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Acquired Aphasia in Children

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Introduction

Children versus Adults

Language disruptions secondary to acquired central nervous system (CNS) lesions differ between children and adults in multiple respects. Chief among these differences are the developmental stage of language acquisition at the time of insult and the developmental stage of the CNS.

In adult aphasia premorbid mastery of language is assumed, at least to the level of the aphasic’s intellectual ability and educational opportunities. Acquired aphasia sustained in childhood, however, interferes with the developmental process of language learning and disrupts those aspects of language already mastered. The investigator and clinician thus are faced with sorting which aspects of language have been lost or impaired from those yet to emerge, potentially in an altered manner. Complicating research and clinical practice in this area is the need to account continually for the developmental stage of that aspect of language under consideration for each child. In research, stage-appropriate language tasks must be selected, and comparison must be made to peers of comparable age and language stage. Also, appropriate controls common in adult studies, such as social class and gender, are critical. These requirements present no small challenge, as most studies involve a wide age range of children and adolescents. In clinical practice, the question is whether assessment tools used for developmental language disorders should be used or whether adult aphasia batteries should be adapted for children. The answer typically depends on the age of the child and the availability of age- and stage-appropri-
priate measures. With childhood acquired aphasias, the question of how language is altered following a CNS insult is inextricably related to how language learning proceeds.

The second major difference between adults and children sustaining language-disrupting neurological insults is the stage of maturity of the CNS at the time of insult. The controversy relating to the degree of early hemispheric specialization versus equipotentiality for language (e.g., Kinsbourne & Hiscock, 1977; Lenneberg, 1967) has sparked much of the research in this area during the past 20 years. Although most of the data appear to evidence considerable early brain specialization for language and other higher cognitive functions (Best, 1988; Molfese & Segalowitz, 1988), the remarkable capacity of young children to recover from major cortical insults has been repeatedly reported. That children recover much more rapidly and completely from focal brain lesions than do adults with comparable insults has become a truism that is generally, although not universally, supported. The rapidity and level of language recovery in young brain-injured children evidence a degree of functional and presumed neural plasticity far exceeding that in adults (Aram & Eisele, 1992). Most (Bates et al., in press; Eisele & Aram, 1993; Lees, 1993; Vargha-Khadem, Isaacs, van der Werf, Robb, & Wilson, 1992) but not all (Martins & Ferro, 1993; Paquier & Van Dongen, 1993) investigators in this field conclude that the pattern of language loss following lesions in children generally do not correspond to what is observed in adults with similar injuries. Rather, as Bates et al. (1997) recently have suggested, the data thus far suggest that “innate regional biases in style of information processing lead to familiar patterns of brain organization for language under normal conditions and permit alternative patterns to emerge in children with focal brain injury” (abstract). An immature CNS at the time of insult does not necessarily lead, however, to a more favorable outcome. In some instances—for example, conditions resulting in diffuse brain involvement—a brain insult incurred at a young age may be more, rather than less, deleterious, a topic returned to later in this chapter. The essential concern here is that just as the child’s language system is in the process of development, so too is the central nervous system, processes that are complete or declining in the adult. Although in many instances the immaturity of the child’s CNS supports alternatives for greater functional recovery, at other times early insults appear to interfere with primary skills, thus precluding later achievements.

Terminology

ACQUIRED VERSUS DEVELOPMENTAL APHASIA

The term acquired is used to modify the term aphasia in children to distinguish language disorders accompanied by known CNS insults from the
much more common form of developmental aphasia. Developmental aphasia, also referred to as developmental language disorders or specific language impairment, is manifest when a child fails to learn to talk normally, but a frank neurological basis is not apparent. Indeed, the presence of a frank neurological abnormality typically is established as an exclusionary criterion for the diagnosis of developmental aphasia or developmental language disorder (Benton, 1964; Tallal, 1988). Although many researchers assume abnormal neurological functions give rise to the developmental aphasias (e.g., Rapin & Allen, 1988), the search for identifiable brain lesions to account for these developmental disorders has been largely unproductive. Also, the language symptomatology that follows acquired unilateral lesions typically has been found to be less severe and less persistent than is often seen in children with developmental language disorders (Aram & Eisele, 1994). This chapter addresses the language abilities only in children with known brain insults, that is, those with acquired aphasia.

ACQUIRED LANGUAGE LOSS VERSUS ACQUIRED BRAIN LESION

According to the literature on acquired aphasia in children, even though the lesion is generally acquired—through stroke, tumor, trauma, or some other form of CNS insult—the language loss is not necessarily "acquired," as frequently the insult occurs before much language has been acquired and therefore not much can be "lost." Few studies have been confined to older children with relatively well developed language at the time of insult; rather most have involved a wide age range at lesion onset, typically extending from pre- or perinatal insults to those incurred during adolescence. Indeed, many studies compare outcome as a function of whether the lesion was sustained prior to 1 year of age (the somewhat arbitrary demarcation for the onset of language) or after 1 year of age, with a broad range of onset ages included in the latter group. Therefore, acquired aphasia, as used here, refers to language discrepancies or abnormalities accompanied by a known brain lesion, irrespective of when during the course of language development that lesion occurred.

Nature of the Studies Available

Although a more homogeneous age of lesion onset would be desirable in reports of acquired aphasia in children, age is only one of several markedly heterogeneous variables complicating interpretation of the majority of studies addressing this topic. The major methodological problems confounding review of work in this area have been reviewed elsewhere (Aram & Whitaker, 1988). Five variables, typically noncomparable both within and across studies, are of particular note:
1. **AGE OF LESION ONSET** was discussed earlier and generally involves a broad spectrum of ages.

2. The **NATURE OF THE NEUROLOGICAL INSULTS** described in studies are typically diverse, and often include such disparate conditions as tumors, head trauma, herpes encephalitis, and cerebral vascular accidents in a single study. This chapter draws not only from studies specifically addressing acquired CNS insults and aphasias in children, but also from studies of infantile or childhood hemiplegias, as the overlap of children included in these studies is considerable. In an effort to reduce the wide diversity of conditions discussed here, this chapter does not address the hemispherectomy or epileptic aphasia literature except when an occasional hemispherectomized or epileptic aphasic child has been included in a group study involving heterogeneous causes.

3. **EXTENSIVENESS OF BRAIN INVOLVEMENT** also varies widely from circumscribed focal lesions to diffuse white and gray matter involvement. Often specification of the degree of actual brain involvement is lacking and can only be inferred from the etiology or clinical pattern. This topic is discussed more fully later in this chapter.

4. **AGE AT FOLLOW-UP** varies both with respect to chronological age and with respect to time elapsed since lesion onset. A few studies have described language during the acute period of recovery, although most have assessed language status years after lesion onset.

