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

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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-

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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-

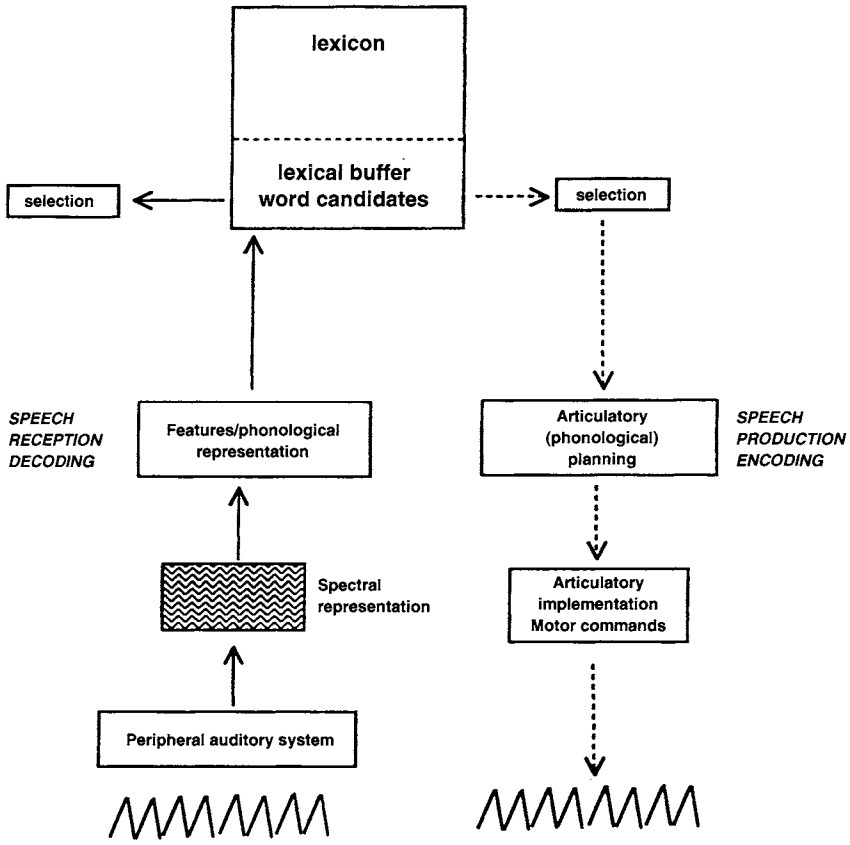

FIGURE 5.1. *A working model of speech production and speech perception. From Blumstein (1994).*

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.

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