and written modalities is essential to characterize the full scope of the disturbances associated with severe TBI in manipulating larger units of language (Chapman, Levin, & Lawyer, 1998). For children, it is important to recognize that the disability consequent to severe TBI is not fully discernible at any single stage postinjury. Although children show relatively favorable prognosis for recovery of previously established functions, skills in the process of development at the time of injury or later developing abilities may be at greater risk for inadequate or deficient acquisition (Chapman et al., 1996; Fletcher, Miner, & Ewing-Cobbs, 1987).

With regard to treatment, the speech–language pathologist should assume a prominent role in addressing the neurolinguistic, neuropsychological, and behavioral deficits after TBI in adult and pediatric populations. In particular, the application of techniques for manipulating discourse content at more global levels of interpretation may prove beneficial for aiding the patient in applying specific cognitive strategies to process and organize new information (Borkowski & Kurtz, 1987; Chapman, Levin, & Lawyer, 1998; Kay & Black, 1986; Ylvisaker & Szekeres, 1989). Providing feedback

to assist the head-injury patient in monitoring linguistic errors and appropriateness of content may facilitate psychosocial functioning. The intrusion of irrelevant and unrealistic statements into the conversational speech of brain-injured patients has been mentioned by various authors (see Levin & Grossman, 1978). It is possible the TBI clients can show improved ability to monitor their discourse content. Intervention should be directed at helping TBI patients plan, monitor, and revise oral and written discourse as relevant to the context of their daily lives.

Summary

The considerable heterogeneity in the mechanisms of traumatic brain injury produced by penetrating missile wounds and closed head injury is reflected by differences in cognitive–communicative symptoms. Nonfluent expressive aphasia is common in patients with left-hemisphere missile injury, but is rarely found in adults with closed head injury. Anomic disturbance predominates in clinically obvious aphasia after closed head injury and may be demonstrated by appropriate testing in patients with relatively intact spontaneous speech. In contrast, acquired aphasia produced by closed head injury in children is characterized by a reduction in output with hesitancy, failure to initiate speech, and possibly mutism. Although the prognosis for recovery from specific language disturbances is generally better in patients with closed head injury than in patients with cerebral vascular disease, persistent deficits in complex verbal skills are likely. Patients with closed head injuries frequently exhibit concomitant neuropsychological deficits, including disturbances in executive functioning, hemispheric disconnection syndrome, verbal memory impairment, and acalculia.

Acknowledgments

Preparation of this manuscript and the authors' research were supported by grant NS 21889. The authors are indebted to A. L. Benton for providing valuable advice and reviewing the manuscript, and to Jennifer Nasits for assistance in manuscript preparation.

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15

Acquired Aphasia in Bilingual Speakers

MICHEL PARADIS

Patterns of Recovery

Most of the literature on bilingual aphasia over the past 100 years has focused on patterns of recovery (see Paradis, 1983, for English translations of papers that appeared in any language between 1843 and 1975; for cases between 1975 and 1988, see Paradis, 1989). Pitres (1895) pointed out that patients did not always lose the use of the languages that they spoke before insult to the same extent, nor did they necessarily regain access to them simultaneously. Although some patients do indeed recover all of their languages to the same degree and at the same time (PARALLEL RECOVERY), some languages may never be recovered (SELECTIVE RECOVERY). A patient may regain only one language at first, then, sometime—possibly months—later, the other or others when the first recovered language has been maximally recovered (SUCCESSIVE RECOVERY). One language may be recovered better than the other, irrespective of the relative degree of premorbid proficiency (DIFFERENTIAL RECOVERY).

Other patterns have also been reported: ANTAGONISTIC RECOVERY, where one language regresses while the other progresses (Chlenov, 1948; Minkowski, 1928, 1933; Paradis & Goldblum, 1989; Shubert, 1940; Wald, 1961; Winterstein & Meier, 1939; Zaorski, 1952); ALTERNATING ANTAGONISM (previously erroneously referred to as alternate antagonism from an infelicitous translation of French *alternatif,* as in *courant alternatif,* in contrast to direct or continuous current, giving the idea of a back and forth or seesaw effect), where exclusive availability alternates between one and then the

other language (Nilipour & Ashayeri, 1989; Paradis, Goldblum & Abidi, 1982); and BLENDING (previously more ambiguously called mixed) recovery, where patients involuntarily blend their two languages, at any level of linguistic structure, for example, speaking one language with the accent of the other, using bound affixes of one language on word stems in the other, blending together monomorphemic words of two languages, or generally violating grammatical code-mixing constraints (Gloning & Gloning, 1965; Halpern, 1941; Herschmann & Pötzl, 1920; Kauders, 1929; Lambert & Fillenbaum, 1959; Ledinsky & Mracek, 1958; Leischner, 1943; L'Hermitte, Hécaen, Dubois, Culioli, & Tabouret-Keller, 1966; Minkowski, 1964; Perecman, 1984; Pötzl, 1925; Stengel & Zelmanowicz, 1933; Wald, 1961; Weisenburg & McBride, 1935). These patterns are not mutually exclusive. Various combinations of languages may simultaneously exhibit different patterns (e.g., two languages may be recovered in parallel with a third selectively lost) or the pattern may change over time (e.g., evolve from alternating antagonism to differential recovery).

More recently, a case of SELECTIVE APHASIA (clear signs of aphasia in one language and no measurable deficits in another) was reported (Paradis & Goldblum, 1989). This condition may have been overlooked in the past because it was assumed not to be possible to have aphasic symptoms in one language but not in another. Anecdotal evidence for selective aphasia can possibly be found even in some cases cited by Pitres, such as those discussed by Lordat (1843), who described a priest who could barely express himself in French, "yet could say whatever he pleased in the Languedoc Patois" (p. 26), and by Winslow (1868), who referred to a 48-year-old woman who seemed to have lost all knowledge of French, "yet she understood English very well and spoke it with no difficulty" (p. 27). There may possibly be a few others, by implication, as in the reference to Bourdin (1877), mention is made only of French (the patient's native language) in which she "was suddenly unable to express herself" (p. 27) without indicating whether there were aphasic symptoms in her English; the same goes for Ore's (1878) patient who "was absolutely unable to understand or speak French, although she could understand and speak patois," and Charcot's (1887) patient who suddenly lost the ability to express himself in French or German, though he could still converse in Russian. Pitres does not specify whether Oré's and Charcot's patients exhibited any aphasic symptoms in their available language.

Three cases of differential aphasia (i.e., a different syndrome in each of a patient's languages) have also been described in the literature. In one patient (Silverberg & Gordon, 1979), the authors diagnosed conduction aphasia in his native language and global aphasia in his second language. Given that the patient spoke the second language only poorly before insult (he

was a Russian-speaking medical doctor who had failed the Hebrew course twice and needed an interpreter when examining patients), it may not be surprising that, after a stroke, he lost the little Hebrew he had managed to acquire. This case could thus easily be described as a selective recovery of the native language. The other two patients (Albert & Obler, 1978; Silverberg & Gordon, 1979) were diagnosed as exhibiting Broca's aphasia in their native language and Wernicke's aphasia in Hebrew (on the basis of the presence of paragrammatism and relatively poorer comprehension). In Hebrew, by virtue of its morphophonological structure, agrammatism is manifested by substitution rather than by omission of bound grammatical morphemes (a traditional characteristic of paragrammatism in English). Thus, given that the patients were very likely to be less proficient in Hebrew premorbidly, it is not unlikely that, in fact, they were exhibiting agrammatism in both languages, albeit with a different characteristic manifestation in each, as well as with poorer comprehension in their weaker second language.

Various factors have been proposed to explain patterns of nonparallel recovery. Ribot (1881), in support of his theory that earlier memories are less vulnerable than more recent ones, briefly alluded to the alleged fact that patients recover their native language better than subsequently learned ones. Pitres (1895) described cases that did not support Ribot's claim and proposed that the language first or best recovered was not the first learned but the most familiar to the patient at the time of insult (which often happens to coincide with the native language). To this day (Heinemann & Assion, 1996; Junqué, Vendrell, & Vendrell, 1995; Obler, Centeno, & Eng, 1995), most papers dealing with bilingual aphasia refer to Ribot's or Pitres's "law," offering either support or contradiction. Minkowski (1928) proposed that the language that is best recovered is the language with the strongest affective ties. In a similar vein, Goldstein (1948) proposed that the language of the environment, of greatest value to the patient, would be best recovered. But none of the foregoing factors are confirmed in more than half the reports published to date. Many patients have been described who recovered a language that was not their native language, nor their most familiar, nor their most favored (Paradis, 1977).

In the 1970s, interest shifted to the question of differential lateralization of language in bilinguals as compared to unilinguals. On the basis of experimental psycholinguistic evidence (mainly dichotic listening and tachistoscopic presentation in visual half fields), bilinguals were suspected of having their languages less asymmetrically represented in the cerebral hemispheres than do unilinguals. It was then hypothesized that differential recovery might be a consequence of one of the languages being less lateralized, and hence less affected by a left-hemisphere lesion. But for

each study that reported a difference in lateralization, another study reported no difference between unilingual and bilingual experimental subjects; the bilingual population alleged to differ grew more and more specific, from all bilinguals to late bilinguals, to late bilinguals only in the early stages of acquiring a second language informally. Some studies, on the other hand, reported decreasing asymmetry as students became more proficient in the second language learned formally. Such contradictory results were often attributed to subtle differences in methodology (Obler, Zatorre, Galloway, & Vaid, 1982; Vaid, 1983). In fact, it is more likely that those various experimental paradigms were simply not valid (Colbourn, 1978; Paradis, 1990, 1992, 1995; Satz, 1977). A recent meta-analysis of the experimental findings (Vaid & Hall, 1991) failed to find any difference. Furthermore, there is no clinical evidence that would provide support for such a conjecture. The grammar of bilinguals is represented in the classical "language areas" of the left hemisphere to the same extent as in unilinguals, as substantiated by electrical cerebral stimulation studies (Ojemann, 1983; Ojemann & Whitaker, 1978; Rapport, Tan, & Whitaker, 1983), sodium amytal testing (Berthier, Starkstein, Lylyk, & Leiguarda, 1990; Rapport et al., 1983), and the incidence of crossed aphasia (Chary, 1986; Karanth & Rangamani, 1988). Thus, in spite of numerous erroneous reports to the contrary (Solin, 1989), there is no greater probability of aphasia subsequent to right hemisphere damage in the bilingual than in the unilingual population.

Another modern attempt at explaining differential and selective recovery has been the suggestion that native and second languages are associated with distinct cortical areas (Berthier et al., 1990; Gomez-Tortosa, Martin, Gaviria, Charbel, & Ausman, 1995; Kim, Relkin, Lee, & Hirsch, 1997; Ojemann, 1991; Ojemann & Whitaker, 1978; Perani et al., 1996; Pulvermüller & Schumann, 1994; Rapport et al., 1983). In fact, some researchers have proposed the very conjecture suggested by Scoresby-Jackson (1867), against which Pitres (1895) argued so forcefully, namely that representations of languages are developed in Broca's area by exposure early in life (and hence this area is no longer available later) and that a second language learned as an adult would therefore have to utilize adjacent cortical areas (Kim et al., 1997). On the basis of two cortical electrical stimulations in which the area where naming of objects in the second language was altered proved substantially larger than the area where naming of the same objects in the more competent language was altered, Ojemann (1983) suggested that the cortical area subserving a language is inversely proportional to its degree of proficiency, with the less proficient language thus requiring more space than the more proficient. On the other hand, Perani et al.'s (1996) positron emission study of nine subjects found the reverse, namely, that a large set of cortical areas was significantly more active for the more proficient native lan-

guage. There is an increasing amount of evidence (Perani et al., 1996; Weber-Fox & Neville, 1996) that languages learned later are processed differently from languages acquired early. They appear to engage more conscious mechanisms, leading to an increased use of declarative memory and a reduced use of implicit memory compared to the native language.

The Role of Implicit Memory

Implicit linguistic competence, the underlying grammatical system whose existence linguists infer from the systematic verbal behavior of speakers, is acquired incidentally, stored without awareness of its form, and used automatically (i.e., without conscious control). It is acquired incidentally by focusing attention on aspects of perceived utterances other than those that are internalized, that is, that become part of the implicit underlying procedures that allow speakers to understand and produce sentences. For example, children (as well as adult learners of a second language) pay attention to the acoustic properties of the sounds of the language they are exposed to, and acquire the proprioception that allows them to reproduce these sounds—without being aware of the phonatory–articulatory processes that are engaged. (No learner is aware of the position or degree of contraction of each of the 100 or so muscles involved, including that of the tongue relative to the teeth and the palate.) Similarly, they pay attention to the meaning of utterances, not to their underlying structure, for the simple reason that the underlying structure which they internalize is not there to be seen, let alone be paid attention. In fact, the procedures that allow speakers to understand and produce sentences remain forever opaque to introspection (as evidenced by the numerous competing theories of the underlying structure of language over the past 40 years—from structural, transformational, tagmemic, stratificational, and government-binding–inspired grammars to the minimalist program). Speakers are unaware of the form of the grammar that allows them to generate the sentences of their language—or indeed whether their ability to produce and understand sentences is subserved by a set of rules (as described in grammars) or by parallel distributed processing (PDP) through network connections established and modified by experience on the basis of statistical probability (as described in artificial intelligence models). These procedures are implicit and are used autonomically, that is, without conscious control (like the heartbeat or kidney functions). This implicit linguistic competence, subserved by circumscribed cortical areas and subcortical structures of the left cerebral hemisphere, is what is impaired in aphasia. Individuals with lesions in these areas exhibit deficits in phonol-

ogy, morphology, and syntax and/or semantics, in comprehension and/or production.

Native languages are normally acquired in this implicit way, whether the child acquires one or more languages during the normal language acquisition period, from birth to about 6 years of age. After this period, vocabulary may increase but the basic structures of the language have been internalized, save for some rare constructions, such as the reversible negative passive that may be acquired as late as age 9 by speakers of English. Vocabulary, on the other hand, will continue to be acquired throughout the speaker's life.

Second-language learners, who are exposed to a new language in a classroom situation, in addition to acquiring some linguistic competence through practice in conversational settings, consciously memorize a considerable body of metalinguistic knowledge, which, contrary to implicit linguistic competence, is learned consciously by focusing on the very items that are consigned to memory, stored explicitly, and available for conscious recall. Its use is consciously controlled. Explicit metalinguistic knowledge, subserved by declarative memory, is represented bilaterally in large areas of associative cortex and its appropriation involves the hippocampal system (Cohen & Eichenbaum, 1993), unlike the acquisition of implicit competence which involves the neostriatum and associated structures (Rauch et al., 1997; Saint-Cyr, Taylor, & Lang, 1988; Willingham, Koroshetz, & Peterson, 1996) and the cerebellum (Gordon, 1996; Ito, 1993; Jenkins & Frackowiak, 1993; Leiner, Leiner, & Dow, 1991). Declarative knowledge, which is impaired in amnesia, is generally spared in aphasia. There is therefore no reason to expect explicit metalinguistic knowledge to be affected in aphasic patients, to the extent that they have retained their episodic and encyclopedic memories. Interestingly, the areas associated with the second language identified in Perani et al.'s (1996) study are precisely those that would suggest an increased participation of declarative memory-related structures (i.e., bilateral activation of parahippocampal gyri), whereas the language areas that were considerably reduced relative to the native language are those associated with implicit linguistic competence (the classical perisylvian language areas, including the left inferior parieto-occipital area and the left inferior frontal gyrus).

What we call a "grammaticality judgment" turns out not to be an insight about the structure of implicit representations but an opinion as to whether or not a given surface construction is acceptable (i.e., commonly used). It is a judgment on the output of the system, not an insight on the form of the underlying system or the processes through which the output was achieved. Native speakers may not be able to explain why a construction is acceptable or not, simply that it is; second-language speakers may accu-

rately judge the grammaticality of sentences and give some coherent explicit grammatical explanation without being able to produce such sentences automatically. Native speakers may not know how they produce speech sounds that they nonetheless consistently produce automatically, whereas second-language speakers may have learned and are able to explain the position of the various phonatory organs and yet speak with a strong foreign accent. Hence differential grammaticality judgments by bilingual aphasic patients in their two languages tell us something about their conscious knowledge of what can be said, but may not be a window on whether they have retained the competence to produce such constructions automatically in either language.

Vocabulary has a special status in language representation and processing in that speakers are aware of the phonological form and referential meaning of words. Hence, these aspects are stored in declarative memory. This may in part explain the special status of words relative to sentence structure. Nonhuman primates are able to acquire comparatively large vocabularies but little syntax. New words may be acquired throughout life, including in a second language, although new inflectional morphology and the use of prepositions (or case, depending on the language) are notoriously difficult. The "savant" described by Smith and colleagues (Smith & Tsimpli, 1995; Smith, Tsimpli, & Ouhalla, 1993) was able to learn a considerable number of vocabulary items in more than a dozen languages, but he would speak these languages with the structure of his native language, English, which was itself deficient in many respects. Access to the lexicon, however, is automatic and thus unconscious, and in spite of numerous psycholinguistic experimental attempts, the mental (and cerebral) organization of the unilingual as well as the bilingual lexicon is unknown. The implicit–explicit distinction between vocabulary on the one hand and phonology and morphosyntax (including the grammatical features of lexical items) on the other may again not apply as fully to a later learned second language insofar as more of the morphosyntax may be declaratively stored and used in a consciously controlled manner, as suggested by the data reported by Neville, Mills, and Lawson (1992) and Weber-Fox and Neville (1996). This may have repercussions on the differential availability of various aspects of each language in bilingual aphasia.

Even though bilingual aphasic patients can have similar impairments in both their languages, they may nevertheless retain their metalinguistic knowledge, which, in some cases, can be far more extensive in the second language and thus give the impression that they have better recovery of the language that they spoke least well before insult. Rehabilitation of verbal communicative abilities can take advantage of this knowledge when it is present for the second language. Such a practice will not necessarily im-

prove patients' implicit competence but may help them communicate. This is in fact one way that individuals with genetic dysphasia compensate for their impairment in implicit linguistic competence, with such a high degree of success that, for some of them, only specific psycholinguistic tests on pseudowords can reveal their condition (Paradis & Gopnik, 1997). In English, for example, these individuals are typically unable to mark automatically the plural of nouns and the past tense of regular verbs, although they are able to memorize each form independently and use it accurately (most of the time) when they consciously control their production.

To say that some bilingual aphasics may appear to have recovered their weaker language better than their native language through the conscious use of metalinguistic knowledge is not to deny that one language system can be selectively inhibited, as evidenced by alternating antagonistic recovery. Cases of true nonparallel recovery may be explained in terms of differential activation threshold.

The Activation Threshold

As early as 1895, Pitres argued that differential and selective recovery patterns could not be attributed to the physical destruction of the underlying neural substrate, for a number of reasons. First, in order to obliterate one language while sparing the other, lesions would have to damage selectively the diverse cortical areas that respectively subserve comprehending, speaking, reading, and writing only in the part subserving that language and not the other or others. This would involve an unlikely distribution of lesions which certainly could not occur with the frequency characteristic of nonparallel recovery. A second argument to this effect is that languages that were selectively unavailable for some time often become available again and therefore could not have been physically destroyed. These observations were used to argue against the notion of differential cortical localization for each of a polyglot's various languages. Subsequently described patterns of antagonistic recovery and alternating antagonism provide further support for this argument. The representations that spontaneously become available again after 1 day, 1 week, or several months must have been there all along but were not accessible. This contributes to the debate over whether aphasia is a problem of access or of loss of representations in that, in some cases, selective nonavailability is clearly a problem of access. There is, however, no denying that the removal of a large enough portion of the classical perisylvian language area will inevitably result in the loss of representations—in both languages.