5. The **METHOD OF EVALUATION** used in various studies has differed. Until the past 15 years, most statements pertaining to language ability were based on nonsystematic clinical observations or on verbal intelligence scores. More recently, most studies have begun to report standardized language tests, and a few have begun to include a more experimental or hypothesis-testing approach to the study of acquired aphasia in children.

Because very few experimentally rich or methodologically sound studies of relatively homogeneous groups of lesioned children exist, the present review is not restricted to those few studies. Rather, an attempt has been made to cull from the diverse studies available and to interpret contradictory findings in light of the differences among subjects and methodologies.

**The Clinical Picture:**

**Language Characteristics**

**Comprehension**

Despite pronouncements that receptive disorders are rare or that beyond the acute period comprehension disorders disappear rapidly and vir-
tually completely (Hécaen, 1976, 1983), until recently few studies provid-
ed objective data substantiating or refuting these claims. Guttmann (1942) appears to be one of the few early observers noting long-standing recep-
tive as well as expressive deficits following temporal lobe lesions in chil-
dren. Alajouanine and Lhermitte (1965), although commenting that recep-
tive disorders were rare, reported that 4 of 32 children with acquired aphasia presented marked comprehension disorders. Several case studies have detailed the recovery of comprehension abilities during the acute pe-
riod, usually (Aram, Rose, Rekate, & Whitaker, 1983; Ferro, Martins, Pin-
to, & Castro-Caldas, 1982; Martins, Ferro, & Trindade, 1987; Pohl, 1979), but not always (Dennis, 1980; Oelschlaeger & Scarborough, 1976), demonstrat-
ing complete or relatively good recovery of comprehension skills. Ex-
cept for the exemplary work of Dennis and her colleagues with young hemispherectomy patients, prior to the past 15 years there appear to be no studies other than IQ results in which findings are detailed relative to com-
prehension abilities among children with acquired aphasia. The studies available have focused predominantly on syntactic and lexical compre-
hension.

SYNTACTIC COMPREHENSION

Dennis (1980) provided a comprehensive study of the acute language status of a 9-year-old girl with a left temporoparietal infarct at 2 weeks and at 3 months after lesion onset. Drawing from an array of standardized and experimental tasks, Dennis (1980) concluded that, although improvement had been observed, at 3 months after lesion onset the child’s compre-
ッション of longer, nonredundant oral commands continued to be impaired, and lower level syntactic structures were better preserved than were more complex structures involving supraordinate schemata such as embed-
dings. On a metalinguistic judgment task in which the interrelatedness of words was assessed, the child seemed to adopt a simplified surface and linear processing strategy for complex utterances. Although still relative-
ly early in recovery, this case study demonstrated significant disruption in all aspects of language, including comprehension, expression, and com-
municative intent; unfortunately, language status after 3 months was not reported. Other case studies have reported notable syntactic comprehen-
sion deficits acutely but with relatively good recovery within the first sev-
eral months after lesion onset (Aram et al., 1983; Ferro et al., 1982; Pohl, 1979).

Several group studies of children with brain lesions, studied well be-
yond the acute period, have assessed syntactic comprehension and gener-
ally found subtle yet persistent comprehension deficits. Findings between studies vary somewhat, presumably reflecting differences in subject vari-
ables, notably the nature and diffuseness of the lesion, concomitant seizure
disorders, and overall intellectual level. Levine, Huttenlocher, Banich, and Duda (1987) found that well after lesion onset, all four groups of children with left, right, congenital, and acquired hemiplegias performed below average on the Northwestern Syntax Screening Test (Lee, 1969); it should be noted, however, that these children's intelligence was below average and half had ongoing seizure disorders. In contrast, Kiessling, Denckla, and Carlton (1983), studying groups of left- or right-hemiplegic children selected because they were functioning well in school, found a significant correlation between right-hand function on the Annett pegboard (used as a measure of left-hemisphere function) and performance on a syntactic awareness task, thus evidencing poorer performance following left-hemisphere injury. However, in a more recent study of comprehension and imitation of complex coordinate, passive, and relative clause structures examined in a group of left-lesioned children and a group of right-lesioned children in comparison to normal controls, left-lesioned children's comprehension was relatively preserved despite significantly impaired imitation (Eisele & Aram, 1994). Right-lesioned subjects' impairment was less pronounced in both comprehension and imitation compared with the left-lesioned group.

Token Test (de Renzi & Vignolo, 1962) results have been reported by several groups of investigators, with findings that apparently reflect the variable subject groups under test. Woods and Carey (1979) reported that left-lesioned subjects differed from controls on the Token Test if lesion onset occurred after but not before 1 year of age. Vargha-Khadem, O'Gorman, and Watters (1985) reported that irrespective of age of onset, their three left-lesioned subject groups (prenatal, early postnatal, and late postnatal) but not the three respective right-lesioned groups performed more poorly than control subjects. Similarly, Aram and Ekelman (1987) found performance of left- but not right-lesioned subjects to be significantly lower than that of controls, and identified no relationship between age of lesion onset and revised Token Test (McNeil & Prescott, 1978) performance among lesioned subjects. Variable performance depending on the Token Test subtest has been noted. Left-hemisphere-lesioned children have been reported to have particular difficulty with subtests that assess syntactic components (Aram & Ekelman, 1987; Cooper & Flowers, 1987; Riva, Cazzaniga, Pantaleoni, Milani, & Fedrizzi, 1986) or that tax verbal memory (Aram & Ekelman, 1987; Rankin, Aram, & Horwitz, 1981), whereas right-hemisphere-lesioned subjects have been reported to do more poorly on items requiring spatial skills (Aram & Ekelman, 1987; Riva et al., 1986). Van Dongen and Loonen (1977), studying a group of acquired aphasic children with mixed etiologies, reported that comprehension deficits on the Token Test during the acute stage were associated with a poor prognosis for recovery.
In summary, the data suggest that syntactic comprehension often is impaired following left brain involvement. Poor performance by right-hemisphere-lesioned subjects appears to be related to spatial demands of the task and/or to more generalized brain involvement. Beyond the acute period, most but not all syntactic comprehension deficits are mild. Poor syntactic comprehension in the acute period appears to be related to a poor prognosis for recovery. Age of lesion onset has not been found to have a consistent relationship to syntactic comprehension.

LEXICAL COMPREHENSION

Until recently, except for a few detailed case studies, lexical comprehension skills among children with acquired aphasia have been studied little beyond the administration of the Peabody Picture Vocabulary Test (PPVT, Dunn, 1965). In one case study of a 10-year-old girl who sustained diffuse traumatic brain involvement after a fall from a horse, Oelschlaeger and Scarborough (1976) documented significant limitations in lexical comprehension 1 year after the trauma was sustained. Most group reports do not indicate deficits as pronounced as that observed by Oelschlaeger and Scarborough (1976), but do evidence mild long-standing deficits in lexical comprehension. Using the PPVT, Aram, Ekelman, Rose, and Whitaker (1985), Cooper and Flowers (1987), Levine et al. (1987), and Riva et al. (1986) all reported lower performance by the lesioned than the control subjects. Although Kiessling et al. (1983) reported a correlation between right-hand function and PPVT performance, most studies (Aram et al., 1985; Levine et al., 1987; Riva et al., 1986) have failed to find a lateralized left-hemisphere effect on PPVT performance. In fact, Eisele and Aram (1993) found that although both right- and left-lesioned subjects scored more poorly than controls on the PPVT-R, the right-lesioned children presented the greatest impairments, leading these investigators to suggest that the right hemisphere may have a specialized role in mediating the acquisition of word meaning. Similarly, Bates and her colleagues (1997), using a parent informant inventory, the MacArthur Communicative Development Inventories (Fenson et al., 1993), also found children with right lesions between 10 and 17 months of age to present greater delays in word comprehension than children with left-hemisphere lesions, contradicting what would be hypothesized from the adult literature.

Finally, one of the few studies (Cooper & Flowers, 1987) to examine lexical comprehension beyond single-word representation assessed meaning in context with the Processing Spoken Paragraphs subtest of the Clinical Evaluation of Language Functions (Semel-Mintz & Wiig, 1982). On this test, significant deficits among the group of chronic brain-injured children were reported. However, because of the diffuse nature of brain involve-
ment for most of the subjects, no attempt was made to relate findings to laterality of brain involvement.

In summary, based on studies predominantly assessing single-word comprehension, at least mild lexical deficits have been noted in most groups of children with acquired aphasia. Lexical comprehension deficits generally have been found in children with either left or right hemisphere damage, and recent studies suggest greater impairment in the latter group of children.

Language Production

Language production is the most extensively studied aspect of acquired childhood aphasia. Although descriptions tend to center on reduced verbal output, descriptions of more fluent-type aphasia and paraphasic behaviors have been reported. Some limited work has also addressed lexical retrieval and phonological production.

REduced Verbal Production

Most early reports of acquired aphasia focused on the diminished speech output and telegraphic speech thought to characterize acquired aphasia in children (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Early descriptions emphasized the striking feature of reduced verbal output, ranging from mutism to a reluctance to speak, and stated that syntax was simplified rather than erroneous (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Equally impressive to many early observers was the rapid recovery of expressive abilities, the absence of fluent Wernicke-type aphasias, and the infrequency of paraphasias (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976, 1983). Although fluent aphasias with neologistic, semantic, and phonemic paraphasias have since been described (see the next section), reduced output continues to be regarded as the dominant feature of acquired aphasia in children (Cranberg, Filley, Hart, & Alexander, 1987; Martins & Ferro, 1987, 1993).

Woods and Carey (1979) provided what appears to be the first experimental study of productive syntax in left-hemisphere-lesioned patients. By using a series of syntactic production tasks, including identifying and correcting anomalous sentences with "that" clauses, ask–tell distinctions, and the sentence completion task from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983), they found that left-lesioned childhood aphasics, sustaining lesions after but not before 1 year of age, differed significantly from control subjects.

Most subsequent studies of children with acquired lesions have found
syntactic limitations to be associated predominantly with left but not right lesions. For example, both Kiessling et al. (1983), in a study of hemiplegic children, and Riva et al. (1986), in a study of acquired lesions of diverse etiologies, found left-hemisphere-lesioned children to perform more poorly than right-lesioned or control subjects on sentence repetition tasks. Similarly, Carlsson et al. (1994) found right-hemiplegic (left hemispheric involvement) children to be more impaired than left-hemiplegic children on several verbal tasks. Also, in an analysis of the spontaneous conversation-al language of left- and right-lesioned children studied well beyond the acute period, Aram, Ekelman, and Whitaker (1986) found left- but not right-lesioned children to perform less well on a range of measures of simple and complex sentence structure. In a more recent study assessing imitation of complex coordinate, passive, and relative clause structures, Eisele and Aram (1994) again found the residual deficits in syntactic ability to be more pronounced as a result of early left in comparison to right brain lesions. This study also compared imitative abilities to comprehension of the same structures, demonstrating a marked dissociation of comprehension and production of syntax among the left-lesioned children.

Significant deficits in syntactic production rarely are reported following right lesions except in cases thought to represent crossed aphasia (Martins et al., 1987). An exception has been a study that used a speech shadowing task (Woods, 1987) in which sentences were to be repeated in correct and reversed word order. On this task both left- and right-lesioned subjects performed more poorly than controls, despite the right-lesioned subjects' normal performance on other language measures. Woods (1987) explained his findings by suggesting that the shadowing task tapped skills dependent on global cerebral function, rather than on more narrowly defined language functions.

Thus it appears that reduced output and simplified syntax are the typical presentation following left-hemisphere lesions. In addition, syntactic deficits may persist for years.

FLUENT VERBAL PRODUCTION

Many of the early descriptions of acquired aphasia in children stated that fluent aphasias did not occur or were extremely rare in children younger than 10 years of age (Alajouanine & Lhermitte, 1965; Guttmann, 1942; Hécaen, 1976). The absence of fluent aphasia in young children was ascribed to an underdeveloped or underautomatized Broca’s area controlling expressive language which was incapable of “running on” in the absence of appropriate input from posterior language areas. More recently, however, several examples of fluent aphasias with jargon or logorrhea have been described in young children. Woods and Teuber (1978) appear
to be one of the first to have described jargon aphasia, defined as jabbering away with unintelligible sounds as though understood, in a single 5-year-old among 65 children studied. Additional case studies of fluent aphasias have been reported by Martins and Ferro (1993), Van Hout and Lyon (1986), and Van Dongen, Loonen, and Van Dongen (1985). These studies detailed young children with logorrheic speech, a high proportion of neologisms, and significant comprehension disorders. Van Dongen et al.'s (1985) three cases also presented a phrase length of at least seven words, a speaking rate of more than 90 words per minute, and normal prosody, articulation, pauses, and effort. These patients also exhibited frequent paraphasias.