Following Luria's (1973) proposal that the activation of an item neces-

sitates the concomitant inhibition of all of its competitors, it has generally been assumed that, when a language is selected by a bilingual speaker, the other language is simultaneously inhibited (Berg & Schade, 1992). However, inhibition of the unselected language is never total (D. W. Green, 1986; Soares & Grosjean, 1984). Hence, the notion of a varying ACTIVATION THRESHOLD has been introduced. The basic assumption is that the underlying neural trace of a mental representation (e.g., a word or syntactic construction) will be activated only when it has received a number of positive neural impulses sufficient to reach the activation threshold of the item's substrate. In the same way that the action potential of a cell is initiated once a critical threshold is reached (Kandel, 1991), we may envisage that the neural substrate (cell assembly or systems of cells, portions of a neural network) of a particular mental representation is likewise activated once its critical threshold is reached. It is further hypothesized that each time an item is activated (e.g., each time a word or construction is used) its activation threshold is lowered so that fewer impulses will be needed to activate it again. However, when the item is not in use, its activation threshold slowly rises, making it more difficult to access with the passage of time (i.e., rendering the activation of its underlying neural substrate more difficult, as more impulses will be required for it to reach its raised threshold).

Although it requires further experimental verification, this tentative explanation is at least compatible with all of the observed phenomena, from the various bilingual aphasia recovery patterns, recency and frequency effects, and priming phenomena, to language mixing and language attrition. Also, one reason why comprehension is generally easier than production (or recognition easier than recall) may be that, in recognition, the external stimulus impinges on the sensory receptors which automatically send impulses that may be sufficient to activate the trace, whereas in recall the speaker must self-activate the trace with resources from within, that is, without the help of external stimuli. Thus a trace with a threshold too high for self-activation (recall, production) could nevertheless be activated when stimulated by perception (recognition, comprehension). In other words, an item that cannot be recalled could nonetheless be recognized and understood—a phenomenon commonly observed among children, learners of a second language, and aphasic patients. This also seems to be true in attrition: when a language has not been used for years, speakers may still understand much more material than they are able to produce. Thus the observed consequences of attrition and aphasia are very similar and may be accounted for by the same underlying mechanism, namely, a heightened activation threshold for the inaccessible item, be it a language (an entire subsystem) or parts of a language (those items less frequently or less recently used, or those affected by pathological inhibition).

Nonce borrowings (or language mixing) occur when the activation threshold of the translation equivalent is lower (i.e., has been used more often and/or more recently) than the item itself. Neurologically intact speakers may take the extra second or so required to retrieve the item in the appropriate language when speaking to unilingual interlocutors, but they will not usually bother to do so in informal conversation with other bilinguals. Instead they will simply insert a word or phrase of the other language to keep the utterance fluent. Aphasic patients may not be able to access the item with the higher threshold within a reasonable time and hence may be forced to mix. They may also be unable to inhibit the unselected item and instead end up with a blend (e.g., *zwörpö,* from German *Zwerg* and Hungarian *törpö* [dwarf], or *Heftag,* from German *Montag* and Hungarian *Hetfö* [Monday], as reported by Gloning & Gloning, 1965).

Some aphasic patients may not have spoken one of their languages—possibly their native language—for a long time. It is a common observation among neurologically intact bilinguals who have not been using one of their languages for some time that it becomes less easily accessible. When each language is consistently used in a different context, say, the native language at home and the second language at work, speakers may have word-finding difficulty in speaking about work-related topics in the home language, even though they do possess the relevant vocabulary in their native language. Therefore, when one language has not been used at all for an extended period of time—1 or more years—speakers experience difficulty in speaking it fluently, a difficulty that is usually overcome after 1 or 2 weeks of total reimmersion in the language. These systematic variations in performance are compatible in every detail with the activation threshold assumptions. We may expect that the speech of bilingual aphasic patients will be similarly influenced by these various patterns of use as they affect the activation threshold of a language as a whole or of specific items within the less frequently or less recently used language. The problems may be compounded by the pathological condition, which may raise the activation threshold in a blanket manner across the affected cerebral areas. These factors must be taken into consideration when comparing a patient's test results across languages—hence the importance of an exhaustive bilingual history profile for interpreting differential scores, as will be discussed later.

The Role of the Right Hemisphere

Over the last 20 years or so, researchers have come to realize that verbal communication is more than just the use of implicit linguistic competence,

the aspect of language that was the focus of attention in aphasiology—rightly so, because it is the grammar (as broadly defined in contemporary linguistics, i.e., incorporating phonology, morphology, syntax, and the lexicon) that is affected in aphasia. On the other hand, patients with right hemisphere damage have been shown to have deficits in interpreting what is meant on the basis of what is actually said. The term DYSHYPONOIA has been proposed to describe this impairment (Paradis, 1998); it is derived from the Greek ὑπονοώ—to grasp what is "understood" (in the sense of the French *sous-entendu*) albeit unsaid—and refers to the difficulty patients have in drawing appropriate inferences from extrasentential information, leading to problems in the interpretation of the tacit component of an utterance (its illocutionary force or pragmatic element) with preserved comprehension of the literal meaning of a sentence (its semantics, derivable from the lexical meaning of words and morphosyntactic structure of the sentence, which is part of implicit linguistic competence and hence not affected by a right-hemisphere lesion).

Although, as discussed earlier, there is no clinical or experimental evidence of less asymmetric representation of implicit linguistic competence in bilingual than in unilingual speakers, there may nevertheless be a sense in which bilinguals may rely to a greater extent on their right hemisphere in verbal communication, namely, when using pragmatic features to compensate for the gaps in the linguistic competence of their weaker language. Bilingual aphasic patients, too, may rely to a greater extent on pragmatic cues when understanding or producing utterances in context.

Therapy can try to capitalize on the bilingual patient's possible habit of greater reliance on right-hemisphere–based pragmatic aspects of verbal communication by using paralinguistic features such as intonation, gestures, and facial expressions to aid the comprehension and production of verbal messages and thus circumvent the loss of linguistic competence which, especially in the elderly patient, may be difficult to restore. These strategies may, of course, help unilingual patients as well (Carlomagno, 1994; Dronkers, Ludy, & Redfern, 1998; Fex & Månsson, 1998; Garrett, Beukelman, & Low-Morrow, 1989; G. Green, 1984; Holland, 1991; Penn, 1984, 1985; Perkins & Lesser, 1993), although bilinguals may be more predisposed to making what may, after all, be only an increased use of familiar strategies.

Assessment of Bilingual Aphasia

Because no one factor has been identified that can predict the status of one language relative to another, it is imperative that aphasic patients be assessed in all of the languages they spoke premorbidly (Paradis, 1983a,

1983b). Not testing one of a patient's languages may have detrimental social and/or clinical consequences. On the one hand, patients may be unaware of which of their two languages is more affected, or may erroneously believe that one of them is better recovered. On the other hand, specific symptoms may be detectable in only one of a patient's languages, either because of the particular structural features of that language (Nilipour & Paradis, 1995) or because of a differential recovery in which the nontested language is the more severely affected.

The Bilingual Aphasia Test (BAT) was developed to assess aphasia in speakers of more than one language (Paradis & Libben, 1987, 1993). Designed to be equivalent across the 65 languages for which it is currently available, the various versions of the BAT are not mere translations for each other, but are culturally and linguistically equivalent tests. The criteria of cross-language equivalence vary with each task and the translation of standard aphasia batteries into languages other than the one in which they were constructed may not be adequate. The stimulus items may be culturally inappropriate or they may test grammatical constructions that present different levels of difficulty from those in the original language. Failure to implement the principles of linguistic and cultural adaptation in the assessment of aphasia will yield a translation that is unlikely to be meaningful and cannot be validly compared to an assessment obtained in the original language. For a review of the issues involved in the assessment of bilingual aphasia see Chapter 4.

Bilingual Aphasia Rehabilitation

The questions raised by bilingual aphasia therapy are many and varied. The major issue, with important social and economic implications, is whether therapy should be provided in both languages or whether the benefits of therapy provided in only one language will transfer to the untreated language. Many factors may influence such a transfer and its directionality. Furthermore, there is no consensus as to which language ought to be treated (Paradis, 1993b).

Relatively few studies to date have addressed language therapy in bilingual aphasia. Various authors report partial transfer of benefits from the treated to the untreated language, only transfer to or greater in the nontreated native language subsequent to therapy in the second language, no transfer to the untreated language, or treatment in both languages resulting in improvement in only one. Some authors suggest that therapy in both languages will exert an inhibitory influence on speech restitution (Chlenov, 1958) and hinder the recovery of all of the patient's languages (Wald, 1948,

1961). Hence they insist that therapy should be provided in only one language, at least initially (Chlenov, 1948; Hemphill, 1976; Lebrun, 1988; Wald, 1961). On the other hand, Bond (1984) suggests that BILINGUAL stimulation would be the most effective approach to language rehabilitation among bilingual aphasics. In her opinion, translation may be an appropriate means of rehabilitating a patient's second language, especially when translation skills are less impaired than other language skills.

The two patients described by Paradis et al. (1982) were able to translate words without hesitation into a language in which they could not name the corresponding objects. Providing them with the translation equivalent seemed to help them overcome their word-finding difficulty in that they were able to produce the word they could not come up with on their own. Patients with similar conditions could be encouraged to evoke in English, for example, the word they need in Spanish when the Spanish word eludes them. This strategy might even help them produce full sentences, given that theses two patients were able to translate sentences easily (whose structure contrasted greatly with that of the target language) in a language they could not speak spontaneously because of their overwhelming word-finding difficulty. Such an approach may work only with patients who, for whatever reason, have developed an automatic translation ability. There is, however, no known factor that might help predict which patients might benefit from such an approach. The first patient described in Paradis et al. (1982) had learned the second language formally at school after the age of 10 years, whereas the second patient had acquired both languages informally before the age of 5 years. Meanwhile, many non–brain-damaged fluent bilinguals find it very difficult to translate on demand, suggesting that translation is a skill independent of fluent mastery of two languages. The fact that some patients are able to access a language through translation when they are unable to do so directly—and that they are not able to translate into a language that they can speak and understand—points to such an independent pathway.

Not only are various languages sometimes spontaneously recovered differentially, but different languages may be differently responsive to therapy. Because it is possible for bilingual patients to be selectively responsive to therapy in only one of their languages (Watamori & Sasanuma, 1976), it is strongly recommended that even when therapy shows no effect in one language, it should nevertheless be attempted in the other.

Therapy in some patients' second language (which was also the language of the environment) has been reported to have greater beneficial effects on their nontreated (native) language (Durieu, 1969; Linke, 1979), even though, in one case, the patient had spoken the second language exclusively for the past 16 years. Thus, lack of noticeable improvement in the treated language may conceal improvement in the nontreated language.

Therefore all of a patient's languages should be tested when assessing the effects of therapy.

According to a recent pilot study undertaken by our research group involving 41 bilingual patients in Spain, Iran, and Finland, the amount of transfer of the benefits of language therapy seems to be proportional to the degree of similarity between the various structural aspects (e.g., phonological, morphological, syntactic, or lexical structure) of the two languages. We do not yet have sufficient data to assess the influence of the type of language treated (the native language or the second, the best or the least well recovered, the most or least fluent before insult, the language of the hospital or that of the home environment), the type of aphasia (Broca's, Wernicke's, conduction, transcortical), or the pattern of recovery (parallel, differential, successive, selective, blending, antagonistic).

One could perhaps expect an interaction between structural distance and type of aphasia in that correction of speech apraxia in patients with anterior lesions might transfer only to the extent that the two languages share the same phonemes, and correction of syntactic problems to the extent that the structures in the two languages are the same, whereas in patients with posterior lesions the transfer may be more extensive, provided that the deficits are of a higher-order semantic nature. The greatest amount of transfer would conceivably be in patients with temporoparietal lesions who speak two closely related languages of which the semantic systems greatly overlap; the least degree of transfer would be in patients with lesions in Broca's area who speak two languages with radically different phonological and/or syntactic structures.

Conclusion

Researchers are becoming increasingly aware of the omnipresence of bilingualism—if only in the form of speakers of a later acquired second language—and language pathologists encounter such individuals with mounting frequency in the clinic. Moreover, as of 1993, the American Speech–Language Hearing Association has mandated that all training programs in speech–language pathology and audiology must incorporate bilingualism issues in their curriculum. In Great Britain, most speech–language curricula now have modules of courses dealing with bilingualism. Nevertheless, there are still more questions than answers with respect to a therapeutic approach to bilingual aphasia. At least, most of the relevant questions have been identified and consequently clinicians and researchers now know more or less what to look for. Now it is a matter of verifying the hypotheses by devising appropriate experiments and running clinical trials.

Bilingual aphasic patients may recover their languages in several different ways. Unless all languages spoken premorbidly, irrespective of their degree of proficiency, are assessed in an equivalent manner, the overall assessment will remain grossly incomplete and possibly dangerously misleading.

Implicit linguistic competence (i.e., the grammar or language system) is affected in persons with aphasia, but there is good reason to believe that whatever explicit metalinguistic knowledge the patient possessed before insult could still be available and could be used to communicate, albeit in a controlled manner. The capacity to draw appropriate inferences from situational context, discursive context, and general knowledge in order to interpret the meaning of an utterance is affected in persons with dyshyponoia but remains available to patients with aphasia and may also be used to improve verbal communication. Bilingual speakers may have developed greater reliance on pragmatic cues to compensate for linguistic competence lacunae in their weaker language, in which they may also possess extensive metalinguistic knowledge. Both pragmatic competence and metalinguistic knowledge can be tapped to boost bilingual aphasic patients' overall verbal communicative ability.

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16

Ethnocultural Dynamics and Acquired Aphasia

JOAN C. PAYNE

Population projections in the United States for the twenty-first century show an increasing diversity as older Americans (65 years and older) of all races and cultures live longer, more active lives. These projections also indicate that nonwhite ethnic groups will make the most dramatic jumps in population growth. The ethnocultural elderly, that is, older African Americans, American Indians/Alaskan Natives, Asian Americans/Pacific Islanders, and Hispanic Americans over the age of 65, will become a major presence in the aging population.

Stroke is probably the most common cause of communication deficits in an older population (Rau & Schulz, 1988). Approximately one million persons in the United States have acquired aphasia (Klein, 1995; National Stroke Association, 1996). Older women outnumber men in every ethnic group. As adults age, their risk for strokes increases. Aphasia is found to be more prevalent in women than in men and to occur more frequently among women with infarcts and cardiac embolisms than among men (Hier et al., 1994).

The elderly are ethnically and culturally distinct from one another within the same language group, within groups of the same national origin, and within different socioeconomic groups of the same ethnic groups (Clark, 1988). Despite this heterogeneity, higher prevalence rates of hypertension, cerebrovascular disorders, and diabetes among specific ethnocultural older persons, for example, African, Hispanic, and American Indians, place them at greater risk for strokes and aphasia at younger ages than their

white age cohorts (Payne, 1997). Similarly, predictors for long-term disability after stroke vary considerably by ethnic group identification (Horner, Marchar, Divine, & Feussner, 1991; Kell et al., 1989; Miles & Bernard, 1992; Pinsky et al., 1985). Not surprisingly, those persons who are most at risk for stroke are also most at risk for long-term physical and language disabilities after stroke.

Loss of language can be particularly difficult for the elderly. Older persons are less likely than their younger cohorts to embrace current views about health and wellness. For example, older migrants from southeast Asia are more likely than their U.S.-born grandchildren to believe that "soul loss" or "bad wind" can cause illness, and are therefore more likely to use folk remedies as a cure for illness (Kang & Kang, 1995).

The impact of stroke-related deficits may, however, vary with the elders' perceptions of the stigma of growing older. For example, older blacks may perceive themselves as younger than their chronological ages because they are afforded esteem and are placed less frequently in long-term care by their younger family members (Tate, 1983). Furthermore, aging blacks are more likely than older whites to remain active in the community and to participate in senior citizen centers and age-homogeneous social activities and clubs (George, 1988). Whether these differences are generalizable to the larger population or have a systematic effect on aphasia outcome across ethnic groups has been largely unexplored.

Significant prognostic indicators for aphasia outcome are typically motivation, absence of physical and cognitive disorders, residual linguistic abilities, potential for neural repair or restructuring, age, gender, and educational level or a history of life-long mentally stimulating activities. Less typically explored are issues related to the aphasic's ethnic and cultural environment that define the quality of life for the individual.

Language as a Sociocultural Phenomenon in Aging

Cox (1988) summarizes several social reality theories of aging for which language is an integral part. One important theory is that an individual attempts to maintain continuity of roles and experiences throughout life. According to this theory, older adults, based on their past, are predisposed toward maintaining a level of consistency in the habits, associations, preferences, and lifestyles that have developed over a lifetime.

Cultural values that prescribe appropriate roles and associated behaviors have enormous impact on the well-being of ethnic elderly. They provide the elderly with a sense of identity and status in what may be per-

ceived as an unfriendly world. Through the maintenance of ethnic ties, the elderly can achieve a sense of continuity throughout the life course as relationships remain structured according to traditional norms and roles that govern interactions (Becker, 1993; Cox, 1988). Adults tend to return to those roles and experiences in late life that give them status and positive self-perceptions within their cultural and ethnic communities. Hence, ethnicity and cultural group affiliations become increasingly important as adults age (American Speech-Language-Hearing Association [ASHA], 1991).

Aphasia, which produces well-documented language deficits, is also an impairment of the continuity of a patient's worldview. Saville-Troike (1986) observed that language is typically described in terms of its expressive features, that is, its vocabulary, grammar, and phonology or orthography. However, a deeper understanding of the complexity of language must extend to its sociocultural uses. As Saville-Troike (1986) declared, "Language is a key component of culture. It is the primary medium for transmitting much of culture" (p. 49). Deficit theorists tend to study aphasia apart from the cultural contexts of the speaker. If language reflects the individual's linguistic capabilities, it is also a deeply personal and social phenomenon that conveys the cultural attitudes, histories, and images of a people.

Culture and Ethnic Perceptions of Illness and Disability

Ethnic groups may or may not be minority groups (Rosenthal, 1986). Ethnicity may be viewed either from the outside as a characteristic attribute or from the inside as a basis of group identification and belonging. Kalish (1988, pp. 16–17) summarizes some of the markers of ethnic group identification as

- Geographical origin
- Migratory status
- Race
- Language or dialect
- Religious ties or faith
- Ties through kinship, neighborhood, and community
- Shared traditions, values, and symbols
- Literature, folklore, and music
- Food preferences
- Institutions that specifically serve and maintain the group
- An internal sense of distinctiveness
- An external perception of distinctiveness

Ethnic identification of an individual and the milieu of the ethnic and cultural community influence not only communication patterns with others, but also the perceptions of self and others within and external to the community. The extent to which an aphasic person is handicapped by linguistic deficits or perceives a linguistic deficit as an impairment that can be treated by rehabilitation is related in part to the ethnic and cultural context in which the individual participates (Coet, 1977). What may be considered a debilitating language handicap in one ethnocultural community may not be perceived in the same way by another because of differences in history, religious beliefs, or group perception of different or aberrant behaviors.

In this way, culture, or the way of life of a people, determines how mind and body functioning is perceived by a handicapped person and also what is most significant to that person's daily life. More frequent attendance at religious services has been associated with low incidence of stroke (Colantonio, Kasi, & Ostfeld, 1992). If stroke occurs, however, the more tolerant a community or family is toward deviant behavior, the more likely the disabled person may remain in the community. The culture of the community determines, therefore, the norms of acceptable behaviors and prescribes appropriate help-seeking modes for the ill (Kang & Kang, 1985, 1995).