PARAPHASIAS

Just as fluent aphasia was thought to be rare or absent among childhood acquired aphasics, paraphasias also were believed to be exceptional (Alajouanine & Lhermitte, 1965; Hécaen, 1976). Their occurrence, however, has been described among children with reduced verbal productions, particularly among the few with more fluent verbal production. Paraphasias appear to be more common during the acute period following acquired brain lesions, and have been described as especially notable and persistent among children with diffuse brain involvement as opposed to more focal or lateralized brain lesions. Visch-Brink and Sandt-Koenderman (1984) and Van Dongen and Visch-Brink (1988) have provided detailed descriptions of neologisms, literal paraphasias, and verbal paraphasias in spontaneous speech and naming tasks in first two and then six children who were 5 years and older at the age of insult. In addition, they presented a single case of phonemic jargon aphasia. These case studies exemplify the early appearance of neologisms and paraphasias after lesion onset and their rapid disappearance, usually within 2 or 3 weeks of lesion onset, at least among head-injured patients.

Van Hout, Evrard, and Lyon (1985) also summarized the verbal and paraphasic errors among 11 children with acquired aphasia. They divided their patients into three groups according to the evolution of their paraphasias, which also coincided with the severity of associated problems. For Groups I and II, the paraphasias resolved in a matter of days and over a few months, respectively; for Group III, the paraphasias persisted for more than a year. Particularly notable among Van Hout's patients was the severity of associated problems in all but Group I and the diffuse nature of brain involvement in all patients. Three of the four patients in Group III, the most severely impaired group, had herpes encephalitis, and the fourth incurred cerebral trauma followed by a Stage III coma for 1 month with decerebrate posturing. All had significant associated problems and appeared to be
Because of the above-mentioned factors, children with acquired aphasia may be grossly demented. Although paraphasic errors may occur in the acute period following acquired focal lesions in young children with either reduced or fluent verbal outputs, in general, their persistence appears to be associated with more pervasive cognitive disorders as a consequence of diffuse brain involvement.

NAMING AND LEXICAL RETRIEVAL

Although a few studies (Van Dongen & Visch-Brink, 1988) have described paraphasic errors on naming tasks, the majority of studies addressing naming and lexical retrieval have simply recorded the correctness of response. Most have reported reduced naming abilities among children with left- but not right-brain lesions. For example, Hécaen (1976, 1983) reported that 44% of left-lesioned but none of the right-lesioned children had naming disorders that he described as being impoverished. Similarly, Van Dongen and Visch-Brink (1988) reported no naming problems in spontaneous speech or on naming tasks among right-lesioned children. Among those with left lesions, they differentiated between children with head injury who demonstrated a successive decrease in neologisms and recovery of all naming errors by 6 months of age, and non-head-injured children (cerebrovascular accident, subdural empyema, encephalitis) who presented more severe and persistent aphasic symptoms with irregular distribution of types of naming errors during recovery. Yet Eisele and Aram (1993) reported that left-lesioned children performed comparably to controls on the Expressive One-Word Picture Vocabulary Test (Gardner, 1979). Riva et al. (1986) and Thal et al. (1991) found deficits among right- as well as left-lesioned subjects, although both Thal et al. (1991) and Bates et al. (1997) found deficits to be the greatest after insult to the left posterior hemisphere.

Aram, Ekelman, and Whitaker (1987) administered the Word Finding Test (Wiegel-Crump & Dennis, 1984) and the Rapid Automatized Naming Test (RAN; Denckla & Rudel, 1976) to left-lesioned, right-lesioned, and control subjects. On the Word Finding Test, left-lesioned subjects were slower than other subjects in latency of response when given semantic or visual cues, and they made more errors when given rhyming cues. On the RAN, left-lesioned subjects were significantly slower than controls in naming all semantic categories (colors, numbers, objects, and letters). In contrast, right-lesioned subjects responded as quickly as or more quickly than control subjects in all access and semantic category conditions, yet produced more errors than controls, suggesting a speed-accuracy trade-off. The overwhelming error type was "no response," and few error types among the lesioned subjects differed appreciably from those of controls. Lesioned subjects, however, were assessed at least 1 year and often sever-
al years after lesion onset and thus the assessments did not capture any naming errors that may have occurred acutely.

Age of lesion onset has been equivocally related to naming deficits among left-lesioned subjects. Woods and Carey (1979) found left-lesioned subjects who sustained lesions after but not before 1 year of age to be impaired on naming tasks. However, Vargha-Khadem et al. (1985) found left-lesioned subjects, irrespective of the age when the lesion was sustained (congenital, early, or late acquired), to perform more poorly than right-lesioned subjects on the Oldfield–Wingfield Object Naming Task (Oldfield & Wingfield, 1964). Similarly, Aram et al. (1987) found no relationship between age of lesion onset and lexical retrieval abilities among either left- or right-lesioned children.

In summary, it appears that naming and lexical retrieval deficits are common after left, but also may be observed after right hemisphere lesions and for the most part they do not appear to be related to age of lesion onset. Paraphasic errors have been described especially in the acute period after lesion onset. Although paraphasic errors may persist for some aphasic children, they do not appear to be a common feature later in recovery.

**PHONOLOGICAL AND ARTICULATORY PRODUCTION**

Aside from the few case reports of phonemic paraphasias (Van Hout et al., 1985; Visch-Brink & Sandt-Koenderman, 1984), very little detail has been offered relative to phonological production or articulatory abilities among children with acquired aphasia. No consensus appears regarding the occurrence of phonological and articulatory problems. Early studies suggested that articulatory disturbances were common. Alajouanine and Lhermitte (1965) stated that if the lesion occurred before 10 years of age, disorders of articulation were always present; these disorders were described as a phonetic disintegration no different from those observed in adults, if the stage of development was taken into account. Hécaen (1976) suggested that articulatory disturbances occurred after either left or right hemisphere lesions and were frequent in children younger than 10 years of age at lesion onset. Hécaen (1983) also reported that articulatory problems occurred 81% of the time after left anterior lesions versus 20% after left posterior lesions. Such a high occurrence of articulatory disorders, as suggested by Hécaen, has not been substantiated in most recent reports or reported after the acute period. For example, Cranberg et al. (1987) reported that only one in eight children with acquired aphasia exhibited dysarthria at follow-up; Dennis (1980) noted dramatic improvement in articulation by 3 months following lesion onset in her 9-year-old child with a left posterior infarct; and Kershner and King (1974) found articulation er-
rors to be no more common among left- or right-hemiplegic children than among controls.

The only detailed study of early phonological ability in children with focal lesions is that of Marchman, Miller, and Bates (1991), who reported a longitudinal study of the phonology observed in babbling and in first words. These investigators noted a small proportion of "true" consonants for the infants with focal injury, along with idiosyncratic patterns of consonant development. In contrast to Hécaen (1983), Marchman et al. (1991) reported most improvement among children with anterior as opposed to posterior brain damage. The limited work on productive phonology is inconclusive. Aram et al. (1987) reported that in response to rhyming (phonemic) cues, left- but not right-lesioned children made more errors in word retrieval than did control children; however, Woods and Carey (1979) reported that left-lesioned children did not differ from controls on a task requiring rhyming and completing nursery rhymes.