Religion and Spirituality in Coping with Disability

Stroke survivors are understandably anxious about and have trouble accepting their deficits. A number of explanatory models are frequently used by older persons to interpret the causes of their strokes and aphasia. These explanatory models appear to be driven by the cultural perspectives of the stroke patient. Fowkes (1988) found from a randomized sample that one in four individuals has no explanation for a disabling condition, whereas others may use a single concept to explain a myriad of health problems. For example, bodily impurity may be given to account for a variety of illnesses or "bad living" may be thought to cause strokes and stoke-related disabilities. A multiple causation model may be used for each illness, as in the explanation that heart disease is caused by a combination of overexertion, mental stress, and diet indiscretions. Other explanations include an iatrogenic model in which some health care provider or health-related entity has caused the illness; a hereditary model in which poor health is inevitable because of family history; a "used up" model in which an illness is caused by not enough rest or overexertion; and an environmental model in which radiation or some air-borne agent is believed to have caused the health problem.

These explanatory models are as varied as the cultural histories of ethnocultural older adults over age 60. Those who believe in a supernatural model may turn to clerics, sorcerers, or fortune tellers for prayers or exorcism to cope with illness and disability. Beliefs about balance in nature may direct the distressed to herbal doctors, acupuncturists, or clerics. Those who believe in social models may seek advice from close friends, members of families, or clerics (Kang & Kang, 1985; Narikiyo & Kameoka, 1992; Neighbors, Jackson, Bowman, & Gurlin, 1983).

In general, older persons of color in the United States tend to adhere to health beliefs that incorporate more of a mind–body interaction than is emphasized in Western medicine. Although only sparse data are available on perceptions of aphasia disability across ethnocultural groups, cultural beliefs about illness, and hence sequelae from acute conditions such as stroke, are known for some ethnocultural groups. Some of the commonly held beliefs are relevant to aphasia.

African Americans

African Americans are a heterogeneous population of individuals representing one of several visible minorities who are physically different from northern Europeans. Cultural values of various African tribal communities that emphasize collectivity, sharing, affiliation, obedience to authority, belief in spirituality, the importance of the past, and respect for the elderly have been transmitted across generations, surviving the disruptions resulting from forced migration to the Americas and slavery (Baker, 1994).

Jackson (1988) observes that age 55 may be a more appropriate marker for old for black Americans because of documented deficits, health disabilities, and early retirement. However, adaptation to health problems and change in employment appear to be related to cultural perspectives. Seiden (1981), for example, found that the white suicide rate increases with age, whereas that of nonwhites decreases with age. From these data it was hypothesized that nonwhites are less prone to suicide because their shorter life expectancies encouraged stronger survivor effects, a philosophical acceptance of hardship, continuity of purposeful activities in aging, and a perception of less downward mobility.

Ethnic and cultural affiliation also influence how African Americans treat illness. Parks' (1988) comprehensive study of rural black elders in Tennessee, Arkansas, and Mississippi confirmed that a large percentage may treat themselves when they are ill with religious healing or with old remedies passed down through generations.

The role of religion in the lives of black elders has been well document-

ed (Payne, 1997), but there are other factors such as superstitions and folklore that are less well reported. The black elder patient's explanatory model is often one of natural as compared with unnatural illness. Natural illness comes from not taking care of one's body; unnatural illness can be caused by roots, witchcraft, or voodoo. Within this explanatory model, aphasia, and all other health problems, can be cured by the health professional/folk practitioner with the greatest power. It is merely necessary to continue to search for the appropriate healer. Ultimately, the power to cure is in the hands of God who can cure any illness if it is God's wish (Richardson, 1988).

The black church provides a belief system that gives coherent meaning to life and a philosophical system for coping with societal inequities (Taylor, 1986). There is greater religious involvement of older blacks compared with whites, although frequency of church attendance and religious affiliation are the same. Blacks are more highly concentrated in Protestant denominations, especially Baptist churches (George, 1988). Approximately 90% of older African Americans are Baptist (Taylor, 1986). Fundamentalist religious beliefs serve to buffer the anxieties of illness and influence how well one copes with disability and frailty. Within the black church, Old and New Testament references are often used as metaphors to explain how calamitous circumstances are used by God to test one's faith. Such expressions as, "God only gives you what you can bear," give encouragement to the individual to shoulder adversities well because they are meted out only in proportion to one's ability to handle them. Illness and disability, therefore, are to be borne with dignity and acceptance.

American Indians/Native Americans

Currently, there are more than 500 federally recognized tribes, 278 reservations, and more than 209 Alaskan villages, each with their own language, geographic location, customs, and systems of governance (G. Harris, 1986; G. A. Harris, 1993). Health beliefs vary by tribes and are not homogeneous. Little information is available on perceptions of disability by this group. However, kinship and religion are particularly important for the health and well-being of elders. Pueblos are taught to take care of their bodies in childhood and young adulthood in order to manage problems or illness in later age. Among Navajos, illnesses are thought to be caused by "bad living" (Stanford Geriatric Center, 1988).

Traditional American Indian medicine is holistic and wellness oriented. It focuses on behaviors and lifestyles through which harmony can be achieved in the physical, mental, spiritual, and personal aspects of one's role in the family, community, and environment (Kramer, 1992). Some Indian people accept a handicap as a special gift from the Great Spirit (G.

Harris, 1986). In general, however, attitudes toward the handicapped vary greatly. In some Indian cultures, there is greater acceptance of the handicapped individual and greater value given to the special role each person plays within the society. In other tribes, handicapped persons are not as well tolerated. As traditional roles and cohesiveness have eroded in some Indian tribes, there have been changes in how the handicapped are accepted within the community (G. Harris, 1986).

Asian Americans

The elderly of this group are extremely diverse in national origin, language, and culture from east and southeast Asia, the Indian subcontinent, Polynesia, Melanesia, and Micronesia (Payne, 1997). Japanese, Korean, and Indochinese health beliefs originated with the Chinese. The Chinese belief system is that health is a state of equilibrium between man, society, and the cosmic forces of the universe. Two energy forces, *yin*, the dark, empty force, and *yang*, the light, full force, need to be in balance for optimal health (Stanford Geriatric Center, 1988).

In the Chinese system of beliefs, disease is a disturbance of the delicate relationship between man and the cosmic forces. Chinese elders are reluctant to acknowledge illness and serious conditions may be neglected. For example, the Chinese are often very reluctant to consider surgery. The Chinese tend to be very tolerant of aberrant behavior whereas Euro-Americans would define a mental illness or dementia as unacceptable or deficient behavior. These same disorders are often tolerated within Chinese families (Stanford Geriatric Center, 1988).

Tongans believe that neurological symptoms come from broken taboo or that the sun has struck them for doing something bad such as eating in a graveyard (Stanford Geriatric Center, 1988). The Laotians believe that sickness comes from the gods and represents an imbalance of bodily humors which can be treated by consistent monitoring of natural restorative processes in the body or by aiding natural processes like digestion and sleep (Mueke, 1983). Filipinos believe that illness is the result of an imbalance of hot and cold; the "hot–cold" belief system is found in Southeast Asian culture. Western medicines are believed to be "hot" and therefore too potent to take in large doses. The imbalances between hot and cold can be caused by cold drafts, overheating, personal disorders, uncleanliness, or by psychic factors such as stress, grief, or a loss of self-esteem. Among the Samoans, sickness can be caused by possession by ancestral spirits as a form of punishment for violating cultural taboos (Moriaka-Douglas, 1988).

The Vietnamese believe that illness can be caused by climate, temperature, and weather changes, nutritional imbalances, and bad fate. Hyper-

tension, a major contributor to stroke, is caused by hard work, fatty or salty foods, alcohol, sadness, or worry. *Phong* or "wind" as a cause of illness can be alleviated by using "coining" or glass cups to draw out ill wind (Lew, 1991).

Indian and Pakistani beliefs are very similar. Illness is thought to be caused by an imbalance in the body from acidity, cough, and gas. Most illnesses are believed to come from God. Under these circumstances, the fatalist view is that there is no treatment available to intervene in illness. The profound belief in *karma* or destiny is manifested in the belief that if illness occurs, the sufferer is at fault, because what is done in the present will influence the future (Stanford Geriatric Center, 1988).

European Americans

European American immigrants began to decline in number from the early decades of this century. Because of its immigration history, the Euro-American population was overrepresented in 1980 among the age categories of old (65 to 74 years) and old–old (75 years and older; Lee, 1986). For these elderly, the most competent Western health care specialist is the one who will rehabilitate the disability (Payne, 1997).

As with nonwhite ethnocultural groups, spirituality and religious beliefs define some of the approaches for coping with disabling illness among European Americans. Among the Portugese, for example, illness in later life is either to be expected as a part of aging or is a problem that can be negotiated with God (Stanford Geriatric Center, 1988). Many, like the Jewish elderly, use their religious institutions as the preferred sources of help and support in times of health crises. Country of origin tends to determine religious group affiliations. Swedish, German, Estonian, and Latvian elderly are primarily Lutheran; Italians are primarily Catholic; and Hungarians are Roman Catholics and Reformist Calvinists. However, religious institutions and ethnic associations, organizations, and societies are not as closely related and compatible as is often thought. Religious associations have often splintered an ethnic community along denominational lines (Kalish & Creedon, 1986). Whatever the denomination, there is little doubt that these elderly as a group value their religious beliefs, and religion often forms the basis for coping and health-seeking behaviors.

Hispanic Americans

The Hispanic elderly originated from Mexico, Puerto Rico, Cuba, and Central and South America (Payne, 1997). For many Hispanic Americans, *espiritismo* is the belief system that the world is inhabited by both good and

evil spiritual beings who can affect the health and well-being of humans in positive and negative ways. This belief system is an integral component of Hispanic cultural tradition, as seen in both folk medicine and religion-based healing. Hispanic *curanderismo, santeria,* and *espiritismo* reflect a perceived interrelationship of humans with their natural and supernatural environments in which each is able to communicate with the other (Applewhite, 1995; Villa, Cuellar, Gamel, & Yeo, 1993).

Mexico is a predominantly Catholic culture. Catholicism, like most religions, forces people to face the reality that they are born to suffer and that suffering is an integral part of life. The ability to endure suffering, or *la sufrida,* is admired. As a religious philosophy, Catholicism instructs people on how to suffer and not on how to avoid suffering. Church beliefs also provide dignity as well as idealized role models to the sufferer. In their religious beliefs, Mexican Americans and other Catholic Hispanics are assured that even if their disabilities and behaviors are unacceptable to mainstream America, there is a set of universal Catholic principles and values and religious group ethos to sustain them (Bach-y-Rita, 1982).

The Role of Supportive Relationships in Aphasia Outcomes

A major cultural factor that has been recently addressed in the literature is the role of social networks in aphasia rehabilitation. Supportive relationships have important implications for recovery of stroke and aphasia. The more dense the network, the better the outcome (Chapman, 1988). Density of support refers to support from a spouse, children, siblings, and confidant friends (informal supports), as well as to groups, institutions, and organizations (formal supports). Rau and Schulz (1988) found that "One can conclude that perceived or actual deficiencies in any of these areas of support will have negative impacts on an older individual's motivation in participating and responding to speech–language therapy" (p. 170).

Each aphasic patient has a unique case history and outcome should be predicated on individual strengths and weaknesses. At present there is little empirical evidence that aphasia outcome is influenced by ethnic or cultural group affiliation, or by quality or quantity of support. There is research in other disciplines that has addressed the importance of support networks in rehabilitation. A reciprocal relationship model is one in which formal and informal supports are used to provide effective therapeutic intervention (Miner, 1995). There are also documented characteristics of elder networks within ethnocultural communities that permit some gener-

alizations about the level and degree of supportive relationships in an aphasic's community.

African Americans

African American elders tend to maintain dense support networks with kin and nonkin persons throughout their lives. Family and community affiliation are of major importance to the African American elderly. They depend heavily on family members for caregiving and life satisfaction (Taylor, 1985). A common arrangement is for an elderly person to live within an extended family with an adult child, usually a daughter, and provide reciprocal support through sharing household expenses and responsibilities (Payne, 1997). Richardson (1990) noted that African American older adults were very likely to be accepted and cared for when they became frail or ill. Religious beliefs reinforce obligations to honor one's parents by helping them when they are in need of support.

American Indian/Alaska Natives

Within tribes that value extended families, Indian elderly are highly valued and occupy an important place in making major decisions for the family and tribe. About three-fourths of rural American Indians between 65 and 74 years of age live with their families, whereas only about one-half of the urban Indian population over age 75 live within a family environment. Those who live with their children do so because of cultural preferences and the ability to share in family resources. Care is generally given by the families or in elderly facilities on reservations (Red Horse, 1990). Other differences between rural- and urban-dwelling elderly can be seen in the rates of nursing home placement. Urban elderly are more likely to be placed in nursing homes than are rural elderly (Manson & Calloway, 1990).

Asian Americans

Korean Americans appear to have the strongest family ties and the strongest indices of family unification of any other Asian group, although Filipino elders also maintain regular contact with their families and children (Keifer et al., 1985; Kim, 1990). Assimilation appears to have eroded many of the traditional values of filial piety; however, despite these changes, most Asian American elderly report similar densities of support kin and nonkin networks within their ethnic enclaves as do the African American and Hispanic elderly (Cho, 1990).

European Americans

Euro-American elders also rely on their families and their adult children to provide support as they age, although the literature suggests that nonwhite ethnocultural elders tend to maintain denser networks of family and friends and closer ties to their networks. Greater income and access to health care enable the European American elder to be more flexible about where they will live in relation to their children. There are also differences in the degree to which family members and nonkin support persons are an integral part of the lives of elderly persons by ethnic group. Sharing intimate concerns with family is characteristic of Jews, Italians, and Slavs, while relatives are more important in obtaining resource support for Italian-American men and Polish-American women (Gelfand, 1981).

Cultural variation should be expected among European ethnic groups in filial obligation and devotion to family. This variation will decrease but not disappear in succeeding generations. Any differences in support levels are more likely to be predicted by socioeconomic rather than cultural factors. When socioeconomic status is controlled, however, some ethnic differences are still likely to be apparent in the domains of affect, norms, and extent of interaction (Rosenthal, 1986).

Hispanic Americans

Hispanic elderly have dense social and support networks. Hispanic cultures and kinship patterns provide strong support for needy elders. The family is viewed as a life-long system of emotional support and, at times, material assistance (Cuellar, 1990). Hispanic elderly are likely to live within the context of large, extended families or live in close proximity to their relatives (Becerra & Shaw, 1984). Even in situations where extended family members are in separate households, nurturance, guidance, support, and control of functions are readily available to assist the elderly (Ho, 1987). A small percentage (3%) live in nursing homes, including those who are over 75 years of age (Cubillos, 1987).

Implications for Aphasia Rehabilitation and Research

The increasing interest among health professionals and social and behavioral scientists in the interplay between ethnocultural identification and coping with illness and disability represents a pivotal point in under-

standing aphasia outcome. Consistent with this interest is a growing awareness that the population of nonwhite ethnocultural older persons will increase rapidly in the next century. Similarly, there is a need to reevaluate European American elderly in light of their cultural and ethnic diversities. Aphasia therapy should be sensitive, therefore, to the ethnic and cultural histories and perspectives of the aging adult.

Among the many issues discussed in this chapter that have implications for treatment and research of aphasic disorders are the following:

1. Stroke can be devastating to language and to other important psychosocial dimensions of life for aged adults.
2. Language has linguistic components but also serves as the primary medium for the culture of the individual.
3. Aphasia is best evaluated for linguistic deficits in the context of the patient's culture and ethnicity.
4. Spirituality and religious beliefs can play definite roles in the formation of attitudes toward and coping strategies in sickness and disability, including language impairment.
5. Successful rehabilitation of language deficits is positively related to the density of an aphasic's support networks, including family and nonkin relationships.

Treatment is often based on the results of extensive testing of the aphasic person's language skills. This has merit in that it pinpoints areas of deficiency and assists the clinician to build on the patient's residual linguistic skills. But treatment based on testing alone limits the amount of data that a clinician has available to develop a culturally sensitive plan of rehabilitation. Data collection should also include information on the patient's ethnic and cultural group affiliations, the patient's/family's worldview on the causes of stroke and aphasia; the patient's/family's approaches to coping with aphasia in the daily living environment, and the density of support available to the patient. Depending on religious beliefs and extent of acculturation, ethnocultural explanations for stroke and stroke sequelae may be quite different from the more traditional or Western view of neuropathological causes. The degree of acculturation plays a dominant role in whether a patient or family embrace the accepted beliefs of their country of origin or are more inclined to accept the principles of Western biomedicine.

Prognostic outlook for aphasia recovery is determined fundamentally by neurophysiological repair and status, which can be more or less measured; subjective factors are more difficult to measure. The field is just beginning to recognize the importance of the subjective domain in recovery,

self-esteem, and perceptions of quality of life. Rau and Schulz' (1988) writing on the influence of social networks in aphasia treatment brings together social network theory and implications for aphasia rehabilitation in older persons. Support networks of the elderly can be characterized as an interwoven network of informal services provided by family and friends and formal services provided by public and private agencies (Lubben & Becerra, 1987). Rau and Schulz (1988) ask the questions, "Why do some individual families seem to cope better than others with a chronic, disabling illness such as stroke? Why aren't all aphasic stroke patients depressed, and all significant support persons feeling burdened?" (p. 168). The answers to these questions lie in the variations in support networks seen within and across ethnocultural groups and in the cultural philosophies toward caring for older, frail persons within these networks.

The California Senior Study found some interesting variations in how Mexican, African, European, and Chinese elderly report on the quality and density of their support networks (Lubben & Becerra, 1987). Mexican and Chinese elderly are much less likely to seek formal help because of their strong informal support systems. Because of their more recent immigration and language barriers, both Mexican and Chinese elderly tend to reside in ethnic enclaves and to depend heavily on institutions within the community for help. Black and whites, however, are more likely to report having close friends than are Mexican elderly, although Mexicans are much more likely than either blacks or whites to report being close to their families. Chinese elderly are less predictable. They report the lowest frequency of regular contact with their children and no weekly contact with a sibling who may reside in another country. Consistent with the reports of Chinese elderly are their complaints that as their children become more acculturated, they tend to move away from their parents and to not visit them regularly. These findings strongly suggest that clinicians should not assume that support networks are homogeneous within ethnic groups. Rather, support networks appear to be more reflective of family acculturation, geographic distance from family, and personal preferences.

Religious beliefs and cultural preferences appear to determine both the patient's outlook on disability (often pessimistic) and the attitudes of caregivers toward their participation in the caregiving and therapeutic process. The religious beliefs of some caregivers, for example, support them in giving assistance to disabled elders. Goldberg (1996) noted that African American caregivers may be more accepting, less judgmental, and better able to handle grief with less anger than white caregivers of dementia patients. This observation was consistent with empirical data (Miner, 1995) that African American caregivers perceive their caring responsibilities as less

burdensome than did whites. The higher the socioeconomic status of white caregivers, however, the more emotional support they feel they are able to give to an elderly parent (Rosenthal, 1986).

Research on aphasia has provided an exhaustive database on the linguistic deficits that are consistent with site of lesion, and type and severity of stroke. Much has been learned already about the varieties of aphasia and their particular characteristics, and the appropriate treatment for each type. Research now should emphasize understanding aphasia within domains that have been less well described, such as the prognostic factors and outcome issues in aphasia that are influenced by the patient's ethnic and cultural environments.

Conclusions

Current descriptions of aged individuals who suffer cerebrovascular accidents and aphasia provide insight into the population that is now 65 years old and older. By the twenty-first century, however, the aging population may look very different in terms of the prevalence of chronic diseases that cause aphasia. Many social inequities will need to be corrected to increase the participation nonwhites in the health care system. However, an advantage to the widespread health promotion and wellness information available through the major media is that most individuals are now alerted to modify the ways they can maintain their health through old age.

Many more nonwhites who will be near retirement in the next 30 to 40 years may have more choices in how they identify themselves as members of ethnic and cultural groups. It will be interesting to observe whether the cultural values and customs related to sickness and disability valued by aging adults in the latter half of the twentieth century will continue intact in the coming years or whether some but not all will be passed down through the generations.

Meanwhile, there is a large population of underserved Ethnocultural older persons with aphasia who deserve treatment and systematic examination. Understanding aphasia within a context of differing cultural perspectives will continue to challenge the profession.

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17

The Psychological and Social Sequelae of Aphasia

JOHN E. SARNO and GUIDO GAINOTTI

The emotional dimensions of aphasia are important considerations because the disorder has ramifications that extend far beyond the pathology in linguistic processing. The loss of normal communication strikes at the very roots of a person's sense of self inasmuch as identity is based primarily on relationships, which, in turn, depend largely on communication (Brumfitt, 1993; M. T. Sarno, 1986).

Literature on the psychological and social sequelae of aphasia has increased steadily, especially in the last decade. As in the earlier editions of *Acquired Aphasia,* this chapter reviews studies on the psychological and social sequelae of stroke and traumatic brain damage for whatever can be gleaned from them that pertains to aphasia. However, there are now more studies that focus on aphasia specifically. New work is also emerging that examines the effects of cultural–ethnic variables on the social sequelae of aphasia (see Chapter 16). Although individual reactions to aphasia are not statistically valid, they enrich our knowledge and clinical acumen; therefore, case studies are also included in this discussion.

Historical Review

Kurt Goldstein (1942) focused on what he identified as the CATASTROPHIC REACTION (CR), characteristic of many patients with aphasia, and argued that it resulted from a disturbance in the patient's ability to

maintain "biological homeostasis" rather than from a sense of inadequacy. Overlooking the concept of the unconscious, which had been well formulated by Freud and his followers by that time, Goldstein postulated that brain damage produced a failure of the entire organism, a "biologic" rather than a psychic response, and that patients' reactions represented their attempts to maintain biologic homeostasis.

One could argue, however, that these same behaviors represent a desperate effort of the individual's psychic apparatus to maintain equilibrium (sanity, emotional stability) in the face of insults to the higher functions (language, cognition, perception) and that psychic stability is an integral aspect of biologic homeostasis rather than a thing apart. Whatever the merits of Goldstein's hypothesis, the description of the CR has been of great value to clinicians (Schuell, Jenkins, & Jimenez-Pabon, 1964).

A number of clinicians have called attention to the importance of the emotional concomitants of aphasia. Schuell et al. (1964), Wepman (1951), and Eisenson (1973) made frequent references to emotional phenomena and emphasized the necessity of considering them in therapeutic interaction with patients. Both Wepman and Eisenson stressed the need for psychotherapy.

A Psychodynamically Oriented Report

In 1961, Friedman reported systematic observations of a cohort of aphasic patients in a group therapy setting over a period of $7\frac{1}{2}$ months. He found that all of the patients expressed feelings of isolation, loneliness, sensitivity, and psychological impoverishment; almost all suffered feelings of lowered self-esteem that caused them to avoid and reject people, in fear of their own rejection. These feelings persisted even in the group therapy setting, from which they tended to withdraw. Behavior was generally regressive, as exemplified by the defensive use of dependency needs, projection, denial, and exaggeration of their deficits. The importance of language as a mechanism for exerting mastery over the environment was evident in problems with reality testing experienced by these patients.

The Ullman Monograph

Ullman (1962) published the results of a systematic observation of behavioral changes in 300 patients admitted to a stroke study over a 3-year period. No attempt was made to categorize emotional responses on the basis of the site or the extent of the lesion. Some of his conclusions are relevant to the purposes of this chapter. In analyzing reactive responses to the stroke, leaving aside the patient with diffuse impairment of brain function,

Ullman found that the severity and duration of "physical disability," in which category aphasia was included, were the most important determinants of the nature of the patient's response: Persistence of a deficit led to depression, hopelessness, and feelings of futility. Next in order of importance was the life situation to which the patient was returning. Premorbid personality was characterized as being "all important" but resisted classification that would predict whether a given patient would respond well.

Ullman described depression as reactive but differing from that seen in the usual psychiatric population. He found that the real problems confronting these patients often made it difficult to distinguish between (a) appropriate feelings of despair, loneliness, and so forth, and (b) depression attributable to the patient's premorbid personality. High on his list of reasons for psychiatric hospitalization of the stroke patient was depression secondary to aphasia. Other factors, not specifically related to aphasia, were antisocial behavior, inability to adapt to altered life circumstances, and latent psychosis precipitated by the stroke. Sexual sequelae, most particularly impotence, was found to be a reaction to psychological conflict, rather than the result of pathophysiologic alterations.

Horenstein's Review

Horenstein (1970) reviewed the effects of cerebrovascular disease on personality and emotionality, drawing attention to a number of important clinical phenomena that accompany or are the result of stroke. Of these, he singled out depression as representing a grief reaction, its existence and severity relating to the type and severity of the neural deficits, as well as to the patient's awareness of the illness, premorbid capacity for adaptation, intellectual level, and feelings of self-worth. Horenstein noted that the reaction is not specific to stroke but occurs with any catastrophic illness. Other determinants of severity and duration of depression are the personal and social situation of the patient, for example: the loss of an accustomed role at home, in the community, or at work; the quality of family relationships; and the adequacy of plans for the posthospitalization period. Because of the importance of the severity and extent of neural deficits, Horenstein emphasized the therapeutic program as a practical means of combatting depression. To the same end, he stressed the need for careful and effective planning for the patient's future. The development of independence is paramount both for practical purposes and for its contribution to the patient's feelings of self-esteem. These observations are all particularly relevant to the patient with significant aphasia.

Horenstein also considered denial of illness (or its manifestations) in a psychological context (distinct from the perceptual phenomenon). This re-

action has been discussed by a number of authors (Baretz & Stephenson, 1976; Gainotti, 1972; Ullman, 1962; Weinstein & Kahn, 1955) and is often encountered in the patient with aphasia. Patients who had difficulty facing reality premorbidly will react similarly to the problems posed by aphasia. However, Baretz and Stephenson (1976) considered denial a necessary stage for many patients, serving the purpose of "buying time" as they struggle to adapt to their new reality, but probably only temporarily staving off the depression that must inevitably come. Baretz and Stephenson (1976) and Horenstein (1970) also called attention to the effect of denial in its various forms on the professionals who work with patients. Most authorities on the subject of denial explicitly or implicitly suggest the need for psychiatric intervention, particularly when the degree of denial interferes with the rehabilitation process.

Horenstein (1970) included in his review a discussion of the pseudobulbar state as possibly being confused with depression. In addition to inappropriate laughing and crying, other possible symptoms included a fixed facial expression, nonspastic articulatory and swallowing problems, partial mutism, and motor compulsions. All of these are rather dramatic manifestations of anatomicophysiological aberrations. Persons with aphasia may cry easily when exposed to emotionally laden material, either happy or sad in content. However, the emotional lability of the aphasic patient may not have the distinctly "organic" flavor of the pseudobulbar state described by Horenstein. Indeed, he stated that in almost all cases that have been documented the bilateral corticobulbar lesions are generally symmetrical and widespread. On the other hand, although the most frequent stroke lesion is unilateral, the impression is that there is a loss of inhibition or control in the labile aphasic patient, suggesting a physiogenic process but also reinforcing the concept that the left hemisphere plays a monitoring rather than a generative role in emotional expression (Gainotti, 1972; Lamendella, 1977).

Gainotti's Study

A report by Gainotti (1972), primarily designed to explore the differences in emotional behavior associated with the hemispheric side of a lesion in stroke patients, made specific reference to emotional reactions in four groups: a category of seven reactions under the heading CATASTROPHIC REACTIONS; five affective states characterized as DEPRESSIVE MOODS; four INDIFFERENCE REACTIONS; and a category of OTHER REACTIONS, which included confabulations, delusions, and hate for limbs. Patients with left-hemisphere lesions manifested catastrophic or anxiety–depression symptoms more frequently than did patients with lesions of the

nondominant hemisphere, whereas indifference reactions were more common among the latter group. When the left-hemisphere patients were categorized according to the presence or absence of aphasia, Gainotti noted further differences. Broca's aphasics had statistically more dramatic, sudden, and short-lived emotional outbursts than other aphasics (Wernicke's aphasics did not weep at all), whereas the amnesic aphasics showed a different pattern of anxiety reaction that seemed to reflect greater awareness and control. Wernicke's aphasics were also quite different from nonaphasics and patients with right-brain damage in the frequent use of emotional language (swearing, cursing, religious imprecations), although in this they were almost matched by Broca's aphasics. The latter group showed the highest incidence of aggressive behavior (25% compared with about 10% for most of the other groups). Twenty-five percent of the fluent aphasics seemed unaware of their language problem, matching the proportion of right-hemisphere patients who were anosognosic, but the fluent aphasics did not manifest other characteristics of "belle indifference" as did the right-brain–damaged patients. In fact, Wernicke's aphasics expressed discouragement in greater numbers than did all other groups and matched others in drawing attention to their failures, clear evidence of some degree of awareness of and concern about their deficits.

The concept that the right hemisphere processes (i.e., recognizes and expresses) emotional phenomena appears to be generally accepted. Judging by the observations of left brain–damaged patients, the left hemisphere apparently functions to control emotions that originate elsewhere in the brain; however, the degree of control may depend on the patient's awareness of the aphasic deficit. According to Gainotti, amnesic aphasic patients are most aware of their deficits and retain the greatest degree of control over their emotional reactions. Those with Broca's aphasia appear to be aware, but their anxiety and/or depression, and possibly their anger, diminish their control. Fluent aphasic patients are the most complicated. Gainotti's (1972) data suggest that fluent patients are also anxious and depressed but present a confusing pattern of awareness and control. The inappropriateness of the behavior of some patients with Wernicke's aphasia is well known to clinicians, and the intimations of psychosis in a small number of patients is now a classical observation (Benson, 1980; Horenstein, 1970). The parapsychotic behavior of the occasional patient with Wernicke's aphasia may be the consequence of both affective and cognitive incompetence resulting primarily from the pathological lesion.

Smythies (1970) suggested that psychoses are the result of qualitative changes in brain physiology (as opposed to quantitative ones in the neuroses) which may be the result of biochemical aberrations. Such changes may be of significant enough magnitude in Wernicke's aphasia to mimic a

classical psychosis. The frequent involvement of the temporal lobe in Wernicke's aphasia lends further credence to a special affective deficit because of the association of that area with emotional processes (Geschwind, 1977; Kluver & Bucy, 1939; Papez, 1937).

Further complicating the problem of fluent aphasia are the suggestions that deficits in auditory processing lead to paranoid ideas in the patient (Benson, 1980) and confusion in the mind of the examiner (Ziegler, 1952). Finally, the jargon of these patients may result in an occasional diagnosis of schizophrenia.

Benson's Review

In this excellent review of the psychiatric problems associated with aphasia, Benson (1980) discussed some common psychosocial sequelae: alterations in lifestyle, change in employment status (sometimes with consequent financial problems), loss of social position and change of roles within the family, simultaneous loss of physical capacity in cases of hemiparesis, real or imagined loss of sexual function, erosion of self-esteem, and reactions of grief and depression. Benson also discussed the changes in emotionality associated with the locus of the lesion: the depression and frustration of nonfluent patients that sometimes leads to a catastrophic reaction with anger, hostility, loud crying, or withdrawal; the refusal to eat or participate in treatment; and the alteration in sleep patterns and personal hygiene. Because of the depth of depression, suicide is a possibility. Based on his experience, however, Benson thought the risk of suicide to be greater in patients with posterior lesions, the fluent aphasic patients. Although the only suicide in one of the author's experiences was a nonfluent patient with a severe right-sided motor deficit, the patient's loss of status as a highly esteemed professional with an international reputation was almost certainly the cause of his demise.

Change in emotional state is a highly individual matter, determined by the intensity of the person's intolerance for his or her altered state. In fact, a common observation among experienced aphasiologists is that little correlation exists between the severity or mildness of the aphasia and its impact on the patient. For example, someone with a relatively mild aphasia may be devastated and inconsolable. The same lack of correlation has been noted between the type of aphasia and emotional impact. As Benson pointed out, the problems in fluent aphasia are different, stemming from the language comprehension deficit and the tendency to use jargon, for both of which patients are often unaware and frequently unconcerned. Because these patients often are not understood and have trouble comprehending what is said to them, they may become paranoid in much the same way as

someone with acquired deafness. When this is combined with impulsiveness, they can be difficult to manage.

Benson also discussed other neurobehavioral disturbances, including confusional states, apathy, memory problems, and dementia. He also examined the difficulties inherent in evaluating how aphasia affects intelligence and how one establishes the legal competence of an aphasic person.

Jaffe's Treatise

Jaffe, one of the few psychiatrists with expertise in the diagnosis and treatment of emotional disorders in aphasic patients, wrote an excellent paper on the subject (1981). He pointed out that patients with aphasia are subject to the full range of psychiatric disorders seen in the general population, but because of the communication problem, the disorders may be difficult to detect and may not be recognized until the patients manifest such behavior as crying, psychomotor retardation, sleep or eating disturbance, significant weight change, manic hyperactivity, delusional behavior (e.g., paranoia), overt suicide attempts, physical destructiveness or assault, drug or alcohol abuse, panic attacks, or noncompliance with treatment regimens. Jaffe suggested that the extreme stress of having developed a "brain disease" may cause psychiatric illness to appear in these patients for the first time in their lives. Jaffe questioned whether some of the "symptoms" of aphasia, such as the catastrophic reaction, are transformed versions of familiar psychiatric disorders, and argued that the so-called Type A personality may react more violently to the frustration of nonfluent aphasia than the more relaxed Type B person. Jaffe's description of the fears and outright phobias that aphasic persons may develop as a result of their linguistic incompetence is particularly pertinent. Patients are very apprehensive about making calculation mistakes (e.g., giving the wrong amount of money to a clerk), taking telephone messages inaccurately, forgetting names, and performing a host of other tasks encountered in daily life. Because of their perpetual concern, patients often withdraw rather than face the embarrassment of making mistakes.

Jaffe also commented that aphasia robs the patient of the solace usually available to people who have suffered a devastating loss by being able to talk about it to loved ones. He described this as "healing conversation" and characterized aphasia as being particularly cruel in that it interferes with a "major vehicle of its own treatment." Jaffe discussed the very difficult problem of determining mental competence in aphasia, which often creates legal problems. When verbal and writing difficulties are complicated by comprehension deficits, as in a person with global aphasia, it may be almost impossible to establish competence. Benson (1980) suggested that

mental competence is probably best judged by someone highly trained in aphasia who knows the patient intimately. Jaffe's paper makes it clear that a knowledge of psychodynamics may be very helpful in assessing the emotional reactions of the person with aphasia, although assessment may still be difficult because of the communication disorder.

Language and the Limbic System

A review of the basic literature on communication and emotional behavior suggests that one is most likely to see a point of convergence by considering the function of the limbic system. The study of language has been limited almost entirely to neocortical systems, but more recent, attractive allusions have been made to the phylogenetically older and primarily subcortical limbic areas.

The term LIMBIC SYSTEM refers to a group of structures deep within the substance of the brain that have gradually come to be associated in humans with emotional behavior and attitudes and more recently with communication drives. The limbic system incorporates both cortical and subcortical structures, which in lower animals primarily serve the sense of smell and which became converted to other functions as biologic imperatives changed with evolution. Papez (1937) is credited with having provided a theoretical base for the idea that the phylogenetically old rhinencephalon was the source of emotional and motivational behavior. Since then a great deal of research activity has corroborated this concept, including the work of Bard and Mountcastle (1947), Brodal (1947), Pribram and Kruger (1954), and MacLean (1952), who was the first to use the term limbic system.

Lamendella's Review

Motivated by the desire to draw attention to the role of the limbic system in human communication, Lamendella (1977) reviewed the subject in detail and presented some original concepts as a result of his study. Because they bear so closely on the subject matter of this chapter, some of his observations and conclusions are reported here.

Lamendella's major thesis was that the limbic system plays an important role in human social and communicative behavior. In fact, he suggested that this system may be responsible for most human nonpropositional communication activity. He sees the limbic system as poised between and acting on both vegetative (species-preserving) functions, through its connections with the hypothalamus, and the neocortex, which

is involved with those higher level functions that define the human species: social and communicative behavior. Following a review of the anatomy, physiology, and phylogeny of the system as it was then conceptualized, Lamendella presented evidence linking the limbic system to primate social and communication function and then identified five levels of forebrain activity that participate in human communication, three of which are included within the limbic system. The highest level, that of propositional communication, incorporates the dominant and nondominant hemispheres, the latter of which he suggested is closely related to nonpropositional processes that originate in the limbic system and are intertwined with affective subsystems. He suggested that the dominant left hemisphere, in addition to being the locus of propositional speech, appears to play an inhibitory role vis-à-vis affective function.

The emotional lability of patients with left-hemisphere lesions, many of whom are aphasic, is given as evidence of the loss of this inhibitory function. On the other hand, the indifference behavior of some patients with right-hemisphere lesions points to an impairment of the limbic–right-hemisphere system which, according to his concepts, underlies affective function.

What is most relevant to the subject of this chapter is the section of Lamendella's paper relating limbic structure and linguistic communication. He presented two ideas in this regard. First, AUTOMATIC speech is to be distinguished from PROPOSITIONAL speech, the latter being represented in the left hemisphere and, therefore, often disordered when there is damage to that area. He believed that automatic speech may be processed in multiple loci, including limbic, basal ganglia, thalamic, or midbrain structures, and, therefore, may be relatively preserved in a patient with classical aphasia (Bay, 1964; Critchley, 1970; Head, 1926; Jackson, 1932; Luria, 1970). Second, Lamendella stated that one type of automatic speech has referential meaning: that which has emotional content. He believed that such speech (including obscenities and vulgarisms) actually originates in the limbic system because of its affective nature and that these utterances have the dual purpose of relieving affective pressures within the individual and evoking "limbic" responses in the person being spoken to. These affective pressures, in his view, include all of the primitive vegetative functions with which the limbic system is involved (colorfully identified by Pribram, 1971, as the "four Fs").

One additional reference suggests a powerful relationship between emotional and communication functions. This is the observation of B. W. Robinson (1976) concerning a patient who was severely aphasic and right hemiparetic and who subsequently developed a manic-depressive psychosis. During the manic phase, both his motor deficit and aphasia disap-

peared, only to return when the mania was controlled with medication. This remarkable pattern could be repeated by manipulating the medication; that is, when the medication was stopped and the patient became depressed again, aphasia and the motor deficit returned. Robinson interpreted this phenomenon to suggest the existence of two separate speech systems, but Lamendella hypothesized that strong emotional stimuli originating in the limbic system might possibly overcome or circumvent the loss of control over speech and motor systems normally exercised by the left hemisphere.

Summarizing his concept, Lamendella suggested that, although high-level neocortical systems may be largely responsible for the communication abilities now enjoyed by human beings, there is reason to believe that the complex systems for communication developed at lower brain levels down through the phylogenetic strata continue to play a role in human communication. Most germane to our topic is that this role is most relevant where emotions and speech conjoin.