Finally, Trauner, Ballantyne, Friedland, and Chase (1996) report what appears to be the only study of affective and linguistic prosody in children after focal brain damage. In this study both right- and left-hemisphere-lesioned groups were deficient on tasks involving expression of affective prosody and linguistic prosody, whereas only right-hemisphere-lesioned children were impaired on tasks of affective comprehension, suggesting no clear brain lateralization for prosody early in development.

In summary, very little systematic investigation of phonology or articulation has been pursued among children with acquired aphasia; the data are not sufficient to generalize as to conditions that may relate to the presence or absence of phonological and articulatory problems.

Reading

Despite relatively good recovery for spoken language, long-term reading and writing problems are often, although not always, reported to persist. Alajouanine and Lhermitte (1965) reported that none of their 32 children with acquired aphasia were able to follow normal progress at school; although they were able to regain what they had previously learned, they had difficulty learning new information. Eighteen of the 32 experienced persistent reading problems, 9 were totally unable to read; 5 had a severe alexia for letters, syllables, and words; and 4 had alexia for letters and somewhat better reading of words. Similarly, both Cranberg et al. (1987) and Cooper and Flowers (1987) found sizable numbers of their acquired aphasic children to have long-term word recognition or reading comprehension deficits.
Although reading usually is reported as more impaired than spoken language among children with acquired aphasia, this is not always the case at least acutely. For example, Dennis (1980) reported that at 2 weeks after lesion onset for a 9-year-old child, reading was higher than oral language, at 3 months, reading but not oral language was age-appropriate. This case demonstrated that, even among children with acquired aphasia, a dissociation between auditory and reading comprehension may exist. Hécaen (1976, 1983) is one of the few to suggest that, although reading problems may occur in the acute period, especially following left-hemisphere insults, reading problems usually disappear rapidly and completely.

Findings relating reading deficits to lesion laterality and age of onset are somewhat equivocal. Several investigators have compared reading in children with predominantly left or right hemisphere lesions. Although some researchers (Kershner & King, 1974; Reed & Reitan, 1969) reported no difference between left or right congenitally hemiplegic children on the Wide Range Achievement Test (WRAT; Jastak & Jastak, 1978), a test of single-word recognition, others found WRAT word recognition to be related to adequacy of right-hand performance, reflecting left hemisphere functioning (Kiessling et al., 1983). Vargha-Khadem, Frith, O'Gorman, and Watters (1983) reported that children with either left or right lesions tended to have more difficulty than control subjects on measures of reading speed and reading comprehension, whereas those with left-hemisphere lesions acquired postnatally were most impaired in their reading skills. Aram, Gillespie, and Yamashita (1990) reported few significant mean group differences between left-lesioned children and their controls or between right-lesioned children and their controls on a battery of phonetic analysis, word recognition, and reading comprehension tests, although mean performance of the lesioned subjects was consistently below that of controls. However, notable individual differences were present within the lesioned subject groups, with 5 of 20 left-lesioned and 2 of 10 right-lesioned children presenting marked reading deficits, in contrast to only 1 of 30 control subjects. Age of lesion onset was not found to differentiate those with and without reading problems, although a family history for reading disorders or involvement of specific subcortical structures was present for all subjects with reading problems.

Overall, it appears that long-standing reading problems involving phonetic analysis, decoding, and comprehension may occur for a sizable proportion of children with acquired aphasia. Although reading problems have been reported following a variety of acquired lesions, they appear to be most common among the children with postnatally acquired left lesions.
Writing and Spelling

Although it has been suggested that written language skills are particularly impaired among children with acquired aphasia, there appear to be no detailed reports of these children's writing ability. Alajouanine and Lhermitte (1965) reported that written language was always more severely disturbed than oral language. Among their 32 children with acquired aphasia, severe alterations in writing were noted in 19, 8 of whom could only copy words and 5 were said to be "dysorthographic" in their spontaneous writing. Even Hécaen (1976, 1983), who considered oral language and reading problems to disappear "rapidly and completely," noted that writing problems among children with acquired aphasia tended to persist and may even become permanent. Beyond a few case study examples in which writing skills were only one aspect described (e.g., Dennis, 1980; Ferro et al., 1982), apparently no studies have explored the nature of these written language impairments.

Spelling deficits, reported to be relatively common among children with acquired aphasia, also have not been described extensively. Cranberg et al. (1987) and Cooper and Flowers (1987) reported spelling problems in 3 of 8 and in 8 of 15, respectively, of their children with acquired aphasia. Woods and Carey (1979) reported that children with left-hemisphere lesions before as well as after 1 year of age were significantly poorer than controls in spelling a series of eight words. Vargha-Khadem et al. (1983) also found that children with left-hemisphere lesions performed more poorly on spelling tasks than those with right lesions or control subjects, especially children with postnatally acquired left lesions. This group had notable difficulty with infrequently occurring words; findings for frequently occurring words were less clear-cut. Vargha-Khadem et al. (1983) appear to be among the few who have provided qualitative data describing the spelling errors made by aphasic children, which were categorized as morphophonemic, orthographic, or preservation of the sound frame.

Thus, despite the observation that writing and spelling problems are relatively frequent and persistent among children with acquired aphasia, especially in those with postnatally sustained left-hemisphere lesions, the problems presented have not been detailed.

Summary of Language Characteristics

Although typically less severe than the deficits observed among adults with acquired aphasia, a range of language deficits have been described in both the acute period and in the long term among children with a variety
of acquired brain lesions. Syntactic comprehension disorders, although more pronounced in the acute phase, have been shown to persist long-term following predominantly left-hemisphere lesions. Lexical comprehension deficits also tend to persist, and may be especially pronounced after right hemisphere lesions. Reduced, syntactically simplified language output is the most commonly described expressive language characteristic observed among children with acquired aphasia, particularly subsequent to predominantly left hemisphere involvement. Paraphasias and more fluent-type aphasias, however, have been described usually during the acute phase of recovery or following diffuse brain involvement. Impaired naming and lexical retrieval exemplified by paraphasias, impoverished vocabularies, and slow rate of retrieval have been reported both in the acute and long-term periods, especially after left-hemisphere lesions. Data addressing phonological production and articulation are equivocal, with insufficient detail available to resolve the contradictory reports. Reading, writing, and spelling deficits are the most frequent, persistent, and significant sequelae among children with acquired aphasia. Reading deficits involving phonetic analysis, word recognition, and reading comprehension have been described, typically in association with left-hemisphere lesions. Writing and spelling limitations also often appear to be long-standing problems, predominantly associated with left-hemisphere lesions, yet data detailing either limitation are sparse.