Personal Reports

A fairly substantial literature comprising the personal reports of people who have experienced aphasia should be mentioned because it is appropriate to include material that may heighten one's awareness of the magnitude of the emotional devastation suffered by the person with aphasia.

Eric Hodgins (1964), a writer of great talent, was painfully successful in conveying his terror in the immediate poststroke period and the great frustration, panic, and despair that characterized his life in the months following his stroke. Guy Wint (1967), a journalist, historian, and writer, succeeded in portraying a sense of utter desolation and isolation. One almost experiences his loss of the flavor of life, his fanatic need at first to find a cure for his condition somewhere in the world, and, failing this, his withdrawal into a gray, peopleless existence.

Wint and other writers have mentioned memory problems in their descriptions of their aphasia (Dahlberg, 1977; Moss, 1976; Segré, 1976). One has the sense that they were not talking about word-finding problems. Another recurrent report has been the loss of the ability to dream (Moss, 1976; Wint, 1967), which has not been explained, to the best of my knowledge.

Wint's (1967) writing is so captivating that it can lead to unjustified generalizations. It is likely that his emotional reactions, although not rare by any means, reflected his own personality. However, the loss of the ability to manipulate language in someone whose life was the written word, who was no doubt equally proud of his ability to discourse on subjects of great moment, must have been almost unbearable. An appropriate generaliza-

tion is that language is so much a part of the personality of each of us that its loss goes far beyond the practical inconveniences of impaired communication, whether mild or severe. We are verbal animals, and our capacity for communications is very likely inextricably entwined with both emotional and intellectual function. Lenneberg (1967) made this point in part, suggesting that the biologic roots of language may reside in a person's emotional apparatus.

Wint appears to have possessed an unusual capacity for psychological insight plus an ability to describe his feelings. His description of hallucinatory experiences (1967, pp. 88–89), including olfactory ones, suggests that these are "organic" rather than psychodynamic in origin, sensory processes gone awry as a result of specific brain damage. This idea is in consonance with current thinking that attributes the psychoses to substantive changes in brain physiology (Smythies, 1970). The subject of hallucinosis and delusional phenomena has been well reviewed by Horenstein (1970); however, as with many other studies, the discussion does not pertain specifically to the aphasic patient. His review supports the impression gained from reading Wint (1967) that disordered sensory processes are probably responsible for hallucinations.

Namnum (1995), a psychoanalyst, provided a valuable personal account of his experience with aphasia, touching on many of the important issues discussed in this chapter. "It was not until I was able to emotionally recognize the implications of my condition that I became ready for therapy," he states. (The information he needed to acquire that understanding was given him by a speech pathologist.) He notes that patients may be unable to profit from therapy in the first few months after onset of the stroke without that awareness.

Dr. Namnum describes the phenomena of personal and social isolation, dependency, denial, and depression and speculates that severe depression may be the counterpart of the grief of normal bereavement. Denial and depression he believes are the greatest impediments to aphasia rehabilitation. An important observation, one that may be a source of great frustration in this era of managed care, is that intermittent therapy should continue for many years for optimum results.

The broad range of language and cognitive deficits possible in the person with aphasia makes it difficult to depend on any one account among the many personal accounts as a primary anecdotal source of the experience of living with aphasia (Buck, 1968; Moss, 1976; Newborn, 1997; Segré, 1976; Wulf, 1986).

For example, Moss, who was 43 years old when he acquired global aphasia, emphasized the loss of intellectual ability (memory loss, concreteness, loss of ability to abstract). He described the inability to use words

"internally" early in the course of his illness. Moss stressed the importance of premorbid personality, noting that he had struggled with feelings of inferiority prior to his stroke and found that he used the same compensatory behavior patterns after the stroke as he had before. He concluded that the basic personality does not change.

Segré (1976) did not provide a clear picture of the site of his lesion, although he mentioned an embolus of cardiac origin. Nevertheless, he wrote an excellent review of his psychic and cognitive reactions that includes many of the reactions that have been mentioned thus far. He drew attention to the importance of the attitude of the patient's family, that it be neither overprotective nor anxious, and stated that when the aphasic is encouraged by family and others with an overly optimistic prognosis, he or she may react with feelings of anxiety, depression, and the "complex of inferiority" when confronted with the reality of the situation.

In another personal account of great value because of its insights from a partner's perspective, Montgomery-West (1995) described the experience of living and coping with aphasia in her relatively young husband. She concluded that lack of knowledge about aphasia and its consequences was a great impediment to adjustment. She now devotes most of her time providing such information to aphasic patients and families through the distribution center of the National Aphasia Association.

Current Concepts of the Psychological Sequelae of Aphasia

Depression

Depression is undoubtedly the most important emotional consequence of stroke and aphasia. Some interesting and provocative work in the study of depression in patients with stroke and aphasia has been reported in the recent literature. R. G. Robinson and coworkers have postulated two distinct forms of post-stroke depression (PSD) (Gainotti, 1992; Robinson & Starkstein, 1989; Starkstein & Robinson, 1988):

1. A "major" variety thought to be endogenous (psychotic), seen primarily in patients with left-hemisphere lesions involving the frontal lobe and underlying basal ganglia, hypothesized to be the result of the disruption of monoaminergic pathways linking the brainstem to the cerebral cortex. It is implied that this severe form of PSD is not a psychological reaction to the onset of a catastrophic disorder.
2. A "minor" form that is dysthymic (reactive, neurotic), not based on

the intrahemispheric locus of lesion but on the patient's psychological response to the illness.

Counterarguments to these hypotheses will be reviewed in some detail because of the high incidence and clinical importance of PSD and the evidence suggesting that psychological reactions are the primary cause of post-stroke depression.

One must first call into question the diagnostic distinction of major and minor depression in people who have suffered brain damage. The Diagnostic and Statistical Manual of Mental Disorders of the American Psychiatric Association (DSM) refers to major or minor EPISODES occurring in people with presumably normal brains. The DSM states that in a major depressive episode, in addition to the obvious mood manifestations of depression, there should be other symptoms including sleep and appetite disorders, loss of energy and concentration, psychomotor agitation or retardation, difficulty concentrating, slowed thinking, and a sense of worthlessness. Clearly, such symptoms may have organic or reactive sources, both important factors in someone who has sustained a stroke. The post-stroke patient is in a state of depression; he or she is not having a depressive episode. A number of studies have failed to support the major–minor distinction suggested by Robinson and his coworkers (Gainotti, Azzoni, Lanzillotta, Marra, & Razzano, 1995; Gainotti, Azzoni, Razzano et al., 1997).

In the Gainotti, Azzoni, Razzano et al. (1997) study, a newly designed Post-Stroke Depression Rating Scale was used to determine whether there was a congruence between the major and minor forms of depression described in DSM and the severe and milder forms seen in depressive stroke patients, as proposed by Lipsey, Spencer, Rabins, and Robinson (1986). This study demonstrated that there is a continuum between the so-called major and minor forms of PSD and that reactive psychological factors were dominant regardless of the severity of the depression in PSD. Furthermore, the depressed stroke patients were more anxious, had more catastrophic reactions, and were more depressed in situations that emphasized their disabilities. By contrast, the non–brain-damaged depressives had more feelings of guilt, more suicidal ideation, an inability to enjoy pleasant experiences, and early-morning depression.

What of the proposed relationship between severe PSD and left-hemisphere frontal lesions? Several authors obtained data inconsistent with the hypothesis that there is a correlation (Dam, Pedersen, & Ahlgren, 1989; Gainotti, Azzoni, Lanzillotta, Marr, & Razzano, 1997; Gordon et al., 1991; House, Dennis, Warlow, Hawton, & Malyneux, 1990; Sinyor, Jacques et al., 1986; Stern & Bachman, 1991). Most of these studies have shown a weak

correlation between severity of depression and proximity of the lesion to the frontal pole in both left and right brain-damaged patients. Gainotti, Azzoni, Lanzillotta et al. (1997) found that many patients with aphasia could not be evaluated accurately because of severe language comprehension disorders, suggesting the purported link between severe PSD and left frontal lobe lesions might be the result of a sampling bias, patients with left retro-rolandic lesions having been inaccurately evaluated.

Proponents of the idea that anatomic–physiologic changes associated with stroke are predominant found a weak correlation between severity of depression and the degree of physical impairment in the acute post-stroke period and concluded that chemical, not psychological, reactions to stroke are the primary reason for PSD. (Finkelstein et al., 1982; Folstein, Mailberger, & McHugh, 1977; R. G. Robinson & Price, 1982; R. G. Robinson, Starr, Lipsey, Rao, & Price, 1985; Sinyor, Amato et al., 1986). Similarly, there was a weak correlation between the degree of depression and the loss of social contacts.

These results were markedly different, however, in the chronic stage of the illness at 6 months and beyond. In the chronic stage there was a strong correlation between physical disability, social isolation, and the severity of depression (R.G. Robinson, Starr, Lipsey, Rao, & Price, 1984, 1985). This has suggested that the degree of depression is more dependent on the patients' perception of the consequences of their disabilities than on the disability per se. Lazarus (1982, 1990, 1993) maintains that the adjustment to an important life event depends on the subjective appraisal the individual makes of that event rather than on its objective characteristics. This would appear to be particularly true for the person with aphasia. In the Gainotti, Azzoni, Razzano et al. study (1997), the most severe depression was found in two patients whose aphasia most powerfully affected their lives. One was an attorney with a large practice and the other a university professor with extensive teaching and lecture commitments. Both were extremely conscientious and both considered professional achievement the most important aspect of their lives.

Hermann, Johannsen-Horbach, and Wallesch (1993) proposed a three-stage classification of post-stroke depression. In the acute stage, during the first 2 months after the pathologic insult, depression is a consequence of neuroendocrinological, neurochemical, and/or structural damage to brain tissue. The second stage, encompassing the period of 2 to 6 months post-stroke, is characterized by deepening depression due to awareness of and attempt to cope with the reality of neurological and neuropsychological deficits. The second period was based on the fact that at the time of the study post-stroke patients in Germany were usually hospitalized for 6

months. Depression in the third, chronic stage includes the first two stages plus reactions to professional, familial, and social problems consequent to continuing disability.

Lacking clear-cut evidence that lesion location and biochemical aberrations are the primary basis for depression, it is appropriate to retain the concept that the major factors in the depression of aphasia are the patient's overall reactions to the catastrophe and to specific aphasic deficits, as described earlier. Although it is enticing to hope that the chemistry laboratory will ultimately provide diagnostic and therapeutic answers to the profound problems that characterize the emotional sequelae of aphasia, it appears for the moment that the best diagnostic tools continue to be a knowledge of human behavior and psychopathology. The onset of aphasia is a personal, familial, and social catastrophe that brings with it profound changes in all of these spheres. The spectrum of possible reactions is broad, based on age, education, station in life, economic status, family composition and dynamics, premorbid personality, time since onset, previous history of illness, awareness of deficit, cognitive deficiencies, and other less obvious factors. Because of this bewildering array of variables, the issue of patients' reactions is exceedingly difficult to study.

Denial, Frustration, Depression, and Acceptance

These psychological reactions to a language disorder—denial, frustration, depression, and acceptance—following the grief response model of Kubler-Ross (1969), were suggested by Tanner and Gerstenberg (1988). Loss of one's sense of self is said to lead to these sequential reactions. With regard to denial, one must distinguish between behaviors resulting from specific neurological deficits and those that are based on psychological reactions, in this case to aphasia, as detailed by Herrmann (1997). As stated earlier, denial may be an important defense for the patient in the early stages of illness. Frustration, anger, and unconscious rage are inevitable in the cognitively competent aphasic person and must lead to depression. Acceptance of an altered state such as aphasia is complex and often not achievable. Experienced clinicians have observed that feelings of loss and grief are experienced by many patients. Whether this is a statistically significant reaction is less important than its recognition by the patient's doctor, therapist, and caregiver.

The loss of the sense of self has been explored by Brumfitt (1993). She argues that language plays a critical role in the establishment of one's "sense of self"; the physical and language alterations brought on by the stroke necessitate finding a new identity and aphasia hinders that quest.

Brumfitt concludes that enlightened therapy for the person with aphasia must be directed not only to improvement in linguistic ability but also to the very difficult process of reestablishing identity.

A variety of emotional reactions to aphasia have been repeatedly documented in the literature. Intense anxiety, lack of inhibition, emotional lability, dysphoria, deep sadness, profound discouragement, feelings of inadequacy, worthlessness, self-deprecation, loneliness, indifference, dependency, guilt, and irritability. Charatan and Fisk (1978) documented the fear of the loss of love, of meeting people, of a second stroke, of progressive deterioration, of permanent disability, and of death.

Psychosocial Consequences of Aphasia

One of the most heartening developments in the world of aphasia assessment and management has been the increasing concern about the impact of the disorder on the family of the patient and the vocational and social ramifications of having someone with aphasia in the family. Although concern with this dimension of aphasia goes back many years, it has never been of more focused interest than now. Wepman (1951) and Eisenson (1949) were among the first clinicians to recognize the importance of psychological factors in aphasia. Turnblom (now M. T. Sarno) and Myers described a group discussion program with the families of aphasic patients in 1952; Bjorn-Hansen (1957) wrote about family problems; and J. E. Sarno and Sarno (1969, 1979) published a guide for families based on questions derived from family educational group meetings. The Functional Communication Profile (M. T. Sarno, 1969) and Functional Life Scale (J. E. Sarno, Levita, & Sarno, 1973) emerged from a concern with the effects of impairment on everyday life, which is highly sensitive to patients' psychosocial state. Based on a 2-year study of ethics in rehabilitation medicine conducted at the Hastings Center in 1985, the ethical–moral dilemmas posed by aphasia were addressed in a Hemphill lecture at the Rehabilitation Institute of Chicago (M. T. Sarno, 1986). On that occasion the formation of the National Aphasia Association (NAA) was announced. The NAA's mission is to advocate for persons with aphasia and their families to the end of helping reduce the psychosocial ravages of aphasia and educating the public about the nature of this communication disorder. The development of similar organizations in Finland, Germany, Sweden, England, and Japan started before the NAA was formed (Klein, 1996).

Early family studies reported a variety of spousal reactions to stroke: shock, guilt, bitterness, depression, loneliness, irritability, and the problems associated with having to assume new roles and endure altered so-

cial patterns (Artes & Hoops, 1976; Kinsella & Duffy, 1978). Christensen and Anderson (1989) identified similar post-stroke problems and reported that they were statistically greater in the spouses of patients with aphasia. Williams and Freer (1986) found that spouses experienced a deterioration in lifestyle, emotional support, and sexual relationships. They cited the need for counseling and education about aphasia for spouses regardless of the severity of the aphasia.

The last 20 years have seen a significant increase in the study of the psychosocial affects of aphasia, which Herrmann (1997) has characterized as a change in paradigm for aphasia research and rehabilitation. Some have advanced the idea that the psychosocial sequelae of aphasia are central, not secondary, to the problems posed by the communication disorder (Code & Muller, 1989; Gainotti, 1997; Herrmann, Britz, Bartels, & Wallesch, 1995; Herrmann et al., 1993; M. T. Sarno, 1986; Wahrborg, 1991).

Herrmann and Wallesch (1989) studied the impact of severe nonfluent aphasia on 20 chronic patients and their families and documented negative sequelae in the realms of psychology (depression, resignation, helplessness), professional matters (job, household work, taking care of finances and property), social function (the stigma of aphasia, loss of status and recreational opportunities), and family (role changes, new tasks, new problems, family depression). Using what are now called the Code–Muller Protocols, Code and Muller (1992) also noted that patients and families often had high expectations for eventual recovery that were not shared by the patients' therapists. The authors attributed this to the lack of psychotherapeutic aid or counseling prior to patients' discharge from the rehabilitation facility.

Studying a group of aphasic patients in the first year post-stroke, Herrmann and his associates (1995) found that these patients and their families suffered more intense problems in the professional and social categories than did stroke patients without aphasia. Both aphasic and nonaphasic patients continued to deteriorate in the sphere of professional activities after 1 year.

The problem of social isolation was strongly documented in a survey conducted by the NAA in 1987. Ninety percent of 210 families who responded said that social isolation was their greatest problem. Seventy percent thought that people consciously avoided them. In preparation for a national conference, the NAA contacted 170 Aphasia Community Group leaders across the United States in 1997 to determine what subjects they would like to have addressed at the conference. The most common concerns were, in order of frequency: coping with depression, coping with frustration, coping with anger, the emotional needs of caregivers (attention, support, encouragement), identifying and accepting changes in relationships, and intimacy.

The sensitive area of sexual function in the face of aphasia was reviewed by Letourneau (1993). Assuming a good relationship before the stroke, patients reported difficulty communicating their wants or needs, feelings of rejection, the need for warmth and tenderness, the need to prove the ability to perform, and in some cases trouble controlling a heightened sexual drive. Spouses reported difficulty resisting the patient's hypersexuality, coping with the physical disability, feelings of grief, inability to adjust to the new situation, and in some cases coping with the need to remain faithful.

Quality of life (QOL) is an especially difficult dimension to measure. In the spirit of the new paradigm, however, addressing QOL reflects concern for the PERSON with the aphasia and his or her family. This concept is important to keep in mind for it encompasses many of the important psychological and psychosocial phenomena that are now being studied. A study that attempted to examine QOL in the first post-stroke year reported QOL improvement across all aphasia types (fluent, nonfluent, global) when patients were provided long-term, intensive rehabilitation that addressed language–communication strategies, functional communication, coping skills, and psychosocial issues (M. T. Sarno, 1997).

The ethical and moral issues posed by aphasia rehabilitation include the patient with aphasia as person, the question of who is selected for treatment, the setting of goals, patient autonomy, and resource allocation. As managed care becomes dominant in the United States, these issues are becoming evermore important (M. T. Sarno, 1993; Taylor-Sarno, 1993).

Management and Rehabilitation

We now review some approaches to the management of psychosocial sequelae in aphasia, including some innovative programs. In the quality-of-life study just mentioned, the program followed an individually designed stimulation–compensatory function model on which was superimposed a functional communication perspective. In addition, patients were offered an extensive, varied menu of group therapies designed to develop visual language skills, educate patients and families about the nature of aphasia and recovery, and address issues of coping with role changes, lowered self-esteem, social isolation, and other aspects of the altered self.

Hibbard, Grober, Gordon, Aletto, and Freeman (1990) studied the treatment of post-stroke depression and developed a set of cognitive therapeutic principles based on detailed knowledge of patients' deficits, both physical and nonphysical, including motor and sensory deficits, level of

alertness and ability to concentrate, memory, capacity for abstract thinking, aphasia, visual–perceptual difficulties, and disturbances in the comprehension and/or expression of emotion. In developing the program a variety of factors were considered: awareness of deficits; effect of deficits on thinking and overt personality; and premorbid personality, lifestyle and interests, including whether there had been appropriate mourning of their loss. The authors also noted the importance of knowing about the reactions of family members.

Borenstein, Linell, and Wahrborg (1987) described an intensive 5-day "course" for patients with aphasia and their families, conducted by a speech pathologist, psychologist, and neurologist. The aims were to educate about aphasia, improve language function through stimulation, and identify and attempt to ameliorate personal and interpersonal problems. Follow-up evaluation 1 year later revealed an improved capacity of patients and their families to deal with psychological problems despite little linguistic change.

Stimulated by this report, Hinckley, Packard, and Bardach (1995) set out to develop a similar program in the United States. What evolved was a series of annual conferences called "Opening Doors," whose programs were based on the questionnaire responses of people intending to attend the conference. The responses fell into four categories: a quest for better lifestyles; the need for continuing information about the nature of aphasia and its sequelae; a desire for specific information on how to improve communication ability (e.g., computer applications and augmentive and assistive devices); and the need to talk and learn about coping with the psychosocial ravages of aphasia. Postconference evaluations revealed that attendees improved socially, had a better understanding of aphasia, and had "an improved attitude toward life." They also reported improved coping styles and skills.