Factors Related to Recovery of Language Abilities

Clearly, recovery of language skills among children with acquired lesions is variable, both within and across studies. The primary factors thought to be associated with how well a child recovers involve the nature of the neurological insult and the age of the child at lesion onset.

Nature of the Neurological Insult

The nature of the neurological insult appears to account for much of the variability in outcome reported for children with acquired aphasias. Several aspects relating to the neurological insult are examined here: the degree of brain involvement, the etiology of the lesion, lesion laterality (i.e., involvement of the left or right hemisphere), the specific site of lesion (i.e., the actual structures involved within a hemisphere), the size or extensive-ness of the lesion within a hemisphere, and the presence and severity of an accompanying seizure disorder.
DEGREE OF BRAIN INVOLVEMENT

Unlike most of the adult aphasia literature, the majority of studies addressing acquired aphasia in children are not confined to focal, more circumscribed lesions. Rather, most have included at least some children with known or presumed diffuse brain involvement, for example, secondary to asphyxia, head trauma, infectious processes, or cranial radiation and/or chemotherapy for the treatment of tumors. Often, however, the degree of more generalized involvement is not addressed; instead, lesions are treated as if the effect were confined solely to a focal area, for example, the location of a subdural hematoma after head trauma, or of a tumor with no mention of cranial radiation or chemotherapy. When results in these studies vary from findings in more circumscribed lesions, the findings need to be interpreted in light of some degree of probable diffuse brain involvement.

The few studies that have explicitly contrasted the effects of diffuse versus focal brain involvement on children's cognitive abilities, have consistently identified bilateral and/or diffuse hemispheric involvement as a poor prognostic sign (Hecaen, 1976; Janowsky & Nass, 1987; Loonen & Van Dongen, 1990; Van Hout et al., 1985). For example, as noted earlier, most of the children with severe comprehension disorders and persistent paraphasias (Van Hout et al., 1985) typically incurred diffuse brain involvement. Annett (1973) apparently was one of the first to study the relationship between the degree of more diffuse brain involvement and higher cognitive functions, including language. She reported an association between a decline in intelligence scores and increased impairment of the non-hemiplegic hand, thus demonstrating involvement of both hemispheres, not merely the more apparent side of hemiplegia. More recently, Loonen and Van Dongen (1990) reported an inverse relationship between recovery of spontaneous language and auditory comprehension skills and the degree of bilateral brain involvement. In general, then, it appears that lesions involving focal areas of the brain are associated with better recovery than are lesions involving diffuse areas of the brain.

ETIOLOGY

Closely related to the degree of brain involvement is the etiology of the brain lesion. In a review of 47 cases of acquired aphasia, Martins and Ferro (1987) reported that the prognosis for vascular and traumatic lesions was better than for encephalitis and tumors. Similarly, Van Hout et al. (1985) found a high incidence of infections, in particular herpes encephalitis, among her patients with the most severe and persistent language deficits. Many investigators have documented the deleterious effects on
higher cognitive function of cranial radiation and chemotherapy for CNS tumors in children owing to the diffuse effects of these treatments (e.g., Fletcher & Copeland, 1988). Guttmann (1942), Van Dongen and Loonen (1977), and Van Dongen and Visch-Brink (1988) suggested that children with aphasic symptoms recover more rapidly from head trauma than from vascular lesions; however, one might question the severity of their patients' head trauma, as the severity of head trauma is thought to be the single most important variable in determining recovery (Fletcher & Levin, 1988). When considering prognosis as it relates to etiology, severity of the injury or disease process must be considered, as well as the concomitant treatment. Thus, in general, etiologies implicating more diffuse brain involvement, including infectious processes, tumors treated with cranial radiation therapy and chemotherapy, and severe head injury are related to poorer outcome than more focal lesions, for example, as a result of vascular problems. Even within groups of children with focal lesions, however, outcome is variable. In these cases, the additional variables of lesion laterality, size, site, and presence of seizures need to be considered.

LESION LATERALITY

Woods and Teuber (1978) reported what is probably the landmark paper addressing lesion laterality and acquired aphasia in children. These investigators noted that since the introduction of antibiotics and mass immunization programs in the 1930s and 1940s, stemming previously common forms of diffuse brain involvement in children, the incidence of aphasia arising from right-hemisphere lesions is no higher than that reported in adults. Excluding earlier studies in which reports of diffuse brain involvement were frequent, these investigators reported that the incidence of aphasia associated with right-hemisphere lesions was less than 10%. If left-handers were excluded, the incidence dropped to 5%. Others have since substantiated that the incidence of aphasia after left lesions is comparable for right-handed children and adults, and the risk is substantially greater after left than right lesions at any age (Carter, Hohenegger, & Satz, 1982; Satz & Bullard-Bates, 1981). Such data prompted Hécaen to change his view regarding the lack of language lateralization in children and state, "One could reasonably conclude, therefore, that studies of acquired aphasia in children support the notion of early cerebral lateralization and even innate cerebral organization for the presentation of language" (1983, p. 586). In this chapter, deficits in syntactic comprehension, syntactic production, and naming and lexical retrieval were all associated with left- and not right-hemisphere lesions. Thus it appears that when lesions in children are confined to one hemisphere, just as in adults, aphasia is associated pre-
dominantly with left hemisphere involvement. Yet, as detailed earlier, right-hemisphere lesions may also have a subtle impact on many aspects of language, in particular, on lexical comprehension where deficits have been more marked after right-hemisphere than left-hemisphere lesions.

SITE OF LESION WITHIN A HEMISPHERE

Unlike most studies of adult aphasics, until very recently few studies with children provided sufficient evidence of lesion localization. In the past 15 years, studies have begun reporting computerized tomographic (CT) scan or magnetic resonance imaging (MRI) data for the majority of subjects, yet even these reports fail to define adequately the involvement of subcortical areas or to address actual areas of brain dysfunction. Nonetheless, some preliminary attempts toward localization have been offered.

Before the availability of CT scans and other noninvasive radiological evidence, findings were based on an array of laboratory (e.g., pneumocephalograms, arteriograms, surgery) and clinical (e.g., sensory and motor abnormalities) findings, with little consensus among reports. For example, Guttmann (1942) suggested that posterior left lesions resulted in more pronounced language deficits, whereas Hécaen (1983) concluded the converse, that anterior lesions produced more severe language deficits than did posterior lesions. The availability of CT scan data has not appreciably clarified the relationship between lesion location and language symptomatology. For example, Cranberg et al. (1987) found nonfluent aphasias to occur following either anterior or posterior lesions, and Visch-Brink and Sandt-Koenderman (1984) were unable to identify a relationship between lesion location as determined by CT scans and the occurrence of paraphasias. Similarly, in our series of studies assessing aspects of language among children with unilateral brain lesions (reviewed in Aram, 1988), we have been unable to identify involvement of localized cortical areas associated with specific language symptomatology beyond a slight trend for somewhat greater deficits following posterior left lesions, although this finding may interact with lesion size. In a series of studies (Aram, Ekelman, & Gillespie, 1989; Aram et al., 1990), however, we presented evidence of particularly pronounced language deficits for a small group of children with involvement of specific subcortical nuclei (the head of the caudate and the putamen) and the adjacent white matter tracts, suggesting that children with involvement of these structures may not recover as well as those with involvement of other portions of the left hemisphere. Whether these findings will be substantiated in work with larger groups of children remains to be seen.
LESION SIZE WITHIN A HEMISPHERE

Several studies have quantified lesion size based on CT scans and have attempted to draw relationships between lesion size and language sequelae, but with contradictory results. Several have suggested that the larger the lesion, the poorer the cognitive performance (Cohen & Duffner, 1981; Levine et al., 1987; Riva et al., 1986), whereas others have found little relationship between lesion size and recovery (Loonen & Van Dongen, 1990; Thal et al., 1991; Vargha-Khadem et al., 1985). Some investigators have suggested that lesion size may be important in older but not in younger age groups (Kornhuber, Bechinger, Jung, & Sauer, 1985). At this point the role lesion size plays in determining recovery is not clear, although it may be that size interacts with site and possibly age of onset.

PRESENCE OF SEIZURES

Most studies demonstrate poorer outcomes when seizures accompany acquired brain injury than when seizures are not present (e.g., Aicardi, Am-sili, & Chevrie, 1969; Annett, 1973; Solomon, Hilal, Gold, & Carter, 1970; Van Dongen & Loonen, 1977). Levine et al. (1987) appear to be among the few whose findings do not support such a relationship. These investigators reported that IQ deficits correlated with the presence of electroencephalographic (EEG) abnormalities; however, when lesion size was entered as a covariate, the relationship between IQ and EEG abnormalities no longer maintained, suggesting that lesion size rather than EEG abnormalities was a more powerful predictor of cognitive recovery.

Most clinicians and investigators, however, present evidence to the contrary, viewing the presence of seizures as a negative factor affecting outcome. Isaacs, Christie, Vargha-Khadem, and Mishkin (1996) reported that brain-lesioned children with a history of seizures were impaired on measures of dichotic listening, manual functions, and IQ, whereas those with no seizures were impaired only on manual functions. As well, Vargha-Khadem (1993) found that children with lateralized brain lesions with an accompanying seizure disorder were more impaired on an array of language and memory tasks than were lesioned children without seizures. Finally, Sussova, Seidl, and Faber (1990) found hemiparetic children with an accompanying seizure disorder to have lower IQs than those without seizures. Taken as a whole, there is a sizeable body of data demonstrating that the concomitant presence of seizure disorders appears to have an adverse effect on cognitive and language outcome, which most probably can be related to the spread of abnormal electrical activity, thus implicating more diffuse brain involvement than the more circumscribed effects of the original lesion. Furthermore, as pointed out by Isaacs et al. (1996), the pres-
ence of seizures also apparently interferes with compensatory mechanisms.

Although the overall prognosis for language recovery in children with seizures in general is poorer for children with seizures than without, one also needs to take into account the frequency, severity, and type of seizures, as well as the effectiveness, dosage, duration, and number of anticonvulsants used.

Age of Insult

Because children with acquired aphasia usually recover more rapidly and completely than adults, age is assumed to be an important factor in determining outcome. The presumed plasticity possible in the immature brain has typically been identified as the mechanism responsible for age-dependent recovery (e.g., Lenneberg, 1967). Also, much of the earlier work with animals suggested better recovery of functions following early rather than later lesions (Kennard, 1936), a position often referred to as the “Kennard principle.”

Evidence from children with acquired aphasia, however, is highly contradictory, and no single relationship between age at lesion onset and outcome is supported. Some researchers have suggested that lesions sustained before the onset of language do not have as significant an effect on language as those sustained after 1 year of age (e.g., Woods & Carey, 1979). Others have suggested that earlier lesions have more pervasive effects on development of higher cognitive functions than do later lesions (Basser, 1962). Most studies have failed to identify a clear relationship between age of lesion onset and recovery (Aram, 1988; Hécaen, 1976; Loonen & Van Dongen, 1990; Woods & Teuber, 1978). Some have suggested that the variable effect of age on outcome may be explained by other factors, for example, different etiologies among the age groups (Martins & Ferro, 1987). Finally, some studies suggest that the importance of age at lesion onset is not for predicting the rapidity or completeness of recovery, but for understanding the specificity of the lesion’s effect, dependent on the stage of language development at the time. For example, Alajouanine and Lhermitte (1965) contrasted language characteristics of children with lesions before and after 10 years of age, stating that reduced verbal expression, disordered articulation, and comprehension deficits are characteristic of the effects of lesions before 10 years of age, whereas paraphasias and written language disturbances are common when lesions are sustained after 10 years of age.

Thus, it does not appear that a single relationship between age of lesion onset and prognosis for language recovery holds; rather, age may interact
with other variables, such as etiology, and may exert a variable effect on language depending on the stage at which language is disrupted.

How Language Recovers

Although considerable theorizing has been offered to explain language recovery among children with acquired aphasia, how and where language recovers are still largely speculative issues. Several mechanisms have been suggested, such as functional substitution of "uncommitted" portions of the same or the opposite hemisphere, and redundant neural representations that make possible the release of existing but previously suppressed pathways (see Aram & Eisele, 1992, for a review of mechanisms proposed for recovery). Much of the evidence for functional reorganization following brain lesions has been derived from work with animals. The limited data available pertaining to reorganization of language functions among children with acquired aphasia has addressed only which hemisphere continues to be active during language tasks as opposed to where within a hemisphere language functions occur. The few studies available on individuals with early lateralized lesions have used dichotic listening, sodium amyotral, or electrophysiological procedures.