Borenstein, Wahrborg et al. (1987) reported experience with eight young people (median age 39.5 years) with chronic aphasia (median months since onset 46.75) who were enrolled in a "Folk High School," a type of institution unique to Scandinavia and Finland, where emphasis is on personal development and social integration rather than on formal schooling. The eight participants were evaluated at the beginning of the course, at 34 weeks, and 10 years later in six domains (Wahrborg, Borenstein, Linell, Hedberg-Borenstein, & Asking, 1997): neurological status, linguistic ability, functional communication proficiency, depression, short-term memory, and quality of life. Although no formal speech therapy was given, all participants showed improvement in linguistic and functional communication proficiency, a tendency to less depression, and a better quality of life

at the end of the 34-week course. In the 10-year follow-up study it was found that in all patients but one the gains in linguistic and functional performance did not persist, but most participants reported a continuing improved quality of life.

In Ontario, Canada, three aphasia centers staffed by speech–language pathologists, social workers, and trained volunteers provide social and psychological services to people with chronic aphasia and their families (Kagan et al., 1995). At the time of writing, they were serving a culturally diverse group of about 360 aphasic families. A large variety of techniques and methods are used to improve linguistic ability and performance, and to enhance the quality of life of patient and family. A guiding principle is that professionals work together rather than in parallel.

Lyon and colleagues (1997) have developed a therapeutic process, identified as Communication Partners, "to restore a sense of purpose, direction and control to daily life for both patient and caregiver." A volunteer from the community is enlisted to be the patient's partner and together with the patient goes through a period of training in which they learn to communicate with each other. A project or activity is then chosen by the patient and the partners set about bringing it to realization. Although formal measures of linguistic ability showed no improvement on testing, patients, their partners, and caregivers noted statistically significant improvement in the patients' well-being and communication ability.

Almost anything one might do to meet the psychosocial needs of patients with aphasia would be welcome after their history of neglect. In general, patients and their families have had to fend for themselves, which undoubtedly has increased their anxiety and depression. Historically, physical concerns have always received priority in medicine, even in the field of rehabilitation medicine, whose avowed philosophy is to concern itself with the whole spectrum of patients' needs. Most physicians training in this specialty are exposed to much more about the sensorimotor sequelae of brain damage than the cognitive, language, or affective disorders. It is perhaps inevitable, then, that the major thrust for remediation in this area would come from speech pathologists, psychologist, psychiatrists, social workers, rehabilitation counselors, and nurses.

Not all of the emotional needs of the aphasic patient (as well as other brain-damaged patients) have been neglected, however. Patients fortunate enough to have received speech therapy have generally found that their therapists have tried to minister to emotional needs in conjunction with their primary task of improving communication skills. Many aphasia therapists have been unsung heroes. Some have sought specialized training in

psychotherapy, or have sometimes enlisted the aid of a psychologist for guidance regarding their interaction with patients.

A successful collaboration of a physician, a speech–language pathologist, and a psychologist is most poignantly illustrated in the following case history. The patient was a married woman in her late 40s who sustained a stroke as a result of a brain hemorrhage. She was severely aphasic, essentially global, and remained so throughout the period described. There was no sensorimotor deficit. At 6 months post-stroke she was profoundly depressed and had disabling pain in both the right upper and lower limbs. As is usual, the pain was attributed to the thalamic syndrome, a poorly defined diagnosis that is inevitably made when a brain-damaged patient has pain. Accordingly, drugs were prescribed, both for the pain and the depression; however, neither problem was relieved, and at one point she attempted suicide.

Her case was reviewed by a rehabilitation team, which concluded that the pain was psychogenic in origin and that, in addition to medication, some form of psychotherapy must be attempted. Her pain was diagnosed as psychogenic regional pain (Walters, 1961), no doubt induced by her unconscious rage at the persistent aphasia. The psychotherapist concluded that the depression resulted from at least three factors:

1. The patient was an exceedingly compulsive, perfectionistic person with a hypertrophied sense of responsibility.
2. In becoming aphasic she believed she had failed her mother, who had always doted on her, and with whom there was a kind of psychological symbiotic relationship.
3. She decided she had also failed her husband, a simple man who had leaned heavily on his wife emotionally. She was the strong, competent one in the family; his primary role was to provide for them financially.

Many painful, laborious months then ensued, during which the psychologist and speech pathologist tried hard to get the patient to understand the reasons for her pain and depression and to establish new perspectives. In the early days of the rehabilitation program, she attempted suicide once more. She was seen by each therapist at least three times weekly; the therapists met frequently to discuss strategies. As expected, the aphasia made psychotherapy difficult but not impossible. Fortunately, the patient's comprehension was better than her expressive skills, and the therapists employed both talking and writing. Medications were continued under the supervision of a psychopharmacologist.

Perseverance was rewarded after many weeks, and the patient gradually emerged from the emotional depths. Although the aphasia remained global, first the pain disappeared, then her mood improved markedly. (As

the patient's depression lifted, her mother's deepened.) The patient gradually came to accept the fact that, although aphasic, she was still a competent person, capable of resuming her roles as wife, companion, and daughter. Eventually she became a volunteer at the hospital and began to travel with her husband. Having no physical disability, she was capable of resuming her role as a homemaker when she was no longer depressed or in pain. Although a few years have passed, she continues to visit the psychologist from time to time.

In addition to the obvious, the case emphasizes the need for a collaborative approach. In this case, speech pathologist, psychologist, physician, and psychopharmacologist worked together. This case also suggests that anatomic–pathologic explanations for symptoms (e.g., pain and depression) may be dangerous, leading to a hopeless prognosis and reliance on drugs as the only therapy available. Furthermore, this patient's story contradicts the commonly held view that psychotherapy cannot be done with a severely aphasic person. A variety of techniques and methods likely can be effective. In addition, the therapeutic value of the sustained efforts of caring professionals is probably beyond measure.

The question of pharmacotherapy for post-stroke depression (PSD) has been well reviewed by Anderson (1997). To our knowledge there are no studies that deal specifically with pharmacotherapy in depressed patients with aphasia. Investigators interested in this subject (Anderson, 1997; Gustafson, Nilsson, Mattsson, Astrom, & Bucht, 1995) have concluded from the available literature that tricyclic antidepressants are contraindicated because of side effects, and that controls do as well as treated patients in the first 7 weeks post-stroke when treated with a selective serotonin reuptake inhibitor, citalopram, but do better with that drug after 7 weeks. It is clear that study of this subject is in its infancy. Future investigation should delineate the time since onset clearly, as PSD may persist for months or years after the stroke or head injury.

One must be impressed with the aphasic's feelings of isolation and loneliness, of traversing a path unknown to the healthy, and of being unable to share the experience with anyone, primarily because of the impaired ability to communicate. Those of us who work with aphasic patients can probably serve them well by demonstrating our awareness of their emotional turmoil through verbalizing for them what they are experiencing. One clinical observation is that patients prefer commiseration to expressions of optimism. If, in addition, as suggested by Baretz and Stephenson (1976), we use strategies designed to help the patient live through the dark days, we will have discharged our clinical responsibilities consonant with the best principles of healing arts.

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18

Recovery and Rehabilitation in Aphasia

MARTHA TAYLOR SARNO

Introduction

Aphasia rehabilitation has been in such a state of flux since the publication of the second edition of *Acquired Aphasia* that it is difficult to describe its current status with accuracy and certainty. Drastic revisions in health service delivery have caused a dramatic downward shift in the accessibility of aphasia rehabilitation to all individuals except those who can support the cost of treatment. When third-party reimbursement is available, its frequency, intensity, and duration are significantly limited. As a consequence, aphasia therapy practices have shifted to meet the challenge posed by this changed health service system. In addition, new categories of patients with communication disorders are seeking services, for example, primary progressive aphasia.

This chapter outlines the approaches and trends that have historically characterized the treatment of aphasia, factors relating to recovery, research issues, and a philosophy of aphasia rehabilitation. A detailed review of the many techniques used in aphasia rehabilitation is not possible in this volume but can be found in a wide range of textbooks and journal articles.

Early Accounts of Recovery and Treatment

Some of the first recorded instances of both natural recovery and intervention were in 1558 by Nicolo Massa and Francisco Arceo whose patients

completely recovered language after surgical intervention following head trauma (Benton & Joynt, 1960). Before the seventeenth century, cautery, cupping, alchemy, and leeching were also tried as interventions for aphasia.

In the seventeenth century, Johann Schmidt (1624–1690) reported on two apoplectic patients with language disturbances whom he treated. One patient recovered letter recognition, and the other achieved full recovery of reading skills with training (Benton & Joynt, 1960).

Samuel Johnson attributed the beginning of his recovery from aphasia in 1783 to the fact that his doctor had "pressed blisters into his back and from his ear to his throat" (Critchley, 1970). Several decades later, Professor Lordat, in France, reported that he reeducated himself in speech and writing to the point where he was able to resume his position as chair of medicine at Montepelier, thereby providing support for his preconceived ideas about the dissociation of speech from thought. Later, his self-observations raised questions about whether he did in fact suffer from aphasia (Bay, 1969; Lordat, 1843).

In the nineteenth century, Thomas Hun, a professor of medicine at Albany Medical College in New York, broke new ground when he reported the rehabilitation of a 35-year-old poststroke aphasic patient (Hun, 1847). Hun recommended that systematic exercises in spelling, writing, and reading be carried out by the patient's wife and he credited her with the patient's recovery. Goodglass (1985) cited an 1879 paper in which a 49-year-old aphasic patient was treated repeatedly by "applying a strong current to his skin with an electric brush."

In the middle of the nineteenth century, Armand Trousseau (1801–1867) treated patients with aphasia using both leeches and reeducation techniques. At about the same time, H. C. Bastian, an English neurologist, employed methods used for teaching the deaf. Like many of his peers, Bastian postulated a model that embraced two types of recovery: spontaneous and compensatory (Howard & Hatfield, 1987). In 1890, Bateman suggested that there was "sufficient evidence that re-education is a valuable means to re-establish man's noblest prerogative—the faculty of articulate language" (1890, p. 229).

Paul Broca was one of the first to discuss the feasibility of retraining in aphasia. In his paper, "*Du siège de la faculté du language articule*" (1885), Broca described retraining experiences with teaching methods in use at French schools at the time. He theorized that because the left hemisphere is differentially developed for language and other intellectual processes, it should be possible to teach language to someone with aphasia in the same way one teaches a child. In this regard, Broca felt that much time was required for aphasia retraining, because children learn to speak over a long

period of time and adults might not learn as easily after a certain age (Howard & Hatfield, 1987).

At the turn of the century, German and Austrian physicians and psychologists were playing a key role in the development of disciplines that addressed communication disorders. Hermann Gutzmann (1865–1922), a phoniatrist, treated aphasic patients beginning in the late nineteenth century and later developed a center in Berlin for posttraumatic combat veterans during the first World War (Howard & Hatfield, 1987).

During the late nineteenth and early twentieth centuries the literature on the treatment of aphasia was found almost exclusively in German publications. Charles K. Mills published one of the first papers in English that addressed recovery and rehabilitation in aphasia (1904). He reported training a poststroke aphasic patient whom he and Donald Broadbent saw in a London hospital (Broadbent, 1879; C. K. Mills, 1880, 1904). Mills also described an experience with a 45-year-old physician patient with whom he, T. Weisenberg, and the patient's secretary systematically worked over a period of 2 years. In this case, the "physiological alphabet" designed by Wyllie (1894), essentially an articulatory–phonetic approach, was the basis for training.

Although Mills' work took place more than a century ago, his observations and approach to aphasia rehabilitation are remarkably similar to many of the practices and ideas that have been in use ever since. His early paper is notable in that it showed concern with some nonlinguistic aspects of the patient's rehabilitation (i.e., emotional factors, premorbid intelligence, and education). He discussed the possible influence of semantic, lexical, and cognitive factors in recovery, suggesting that different methods were appropriate for different patients and syndromes. Finally, he noted that aphasia after trauma had a better outcome than after cerebrovascular accident (CVA), that not all patients benefit to the same degree from retraining, and that spontaneous recovery may influence the course and extent of recovery.

During World War I several hospitals were established for the treatment of the brain injured, particularly in Germany, despite the fact that the treatment of aphasia was not generally accepted by neurologists (Goldstein, 1942). Emil Froeschels, a physician and follower of Gutzmann, with expertise in psychotherapy, emigrated to the United States at the beginning of the second world war, where he later founded the International Association of Logopedics and Phoniatrics. He gave a report on aphasia rehabilitation at the first meeting in 1924. Between 1916 and 1925, Froeschels treated a large number of brain-injured patients with aphasia (Schuell, Jenkins, & Jimenez-Pabon, 1964). In the same period, Isserlin (1929) had a program

in Munich, Poppelreuter (1915) in Cologne, and Henry Head (1926) published a two-volume treatise on aphasia based on his experience in England with 26 posttraumatic World War II patients. Frazier and Ingham (1920), Gopfert (1922), and Franz (1924) also reported experiences retraining aphasic patients during this period.

Kurt Goldstein's experiences and observations in Frankfurt, especially at the Institut Zur Erforschung der Folgeerscheinungen von Hirnverletzungen, provided one of the most comprehensive descriptions of the systematic treatment of a large number of head-injured patients, of whom 90 to 100 were followed for a 10-year period (Goldstein, 1942, 1948). Goldstein viewed aphasia rehabilitation as a process of intervention intended to help the patient develop compensatory communication strategies and reduce the stress of the struggle to cope with a changed personality and a communication disorder. His approach was highly individual, depending on the communication disorder and the patient's personality.

Singer and Low (1933) reported on a 39-year-old aphasic woman who suffered a vascular brain infarct after a full-term delivery. She showed continuous language improvement with consistent training over a 10-year period. This was among the first reports of retraining in aphasia of vascular etiology. Weisenburg and McBride's (1935) landmark 5-year study of 60 primarily poststroke aphasic patients who were younger than 60 years of age made little reference to recovery but did comment on the effectiveness of reeducation, concluding that reeducation increased the rate of recovery, assisted in facilitating the use of compensatory means of communication, and improved morale.

In England, Butfield and Zangwill (1946) followed the recovery course of 66 primarily posttraumatic aphasic patients and reported that speech was judged to be significantly improved, both in those who began therapy less than 6 months after onset (half of the group) and in those who began therapy more than 6 months after onset (one third of the group). The best outcome was obtained in those with aphasia due to trauma rather than due to other causes.

One of the first comprehensive reports of aphasia rehabilitation to emerge from World War II was Joseph Wepman's (1951) book based on data obtained from 68 aphasic patients (mean age = 25.8) who began retraining 6 months posttrauma at the Aphasia Center of DeWitt General Hospital in Auburn, California. In Wepman's study aphasic patients made a gain of better than five school grades in language skills, and he observed that premorbid personality had a profound influence on outcome. He also reported that patients with expressive aphasia, followed by those with receptive and global aphasia, recovered the highest levels of language performance. Wepman (1951) drew attention to the large population of un-

treated civilians who, like the combat veterans, might also benefit from therapy.

Luria (1948) also reported the outcome of a large series of patients with traumatic aphasia treated at the Institute of Neurology, Academy of Medical Sciences, in Russia. He concluded that systematic retraining based on a careful psycholinguistic analysis and aimed at the reorganization of functions provides the foundation for the successful restoration of verbal skills.

In the last quarter of the twentieth century, the volume of clinical and research activity addressing issues of recovery and rehabilitation has multiplied many times. Furthermore, a significant increase in the average life span, stroke survival rate, and incidence of aphasia resulting from head injuries sustained in vehicular accidents, together with an increase in the size of the general population since World War II, have enlarged the pool of individuals with acquired aphasia to a current estimate of 1.5 million persons in the United States (National Institutes of Health, 1990). Stroke alone causes an estimated 100,000 new cases of aphasia every year in the United States (Damasio, 1992). As a result, the facilities offering aphasia rehabilitation services, the majority of which are located in hospitals and rehabilitation centers, have grown in number and scope. In the post-World War II period, the field of speech-language pathology grew rapidly. Membership in the American Speech-Language-Hearing Association (ASHA) increased from 1623 in 1950 to 68,528 in 1997.

A new medical specialty, the American Board of Physical Medicine, was established in 1947. In the following year, Dr. Howard A. Rusk set out to develop medical rehabilitation as a specialty, separate from physical medicine, where the emphasis was on the restoration of overall life function and the psychosocial sequelae of chronic illness. By 1953, physical medicine and medical rehabilitation were joined and the American Board of Physical Medicine and Rehabilitation was established. In the 1960s, partly through his role as medical editor of *The New York Times*, Rusk succeeded in increasing public awareness of the needs of the disabled and was instrumental in the establishment of government grants for the training of large numbers of physicians and other health professionals in rehabilitation medicine. This specialty is now an integral part of the health care system and specialized rehabilitation centers and programs in hospitals and in skilled nursing facilities can be found across the nation.

Changing attitudes about chronic disease, the hope of modern medicine and technology, and a more vocal lay press continued to enhance the demand for aphasia rehabilitation services. Reports appeared in the press about public figures with stroke-induced aphasia, such as Sir Winston Churchill in 1953, President Dwight D. Eisenhower in 1957, and Ambassador Joseph Kennedy in 1961. Within a few days of President Eisenhow-

er's stroke, Eugene J. Taylor, a medical writer and close associate of Rusk, wrote a series of landmark articles that appeared in *The New York Times* describing stroke, particularly aphasia, to the lay public. The presence of an article about aphasia and stroke on the front page of a prominent newspaper represented a groundbreaking event and may have been the first time that the term *stroke* was used in a serious article which had wide public exposure.

Informational publications designed for families and friends of patients with aphasia also appeared for the first time after World War II and served as a catalyst for many others which followed (American Heart Association, 1969; Backus, Henry, Clancy, & Dunn, 1947; Boone, 1965; J. E. Sarno & Sarno, 1969, 1979; Simonson, 1971; Taylor, 1958). Workbooks by Longerich and Bordeaux (1954) and Taylor and Marks (1955, 1959) were among the first of similar materials now available. Personal accounts of living with aphasia followed and have provided rich documentation of the experience (Buck, 1968; Cameron, 1973; Dahlberg & Jaffee, 1977; Hodgins, 1964; Lavin, 1985; Moss, 1972; Newborn, 1997; Ritchie, 1961; Wint, 1967; Wulf, 1973).

The development of speech pathology as a health profession, the emergence of rehabilitation medicine as a medical specialty, the mass media explosion, a larger and more affluent middle class, and increased public expectations of medicine in an age of technology, all undoubtedly contributed to the advent of aphasia rehabilitation services as an integral part of health care. Concurrent with these changes, chronic disease and disability, no longer taboo, were openly and widely discussed.

In summary, interest in recovery and rehabilitation of aphasia was minimal before the second world war and limited primarily to the management of wartime missile wound casualties. Since then, changing attitudes toward the acceptance of the disabled, combined with the expectations and demands of an affluent society, have provided the impetus for much research and clinical activity in many disciplines.

Research Issues in Recovery and Rehabilitation

Research on recovery and rehabilitation is complicated both by the difficulties imposed by how investigators define RECOVERY and by methodology issues. Few studies are based on gains measured across the range of domains that contribute to effective communication, that is, the linguistic, cognitive, functional, psychosocial, and quality-of-life dimensions of communication behavior. Subject selection criteria; subject classification; aphasia severity; characteristics of control groups; premorbid language compe-

tence, age, education, socioeconomic status, intellectual level, and personality of patient; time since onset; frequency and intensity of treatment; nature of interventions; and the skill of the therapist are some of the factors which must be considered in aphasia recovery and rehabilitation research. In addition, these variables interact with each other and cannot be studied in isolation. The appropriateness of the measures used, the generalizability of the findings, and the method of subject accession and selection are among the many factors that require consideration in aphasia research design. The virtual impossibility of finding homogeneous groups of patients when testing the effectiveness of treatment methods limits the validity of all studies. It is also difficult, if not impossible, to obtain comparable control groups of patients as it is an ethical imperative that all patients with aphasia have the opportunity to receive treatment. The issues that must be addressed in the design of research in aphasia rehabilitation have been discussed in the literature. Many of these issues have lead to a controversy as to whether single-subject or group-designed studies are the best method for studying the efficacy of aphasia treatment (Caramazza & McCloskey, 1988; Coltheart, 1983, 1991; Darly, 1972; David, 1989; Howard, 1986; Pring, 1986; Zurif, Gardner, & Brownell, 1989).

Defining Recovery

The word RECOVERY, used literally, means to return to a normal state. However, to those who study aphasia, the term has been used to refer to a spectrum of positive changes ranging from specific improvement in isolated verbal tasks to the restoration of premorbid communication function. It is sometimes used in the early stages of language rehabilitation, when the word IMPROVEMENT would be more appropriate. In general, few patients who have aphasic symptoms at 3 to 4 months postonset have complete recovery of premorbid language function.

Patients with aphasia assign a wide range of interpretations to the term "recovery." Most do not consider themselves "recovered" unless they have returned to premorbid levels of language use (Yarnell, Monroe, & Sobel, 1976). The meaning assigned to recovery can be based on a patient's subjective perception, a caregiver's perception of communication burden, change scores on language tests, and functional communication ratings derived from direct observation or inferred from a limited sample. Recovery can also include the degree to which the patient is integrated into family and community life.

Part of the difficulty posed by any assessment of recovery is the consequence, in part, of the many sequelae of aphasia that reach beyond the lin-

guistic domain. For the great majority, aphasia has the immediate effect of altering personal identity, self-perception, ability to interact easily in speaking situations, mood, activational level, and a host of other reactions and realities that impede the possibility of living as before and/or finding life fulfillment. Furthermore, improvement usually occurs at different rates in different domains. For example, a patient may recover the use of certain syntactic operations, but be so impaired in speed of performance or degree of initiation that for all practical purposes these skills are not useful in day-to-day interactions. As a result, even restored skills may not be perceived as recovered.

In the final analysis, the best definition of recovery should go beyond language improvement and include patients' perceptions of the quality of their lives, their sense of well-being, their emotional state, and the presence of adequate support. Measures of life function that include activity levels, socialization, mobility, and community reintegration may be used for this purpose (M. T. Sarno, 1997).

Spontaneous Recovery

Although it is generally agreed that spontaneous recovery occurs in the majority of patients in the hours, days, or weeks immediately following onset, the duration of the spontaneous recovery period remains controversial. Some investigators report significant spontaneous improvement in the first 2 to 3 months (Basso, Faglioni, & Vignolo, 1975; Kertesz & McCabe, 1977; Lomas & Kertesz, 1978; M. T. Sarno & Levita, 1971; Vignolo, 1964), whereas others (Hagen, 1973; Lendrem & Lincoln, 1985; Luria, 1963; Sands, Sarno, & Shankweiler, 1969; Wepman, 1951) report spontaneous recovery up to 6 months postonset. In a survey of 850 acute poststroke aphasic patients, 74% improved and 44% cleared in the 4- to 12-week poststroke period (Brust, Shafer, Richter, & Bruun, 1976). Although spontaneous recovery is probably complete in most patients in 6 months, aphasia clinicians are aware of the occasional patient who seems to improve without treatment for months beyond that time. This poses a problem in designing research that addresses the efficacy of aphasia treatment.

Predicting Recovery

Both clinical and demographic factors have been considered predictors of recovery. However, Basso's (1992) review of this topic concludes that factors such as age, gender, and handedness do not affect recovery from aphasia. Although age has been reported as significant (Holland, Greenhouse,

Fromm, & Swindell, 1989; Sands et al., 1969; Vignolo, 1964), many do not support this view (Basso, 1992; Basso, Capitani, & Vignolo, 1979; Kertesz, 1984; Pedersen, Jorgensen, Nakayama, Raaschov, & Olsen, 1995; M. T. Sarno, 1992; Wertz & Dronkers, 1988). Gender does not appear to have an important influence on outcome (Basso, 1992; Borod, Carpwe, & Naeser, 1990; M. T. Sarno, Buonaguro, & Levita, 1985). The finding that traumatic aphasia carries a better prognosis than vascular aphasia may be influenced by the fact that traumatic patients are generally neurologically healthy, whereas patients who have had strokes may have widespread vascular involvement (Basso, 1992).

Type and severity of aphasia appear to carry predictive value, with global aphasia having the poorest prognosis (Basso et al., 1979; M. T. Sarno & Levita, 1979; Schuell et al., 1964; Selnes, Knopman, Niccum, Rubens, & Larsen, 1983). Basso et al. (1979) reported that when fluent and nonfluent patients of the same severity were compared there were no differences in degree of recovery. In 881 consecutive acute stroke admissions to a community-based hospital, it was possible to make valid prognoses within 1 to 4 weeks after stroke depending on the initial severity of aphasia (Pedersen et al., 1995). Global aphasia sometimes evolves to severe Broca's aphasia when there is significantly improved comprehension. Broca's aphasia may become anomic aphasia, and Wernicke's aphasia may evolve to anomic or conduction aphasia (Kertesz, 1981a; Kertesz & McCabe, 1977; Pashek & Holland, 1988).

Patients whose computerized tomography scans show large dominant hemisphere lesions, many small lesions, or bilateral lesions are less likely to recover than those with smaller or fewer lesions (Kertesz, 1979; Yarnell et al., 1976). Lesions in Wernicke's area or those that extend more posteriorly tend to lead to severe and persistent aphasia (Ludlow et al., 1986; Mohr et al., 1978). The neuroradiologic correlates of aphasia recovery have been addressed by other investigators as well (Demeurisse, Verhos, & Capon, 1984; Goldenberg & Spatt, 1994; Kaplan, Goodglass, & Weintraub, 1983; Knopman, Selnes, Niccum, & Rubens, 1984; Knopman et al., 1983; LeMay, 1977; Selnes et al., 1983).

Comprehension tends to recover to a greater degree than expression (Basso, Capitani, Zonobio et al., 1982; Kenin & Swisher, 1972; Lebrun, 1976; Lomas & Kertesz, 1978; Prins, Snow, & Wagenaar, 1978; Vignolo, 1964). Patterns of recovery have been addressed by Kenin and Swisher (1972), Reinvang and Engvik (1980), and C. M. Shewan (1988). In a controlled study by Ludlow (1977), patients did not develop a new and simplified language system in connected speech but tended to recover the same structures used premorbidly.

Educational level or occupational status before illness do not always correlate with recovery (Keenan & Brassell, 1974; M. T. Sarno, Silverman,

& Sands, 1970; Smith, 1971). On the other hand, M. T. Sarno and Levita (1971) reported that aphasic individuals who were employed at the time of stroke recovered more than those who were unemployed.

The presence of depression, anxiety, and paranoia have been cited as negative factors in recovery (Benson, 1979a, 1979b, 1980; Damasio, 1992; Lebrun, 1980; M. T. Sarno, 1993). Premorbid traits have been identified as important prognostic factors (Eisenson, 1973; Herrmann, Britz, Bartels, & Wallesch, 1995; Wepman, 1951). Eisenson (1949, 1964, 1973) felt that patients with outgoing personalities had a better prognosis than those with introverted, dependent, or rigid personalities.

Time since Onset and Recovery

Time since onset is often noted as an important recovery variable (R. C. Marshall & Philipps, 1983; M. T. Sarno & Levita, 1979). The general consensus has been that language gains take place earlier rather than later. Sands et al. (1969) found greater improvement in a group of patients who began treatment up to 2 months poststroke when compared to a group that started treatment after 4 months. Reinvang and Engvik (1980) reported a significant degree of improvement in the period 2 to 6 months postonset. In the Veterans Administration Cooperative Study, the deferrment of treatment for 12 weeks did not influence outcome (Wertz et al., 1986). Basso et al. (1975) reported the least recovery in patients with the longest duration of symptoms, and Prins et al. (1978) noted significant changes in spontaneous speech variables in the first year poststroke.

In a study of 34 treated poststroke aphasic patients who were systematically examined during the first year, little change was noted in the 4- to 8-week period. However, in the 12- to 26-week period, improvement was noted on all measures. The greatest changes were made by the global group and the smallest gains by the fluent group, which was the reverse of the findings in the 3- to 6-month period. The primary study finding was the continuation of improvement in all patients up to 1 year poststroke (M. T. Sarno & Levita, 1979). Gains after the first year poststroke were noted by Marks, Taylor, and Rusk (1957) and Sands et al. (1969), whereas Kertesz and McCabe (1977) reported little or no change after the first year.

Approaches to the Treatment of Aphasia

Therapeutic intervention has been based on theoretical concepts of the nature of language and the pathology of aphasia. Treatment approaches

have generally followed one of two models, a SUBSTITUTE SKILL MODEL or a DIRECT TREATMENT MODEL, both of which are based on the assumption that the processes that subserve normal performance need to be understood if rehabilitation is to succeed (Goodglass, 1987). Treatment methods can be grossly categorized as those that are largely indirect stimulation–facilitation and those that are essentially direct, structured, and pedagogic (Benson, 1979b; Burns & Halper, 1988; Darley, 1975; Kertesz, 1979; M. T. Sarno, 1980; Taylor, 1964a). Howard and Hatfield (1987) identify eight schools of aphasia intervention, which are used here as a framework for discussion.

The Didactic School

Language skills are essentially retaught based on traditional methods of teaching reading, writing, and grammar in the classroom, frequently arrived at intuitively rather than according to a preset structure.

The Behavior Modification School

Therapy is again viewed as a relearning process and the emphasis is on method, that is, HOW the treatment is administered. This school follows the classic Skinnerian model of reinforcing correct responses and using carefully graded small steps. Intervention based on this model was popular in the 1960s (Edwards, 1965; Filby & Edwards, 1963; Goodkin, 1968; Holland, 1967, 1968, 1969, 1970; H. Lane & Moore, 1962; Rosenberg, 1965; Taylor, 1964b; Tikofsky & Reynolds, 1963).

The Stimulation School

Intervention is viewed as a process of providing the appropriate stimulation to facilitate regaining access to language abilities that are preserved. Followers of this school do not believe that language is relearned but rather that it is facilitated. The work of Schuell et al. (1964) and Wepman (1951, 1953) are examples of this approach.

The Reorganization of Function School

This approach views language ability as a set of independent subsystems which requires the patient to learn to use preserved subsystems to bypass those that are impaired. Luria (1963, 1966), a strong proponent of this model, identified three types of restoration: de-inhibition of temporarily depressed functions, substitution of the opposite hemisphere in language remediation, and radical reorganization of functional systems.

The Pragmatic School

Followers of this model view aphasia as more of a communication problem than a language problem and concentrate on using residual functions to compensate for impairment. The Functional Communication Treatment (FCT) described by Aten, Caligiuri, and Holland (1982) falls into this category. Emphasis is placed on the restoration of communication in the broadest sense by focusing on information processing in everyday life activities.

The PACE (Promoting Aphasics Communicative Effectiveness) method (Davis & Wilcox, 1985) is an approach intended to reshape structured interactions into more natural communicative exchanges, including several pragmatic components common to natural conversation. The PACE approach emphasizes strategies over linguistic performance and patients are encouraged to convey a message in whatever way possible using verbal and nonverbal means.

The Neoclassical School

Generally speaking, the strategy here is to access intact linguistic abilities by a variety of alternative methods. There is considerable variation among the followers of this school as to the importance of neurological lesion localization. Nevertheless, a number of innovative therapeutic programs have been developed (Goodglass & Kaplan, 1972).

Howard and Hatfield (1987) categorize Melodic Intonation Therapy (MIT; Sparks, Helm, & Albert, 1974), Visual Communication Therapy (VIC; Gardner, Zurif, Berry, & Baker, 1976), and Visual Action Therapy (VAT; Helm & Benson, 1978; Helm-Estabrooks, 1983; Helm-Estabrooks, Fitzpatrick, & Barresi, 1982) in the neo-classical school.

MELODIC INTONATION THERAPY

Based on the observation that language may not be available in spontaneous speech but can sometimes be produced in association with an intoned melody, the system presumes an intact right hemisphere, thought to be the locus of melodic production. In a series of carefully graded steps, the therapist slowly introduces melody, rhythm, and verbal content, gradually including the patient's participation and diminishing his or her own, which if successful leads ultimately to speech. The authors state that the best candidates for this approach are those with good auditory comprehension, facility for self-correction, markedly limited verbal output, reasonably good attention span, and emotional stability.

VISUAL COMMUNICATION THERAPY

The VIC approach, derived from the early work of Glass, Gazzaniga, and Premack (1973) and Gardner et al. (1976), is a technique designed for global aphasia that uses an index card system of arbitrary symbols representing syntactic and lexical components. Patients are trained to recognize the symbols and manipulate them so as to respond to commands and express needs, wishes, or emotions. Experimental evidence based on the use of the VIC approach suggests that the cognitive operations entailed in natural language are preserved despite severe aphasia. An adaptation and application of the VIC system called the Computer-Aided Visual Communication system (C-VIC) was developed by Steele and coworkers (Steele, Weinrich, Kleczewska, Carlson, & Wertz, 1987; Steele, Weinrich, Wertz, Kleczewska, & Carlson, 1989; Weinrich, Steele, & Illes, 1985; Weinrich, Steele, Carlson, & Kleczewska, 1989; Weinrich, Steele, Kleczweska et al., 1989). Weinrich, McCall, Weber, Thomas, and Thornburg (1995) demonstrated that C-VIC training can lead to improved spoken language.

VISUAL ACTION THERAPY

This method was designed to train global aphasic patients to use symbolic gestures representing visually absent objects (Helm & Benson, 1978; Helm-Estabrooks et al., 1982). The tasks used in this approach include associating picture forms with specific objects, manipulating real objects appropriately, and, finally, producing symbolic gestures that represent the objects used (e.g., cup, hammer, razor).

HELM-ELICITED LANGUAGE PROGRAM FOR SYNTAX STIMULATION

A syntax training program called the Helm-Elicited Language Program for Syntax Stimulation (HELPSS; Helm-Estabrooks, Fitzpatrick, & Barresi, 1981) consists of a hierarchy of constructions that are elicited first by the repetition of a sentence produced by the therapist as a story completion, and then generated as a completion by the aphasic patient alone.

AMERICAN INDIAN SIGN LANGUAGE

Howard and Hatfield (1987) categorize American Indian Sign Language (Amerind) and other gesture systems under the neoclassical school. Amerind has been reported as an effective technique in selected cases (Rao et al., 1980; Rao & Horner, 1978; Skelly, 1979; Skelly, Schinsky, Smith, & Fust, 1974). Others have described variations of sign language intervention (Kirshner & Webb, 1981; Moody, 1982). Coelho and Duffy (1987) re-

ported a significant relationship between the severity of aphasia and success in acquiring and generalizing manual signs.

DRAWING

Several investigators have used drawing as a potential means of communication (Hatfield & Zangwill, 1974; Lyon, 1995; Rao, 1995). In addition, narrative discourse has begun to be explored as a basis for language therapy (Ulatowska & Chapman, 1989).

NEW TECHNOLOGY

New technology, especially synthetic speech and microcomputers, can serve either as an alternative/augmentative means of communication or as a facilitory technique to enhance treatment. A review of the topic can be found in Kratt (1990). The use of alternative/augmentative communication systems and devices have been reported by Seron, Deloche, Monlard, and Rousselle (1980), Beukelman, Yorkston, and Dowden (1985), Colby, Christinaz, and Graham (1978), Colby, Christinaz, Parkinson, Graham, and Karpf (1981); Johannsen-Horbach, Cegla, Mager, and Schempp (1985), Garrett, Beukelman, and Low-Merrow (1989); Doyle and DeRuyter (1995); V. W. Lane and Samples (1981), Bailey (1983), Katz and Nagy (1983), R. H. Mills (1982), R. H. Mills and Hoffer (1985), and Bruce and Howard (1987).

Computers are also frequently used as a facilitation tool for the restoration of language skills. For example, software has been designed specifically to train a broad range of language skills by reinforcing through feedback and frequent repetition and practice. In light of the contemporary restrictions on the provision of treatment by aphasia specialists, computer facilitation as an intervention tool can play an important role both in formal treatment programs and in home practice. However, the selection of software requires professional guidance.

The Neurolinguistic School

Scargill (1954), Morley (1960), Pincus (1965), and Luria (1963, 1970) were among the first to use a linguistic model in the theory and practice of recovery and rehabilitation in aphasia. Jakobson's (1955, 1956, 1961, 1964) elaboration of his own linguistic theory of aphasia motivated some therapists to apply linguistic principles to aphasia intervention. The origins of the linguistic subspecialty called neurolinguistics has been attributed to the neurologist Hécaen and to the linguist Dubois, who in 1969 proposed establishing a field of study whose objective would be to analyze neurologically caused verbal disorders and establish a "purely linguistic typology" (Hécaen & Dubois, 1971; Howard & Hatfield, 1987). Beyn and

Shokhor-Trotskaya (1966) reported success in preventing the appearance of "telegraphic style" responses in 25 poststroke aphasic patients by avoiding the teaching of nominative words. Nouns were introduced only when they appeared in a patient's spontaneous speech. Wiegel-Crump (1976) also reported significant improvement in syntax as a result of a linguistically based treatment program. More recently, the work of Weniger, Huber, Stachowiak, and Poeck (1980), de Bleser and Poeck (1985), Poeck, Huber, and Willmes (1989), and their colleagues continue to explore neurolinguistic approaches to the treatment of aphasia.

The Cognitive-Neuropsychology School

Followers of this school contend that an information-processing model should be the basis of deficit analysis (diagnosis) and intervention in aphasia and that the traditional practice of classifying individuals with aphasia into syndrome categories is insufficient. The assumption is that language processing is organized in a modular fashion, that is, each of the components of cognition (i.e., language processing) is independent (Fodor, 1983). Studies of cognitive processing in aphasic subjects have been used to generate models of normal language.

Although it is generally agreed that cognitive neuropsychology has an important role in the analysis of language impairment in aphasia, controversy persists with regard to its role in the rehabilitation of aphasia. Caramazza (1989), Caramazza and Hillis (1993), and Hillis (1994) have challenged the possibility that "the cognitive neuropsychologist can contribute much to a theoretically driven approach to remediation." The cognitive neuropsychology school advocates assessing each patient's pattern of performance, analyzing his or her performance according to a processing model, and designing treatment that addresses the underlying cognitive-neuropsychological deficits (Berndt, Haendiges, Mitchum, & Sandson, 1987; Byng, 1988; Byng & Black, 1995; D. Caplan, 1993; Hillis, 1994; J. Marshall, Chiat, & Pring, 1997; Schwartz, 1984; Schwartz, Saffran, Fink, Myers, & Martin, 1994; Thompson, Raymer, & LeGrand, 1991; Thompson, Shapiro, & Roberts, 1993; Thompson, Shapiro, Ballard, Jacobs, Schneider, & Tait, 1997). For a detailed review of mapping theory and therapy, see J. Marshall (1995). Chapter 7 by Rita Berndt in this volume reviews mapping and other sentence-processing interventions of the cognitive-neuropsychological school.

To some extent, a formalized, all-encompassing categorization of the "schools" of aphasia treatment is impossible. Some approaches, especially some recent interactive models, were not included in the Howard and Hatfield (1987) account. The Communication Partners approach (Lyon, 1992,

1995, 1996, 1997) and the Supported Conversation model (Kagan, 1995b; Kagan & Gailey, 1993) are examples. The Conversation Partners (Lyon, 1992, 1995, 1996, 1997) treatment plan is designed to enhance communication and well-being in settings where the aphasic person and caregiver live. The approach depends on the participation of a volunteer from the community in which the person with aphasia resides as a conversation partner. The volunteer and patient are trained in ways of effectively interacting with each other that maximize the aphasic person's communication performance to restore the individual's sense of self and thereby increase the possibility of social experience and community interaction.

The Supported Conversation (SCA) model introduced by Kagan (1995b; Kagan & Gailey, 1993) is based on the idea that aphasia masks competence normally revealed through conversation. When an individual has difficulty talking and understanding what is said, it is hard to see the active mind and difficult to envision the person's capacity to make life decisions or be regarded as a social being. These perceptions have a negative impact on the way one is treated. As a result, many individuals with aphasia have greatly reduced access to social interactions which depend on conversation, which in turn leads to social isolation and a loss of the individual's place in society. At the North York Aphasia Center in Ontario, volunteers are trained as conversation partners for chronic aphasic individuals. The Supported Conversation approach was designed to train caregivers, health professionals, and volunteers to facilitate conversation in the aphasic adult using all available modalities and activities (e.g., gestures, role playing) in order to reveal the individual's competence and thereby permit interaction.

N. Simmons-Mackie (1993) presents a social model of aphasia that focuses on the fullfilment of social needs (e.g., the need to affiliate socially, to "look good"), which she views as a preferred course of intervention over traditional aphasia therapy because traditional therapy does not prepare the person for life in the community. Simmons-Mackie distinguishes between "transaction," which involves the exchange of information, opinions, and feelings, and "interaction" which involves social connection. The approach is entirely interactive and encourages the use of compensatory strategies and the assumption of a greater conversational burden on the part of communication partners. Partners are specifically trained to facilitate interaction through a dynamic program aimed at changing some of the communication partner's interactive behavior. The approach attempts to approximate the flow of natural interactions as close to natural discourse as possible, avoiding the use of strategies that might interfere with interaction in certain social settings (N. Simmons, 1993; N. N. Simmons-Mackie & Damico, 1995, 1997).

Treatment of Speech Dyspraxia

Speech dyspraxia is seldom, if ever, manifest in the absence of a coexisting Broca's aphasia, however mild. The condition is especially amenable to direct therapeutic intervention approaches using traditional articulation therapy, including stress and intonation drills. This includes techniques to improve articulation accuracy through imitation, stress, and intonation drills. Exercises generally begin with nonoral imitation, followed by sounds, words, phrases, and finally utterances. Descriptions of techniques abound in the literature (Deal & Florance, 1978; Dronkers, Redfern, & Shapiro, 1993; Halpern, 1981; McNeill & Kent, 1990; Rosenbek, 1984, 1985; Rosenbek, Lemme, Ahern, Harris, & Wertz, 1973; Rosenbek, Kent, & LaPointe, 1984; Rubow, Rosenbek, Collins, & Longstreth, 1982; Square-Storer & Apeldoorn, 1991; Wertz, 1984; Wertz, LaPointe, & Rosenbek, 1984; Wiedel, 1976). Sands, Freeman, and Harris (1978) reported a case of a Broca's aphasic patient with speech dyspraxia who received speech therapy for 10 years. In this case, features of place and manner of production improved, although voicing and addition errors persisted and errors of omission were virtually eliminated.

Pharmacological Intervention

The potential effect of pharmaceutical agents on language performance in aphasia has been addressed by some investigators (Albert, Bachman, Morgan, & Helms-Estabrook, 1988; Darley, Keith, & Sasanuma, 1977; Gupta & Mlcoch, 1992; Linn & Stein, 1946; Sabe, Leiguarda, & Starkstein, 1992; Tanaka, Miyazaki, & Albert, 1997; West & Stockel, 1965). Certain features of aphasia may be amenable to pharmacologic intervention (Bachman & Albert, 1990). In spite of some promising findings in the use of catecholaminergic agonists, D-amphetamine, and bromocriptine, further research is needed before these can be considered efficacious treatments for aphasia (Mlcoch & Gupta, 1995).

Group Speech Therapy

Group therapy has been used in aphasia intervention for several decades either in conjunction with individual therapy or as the only form of treatment (Backus & Dunn, 1947; Bloom, 1962; Corbin, 1951). It is the preferred treatment form for some approaches (Kagan, 1995a; N. N. Simmons-Mackie & Damico, 1995, 1997). The goals of group therapy relate to improving interactive communication, peer interaction, and conversation-

al practice, whereas individual treatment focuses on the remediation of specific deficits and/or the restoration of specific language forms. Today the rationale for group therapy is often its cost-effectiveness. Some patients dislike group therapy and refuse to participate, and others prefer it. Interest in the potential of group therapy to enhance improvement in chronic aphasia has increased in recent decades (Aten, 1981; Aten et al., 1982; Bollinger, Musson, & Holland, 1993; Fawcus, 1991; Kearns, 1986; Wertz et al., 1981).

Kearns (1986) identified five types of group treatment: direct (structured, didactic, directed by clinician), indirect (unstructured, open-ended in content and activity), sociolinguistic (focus on interactive communication among participants, clinician does not direct), transition (reinforcement of skills obtained in individual treatment through group interaction, and maintenance (social stimulation, focus on leisure activities). Aten et al. (1982) showed significant gains in functional communication, as measured by the Communicative Abilities of Daily Living (CADL) but not on other measures, in chronic aphasia resulting from group treatment; Wertz et al. (1981) showed gains with group therapy in a 44-week period of intervention; and Bollinger et al. (1993) provided two 20-week intervals of group treatment alternating with two 10-week periods of no treatment to 14 chronic subjects and reported gains in overall communication ability. Bollinger noted that "careful structuring of group communication intervention enables a strengthening of the nonlanguage and language cognitive processes that underlie meaningful communication" (p.312).

The results of the aforementioned group therapy research highlights many of the problems associated with mounting group therapy programs. It is not simply a matter of gathering aphasic individuals together at an appointed time; it requires careful consideration of the program goals, criteria for patient selection, method for assessing efficacy, and termination criteria. Furthermore, group therapy requires an experienced clinician who is knowledgeable about and sensitive to the characteristics of the recovery stages and who has expertise in group dynamics, counseling, adaptation, and quality-of-life assessment. Effective group therapy cannot be implemented by inexperienced clinicians for it can require more creative design and maturity than individual treatment which deals with the problems of only a single individual with guidelines that have been established over a long period. The approaches introduced by Kagan (1995a), Kagan and Gailey (1993), N. Simmons-Mackie (1993), and N. N. Simmons-Mackie and Damico (1995, 1997) provide guidelines for group therapy design.

Studies of the Efficacy of Aphasia Treatment

Despite the problems associated with designing and implementing efficacy studies, the topic has engaged the interest of many investigators. The difficulties include subject selection criteria, no-treatment control groups, random assignment to treatment conditions, single-subject versus group study design, frequency, intensity and duration of treatment, specification of treatment, the testing schedule, and the appropriateness, reliability, and validity of the measures used. The heterogeneity of groups of individuals with aphasia has been one of the major criticism of studies based on group data. On the other hand, objections to single-case studies include the possibility of idiosyncratic performance, the difficulty of replication, and the fact that no single study can provide definitive data which could support the effectiveness of aphasia therapy (Avent, Edwards, Franco, Lucero, & Pekowsky, 1995; Basso, 1992; Damasio, 1992; Ingham, 1990; K. J. Kearns, 1990; Olswang, Thompson, Warren, & Minghetti, 1990; Prins, Schoonen, & Vermeulen, 1989; Wertz et al., 1981, 1986).

A meta-analysis of efficacy studies published in the period 1946 to 1988, comprising 45 studies, highlighted many gaps and open questions (Whurr, Lorch, & Nye, 1992). Robey's meta-analysis (1998) includes the clinical outcomes of 55 reports which showed a clear superiority in performance of those who received treatment by a speech–language pathologist (Robey, 1994, 1998). In addition, his findings confirmed that the more intensive the treatment the greater the change; outcome is greatest when treatment is begun in the acute stage of recovery; treatment for the moderately severe and severe can have a large impact when it is begun in the chronic stage; and treatment in excess of 2 hr per week yields greater gains than less intensive treatment. Robey's meta-analysis challenges third-party payor policies that deny reimbursement for later stages of recovery (Robey, 1998). Investigators have addressed the efficacy question using a wide array of methods, making it difficult, if not impossible, to compare study results. A discussion of the issues pertaining to the study of aphasia intervention efficacy can be found in R. David (1991) and Holland, Fromm, DeRuyter, and Stein (1996).

Treatment efficacy studies include those that address the effectiveness of aphasia intervention, efficiency of specific treatments, and the effects of treatment (Olswang et al., 1990). Data may be derived from experimental studies, program evaluation measures, and single case studies. However, the lack of consistency in both the design and methodology of efficacy studies makes it difficult to compare results. Efficacy studies that do not include a functional communication measure provide an incomplete picture

of outcome because they do not include information that addresses the primary objective of aphasia therapy: improvement of the quality of life (David, 1991, in Code & Muller, 1991). A list of the primary efficacy studies is shown in Table 18.1.

Summary

The foregoing material has briefly reviewed the aphasia rehabilitation experience and relevant literature, especially since the second world war. The approaches used have usually been based on theoretical concepts of the nature of aphasia and its recovery. This has been inevitable, as the definitive neurophysiology of communication has not yet been described. Historically, clinicians have adopted those strategies and systems which they believe are the most effective and best suited to the individual patient.

It is probably appropriate that there have been few studies comparing treatment methods in view of the seemingly insurmountable methodological problems associated with such research and our present state of knowledge. Although we cannot be absolutely certain of how to approach the

TABLE 18.1
A Selected List of Efficacy Studies

Marks et al. (1957)
Vignolo (1964)
Hagen (1973)
M. T. Sarno et al. (1970)
Basso et al. (1975)
Butfield and Zangwill (1975)
Kertesz and McCabe (1977)
Deal and Deal (1978)
Basso et al. (1979)
Holland (1980)
Wertz et al. (1981)
R. David, Enderby, and Bainton (1982)
Lincoln et al. (1984)
C. M. Shewan and Kertesz (1984)
Wertz et al. (1986)
Hartman and Landau (1987)
Poeck et al. (1989)
Levita (1978)
Prins et al. (1989)
R. C. Marshall, Wertz, Weiss, and Aten (1989)
Mazzoni et al. (1995)

problem therapeutically, there are large numbers of patients who need treatment and require management. The following section presents a philosophy of aphasia rehabilitation and related ethical questions and dilemmas.

A richness and diversity of approaches to aphasia have built up over decades of study and clinical work. However, there is as yet no defined philosophy of aphasia intervention. Aphasiology embraces a broad scope of concerns ranging from the neurolinguistic and cognitive to the pragmatic, functional, emotional, and social. There has been a tendency to view the schools of intervention as reflecting a crisis of theory and practice. But human behavior as complex and all-encompassing as communication may benefit from a diversity of approaches by avoiding a reductionist model and perpetually seeking a coherent philosophy of treatment (Petheram & Parr, 1996).

A Philosophy of Aphasia Rehabilitation

A critical reality in aphasia rehabilitation is the fact that once the condition is stabilized very few patients recover normal communication function, with or without speech therapy. This means that aphasia rehabilitation must be viewed as a process of patient management in the broadest sense. The task becomes one of helping the patient and his or her intimates facilitate communication, enhance social interaction, develop strategies that facilitate these interactions, and adjust to the alterations and limitations imposed by the disability. This poses a complex challenge to the aphasia therapist, who must consider all of the factors that contribute to the individual's life experience, value systems, expectations, and needs for fulfillment. The interaction of the type and severity of aphasia, physical and neuropsychological deficits, and emotional state with levels of activation and initiation, social isolation, physical condition and/or disability, and degree of available emotional support also need to be considered (Benson, 1979b; Darley, 1970, 1972, 1975; Eisenson, 1973; Reinvang, 1980; M. T. Sarno, 1980, 1993, 1997; Schuell et al., 1964; Wepman, 1951). Furthermore, in our ethnically diverse society cultural factors must be considered (see Chapter 16). Management issues can be further complicated in posttraumatic aphasia because these patients often also present a greater number of behavioral, cognitive, and perceptual deficits. Their generally younger age poses social and adjustment problems that are different from the middle-aged and older population (M. T. Sarno, 1980b; M. T. Sarno, Buonaguro, & Levita, 1986, 1987).

The multiplicity of factors involved in rehabilitation suggests that the

ideal context for effective intervention requires the participation of several disciplines, including medicine, psychology, physical and occupational therapy, social work, vocational counseling, and, most critically, aphasia therapy. The greatest responsibility in the intervention process usually falls to the aphasia therapist. The selective and discriminating use of speech therapy to stimulate and support the patient through the various stages of recovery can be an effective management tool (Brumfitt & Clarke, 1980; M. T. Sarno, 1980; Tanner, 1980).

The Aphasia Therapist

In view of the current restraints on treatment for aphasia, it is not possible to carry out programs appropriate to its devastating and disabling effects. The therapist is challenged to provide the most meaningful intervention possible with full awareness that the contact will be limited in terms of intensity and duration. Indeed, this may be the only contact the individual will have with a professional who is expert in the management of his or her newly acquired communication disorder. Ideally, the aphasia therapist and patient would embark on a therapeutic partnership not limited by time constraints. The relationship would extend for the many months required to restore sufficient functional communication, compensatory strategies, and self-confidence to allow the person to come to terms with the realities of the impairment and to reach a level of adaptation and interactive skill that fosters community reintegration and some degree of life fulfillment.

It is clear to the experienced clinician that no single technique is adequate to produce normal communication function, and the ideal overall approach, given our present stage of knowledge, remains eclectic and specifically tailored to the individual patient. Fundamental to this therapeutic philosophy is the acknowledgment and appreciation of the uniqueness of the individual. This is not simply to pay lip service to the humanitarian idea that we are all unique, but to underline that no two aphasic persons are exactly alike in pathology, personality, linguistic deficits, reactions to catastrophic illness, life experience, spiritual values, and a host of other factors; that the influence of these factors carries different weight and strength at different stages of recovery; and that they are all inextricably related to recovery outcome.

There are a number of principles that underlie practice. An important one, stated by Brumfitt and Clarke (1980), is that speech therapy is a "special case of the general art of psychotherapy." Basic to each therapeutic technique is the concept that it must contribute to the patient's psycholog-

ical comfort and, conversely, should never feed into depression, frustration, anger, feelings of low self-esteem, or other negative emotions. This is uppermost in the mind of the experienced, enlightened therapist and dictates the overall management plan. The therapist chooses those techniques or exercises that allow the patient to use preserved skills, thereby increasing the chances for successful performance. The patient needs to be shown that he or she is able to perform and can adapt to such a catastrophic event. Aphasia therapists must become the patient's advocate while learning in detail what the patient can and cannot do and what strategies are the most effective in the facilitation of communication.

Kindness and good intentions are insufficient for effective aphasia therapy. The competent therapist must have a thorough knowledge of aphasia to be able to discuss the symptoms, reactions, and behavior associated with aphasia with the professional staff, patient, and family. These explanations often require endless repetition before they can be comprehended and incorporated.

The subject of motivation always arises in the treatment of disabling neurological disorders. Experience suggests that, except for severely depressed individuals who are unable to put forth much effort, patients generally produce everything of which they are capable. Therapists must not allow their expectations to contaminate the therapeutic interaction; this is not uncommon and is usually motivated by laudatory aspirations—therapists want to see their patients improve—and this can be counterproductive. Patients with involvement of subcortical areas may have low levels of activation, a purely physiological process independent of psychological motivation. The distinction between these two processes must be understood.

A clinical observation with particular relevance to aphasia therapy is the fact that anxiety has a negative effect on performance. The therapeutic setting should be undistracting, quiet, and comfortable in order to elicit a patient's best performance. Families sometimes report that the patient performs better at home than in therapeutic sessions, which is probably related to the familiarity of the home setting.

With the continuing reduction of access to rehabilitation services, there has been an effort to involve the family in the therapeutic process by providing educational programs for family members and training them to carry out a structured home program with the patient (Burns, Dong, & Oehring, 1995; Hinckley, Packard, & Bardach, 1995). This is an added responsibility for the aphasia therapist. With the current shift of therapeutic responsibility to the family, this area will undoubtedly receive greater attention and development in the near future.

Experienced aphasia therapists evoke much respect and admiration for

they engage in work that is always difficult and frequently underestimated considering the skill, wisdom, patience, and understanding that are required. Through knowledge of aphasic deficits, specific treatment techniques, and the needs of the patient, they make ever-changing judgments and decisions about patients' requirements from moment to moment, often balancing patients' needs for nurturance with the reality of the limitations imposed by the aphasia and the drastic time constraints mandated by the health care system.

Many ethical and moral dilemmas face those who manage the rehabilitation of aphasic patients. The traditional ethical issues of patient selection, autonomy, termination of treatment, and beneficence seem less relevant in today's managed care system than than they were a decade ago (A. Caplan & Haas, 1987; M. T. Sarno, 1986). Third-party payor demands for cost effectiveness and the short exposure to professional help raise new dilemmas for the clinician revolving around how best to spend the limited time, how quickly the patient and family can integrate information about the condition and how they might proceed to assist in the enhancement of communicative interactions.

The current constraints on the delivery of health services, especially for chronic conditions, will undoubtedly force an increase in group therapy for individuals with aphasia to replace the high costs of individual treatment. In anticipation of this, aphasia clinicians need to explore ways for using group therapy as one of several methods to provide aphasia rehabilitation services for a longer period of time postonset. This might include the development of peer groups which meet regularly, of Aphasia Community Groups (ACG), perhaps under the leadership of a family member, or of a peer telephone contact system. Aphasia Community Groups, which comprise the national support network of the National Aphasia Association (NAA), stroke clubs, and other social groups are resources which can be effective tools in clinical management. They generally serve best after the acute spontaneous recovery period, when patients are more aware of their deficits and desirous of meeting peers and accessing opportunities for social interaction. By this time the patient may be more interested in and capable of interacting with others and gain support by sharing feelings with those who share the experience.

Concluding Comments

Historical realities suggest that the realm of recovery and rehabilitation in aphasia is beginning to establish a firm basis in objective data. Until now a great deal of what is known and practiced depends on clinical experience

and the slow development of maturity and skill in those who undertake this difficult work. Systematic investigation to date has not sufficiently clarified the physiology and pathophysiology of communication or provided therapeutic blueprints for disorders such as aphasia. To a large extent, successful treatment is compensatory in nature and the effective therapist is one who has learned from experience how to maximize the process of adjustment. The projected growth of the population at risk for acquiring aphasia and the realities of the contemporary health care system represent a major challenge to aphasia rehabilitation.

In spite of the reality of the great challenge posed by aphasia, those who engage in this work have not been deterred. New knowledge and research have opened promising areas for exploring and developing more sophisticated intervention techniques. Contemporary studies on language processing, new applications of microcomputer technology, the development of alternative communication systems, and research in the management of the psychosocial sequelae of aphasia depression offer hope for improving treatment management. The study of human brain mechanisms is one of the last frontiers of biology and as such will continue to be an irresistible challenge.

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