Evidence from Dichotic Listening Tests

Several investigators have used dichotic listening tasks to infer hemispheric laterality following early lateralized lesions. The dichotic paradigm presents different stimuli independently but simultaneously to each ear; the hemisphere contralateral to the ear through which the higher score of accurate recognition is obtained is considered to be dominant for that aspect of language (Berlin & McNeil, 1976). By presenting two-digit dichotic pairs to children and adolescents with lateralized seizures and hemiplegia, Goodglass (1967) found a dramatic inferiority of report from the ear opposite the injured hemisphere in most cases, with several instances of total suppression. Distinguishing between a "cerebral dominance effect," which usually favored one ear by only a small difference, and "a lesion effect," where difference ranged up to 100%, Goodglass suggested a parallel between lateralized suppression of auditory input and visual or tactile neglect, extinction, and displacement.

Subsequently, several other investigators have reported similar findings of inferiority of verbal recognition contralateral to the lesioned hemisphere and instances of total auditory suppression (Ferro et al., 1982; Isaccs et al., 1996; Martins et al., 1987; Pohl, 1979; Yeni-Komshian, 1977). Both Pohl (1979)
and Yeni-Komshian (1977) administered dichotic tests during the course of recovery and related dichotic findings to language improvement. Pohl (1979) studied a 6-year-old boy with a left middle cerebral artery occlusion at 8 months and again at 13 months after lesion onset. Under dichotic testing conditions, total right-ear extinction was found at both times and was not modified through verbal training. At 13 months, however, the right-ear extinction disappeared if words were presented monaurally to the right ear with white noise presented on the left. Pohl interpreted the right-ear extinction as signaling a switch in hemisphere dominance for speech from the left to the right hemisphere. Similarly, Yeni-Komshian (1977) described dichotic findings and language skills over time for four children with acquired brain damage. The three children with bilateral brain involvement all showed marked right-ear advantage initially and were unable to process competing stimuli, although they regained some capacity to do so over time. A relationship between the degree of language loss and the ability to process two competing stimuli was noted. Yeni-Komshian interpreted the pronounced right-ear advantage, which coincided with significant recovery of language, as an indication that language was originally represented in the left hemisphere and that recovery also took place in the damaged left hemisphere. In contrast, her fourth child, an 11-year-old boy with a total destruction of the left hemisphere, persisted in demonstrating a marked left-ear advantage, and, despite intensive therapy, a severe aphasia remained at 14 months following lesion onset. Yeni-Komshian proposed that the strong left-ear advantage was suggestive that language recovery, although impaired, was taking place in the right hemisphere. More recently, Isaacs et al. (1996), in a large-scale study of 62 children with left hemisphere injury and 53 with right injury reported a left-ear disadvantage for dichotic digits for 60% of children with left congenitally acquired lesions compared with 26% of control children, suggesting that for these children language representation had shifted to the right hemisphere.

Finally, Nass, Sadler, and Sidtis (1992) studied dichotic speech and complex pitch discrimination in children with congenital left or right hemisphere lesions. In contrast to adults with similar lesions, neither left- nor right-lesioned congenital groups were significantly impaired on speech discrimination, although both performed more poorly than controls in complex pitch discrimination. Nass et al. interpreted their findings as evidence for the relative sparing of function following congenitally acquired lesions. This also was evidence for the earlier maturation of the left hemisphere, rendering it less capable of assuming functions from a damaged right hemisphere, and for "crowding" of functions transferred to the right hemisphere, thus accounting for the proper performance of both groups on complex pitch discrimination.
These studies demonstrate the utility of dichotic listening tasks in providing one approach for identifying which hemisphere assumes language functions following acquired lesions.

Evidence from Sodium Amytal Studies

Probably the strongest evidence of hemispheric dominance for language comes from sodium amytal studies, also referred to as the “Wada procedure” after the neurologist who developed the technique. This technique uses a short-acting barbiturate injected into either the left or the right internal carotid artery, which is repeated on a separate occasion in the alternate carotid artery. During the short period in which the drug circulates through the hemisphere, functions normally sustained by that hemisphere are significantly impaired, thus permitting determination of that hemisphere’s role in a specific function. Because of the invasive nature of the technique, sodium amytal studies are usually restricted to preliminary assessment before surgical resection of the brain to relieve intractable seizure disorders to determine the effect of the surgery on language and memory functions. Thus, the patients for whom these data are available consist predominantly of persons with severe and often long-standing seizure disorders; nonetheless, for these patients a direct indication of hemisphere dominance for language can be obtained.

Rasmussen and Milner (1977) provided one of the most extensive summaries of sodium amytal findings as they pertain to lateralization of language functions following left hemisphere lesions sustained early in life. In a review of 134 patients in whom the epileptogenic lesions all occurred before 6 years of age, and in most instances from the prenatal period, the following data were reported. First, 81% of their left-lesioned patients who remained right-handed were also left hemisphere dominant for speech, suggesting that an early left-hemisphere lesion that does not modify hand preference is unlikely to change hemispheric dominance for language. In contrast, among the non-right-handed subjects with left-hemisphere lesions, 53% had right-hemisphere language representation and 19% had evidence of bilateral representation. Second, Rasmussen and Milner reported that speech could be mediated asymmetrically in the two hemispheres, with the anterior speech areas in one hemisphere and the posterior areas in the other hemisphere. Third, even gross lesions that did not involve the primary speech zone (the inferior frontal and posterior temporoparietal regions of the left hemisphere) rarely altered speech lateralization, whereas damage to either of these critical areas usually resulted in right or bilateral speech representation. Finally, they speculated that after 5 years of age recovery is achieved by intrahemispheric reorganization rather than by a
shift of hemispheric dominance, which suggested that upward displacement of the posterior speech zone to include more of the parietal cortex may provide such a compensatory mechanism.

In a more recent review, Mateer and Dodrill (1983) likewise found that left-hemisphere lesions involving the inferior frontal and posterior temporoparietal regions usually resulted in either right-hemisphere or, more rarely, bilateral hemispheric representation for language. In addition, they reported that for their group of patients with bilateral speech representation, the early brain injuries appeared to be diffuse and not lateralized to a single hemisphere. However, they pointed out that all instances of bilateral damage did not necessarily result in bilateral language representation. Mateer and Dodrill suggested that early diffuse injury may either actively inhibit language lateralization or possibly require contributions from both hemispheres for the support of language development.

In general, the data from sodium amytal studies do provide evidence for right or bilateral representation of language functions for some left-lesioned subjects, particularly when the patient is left-handed, when the primary speech zones are involved, and when injury occurs at a young age. Yet these data are derived from a small subgroup of individuals with intractable seizure disorders who require an invasive procedure for determining lateralization. Whether or not these findings generalize to other groups of brain-injured children is unclear.

Evidence from Electrophysiological Findings

Electrophysiological procedures such as those involving auditory evoked potential provide a noninvasive means of determining brain activity in response to language stimuli. As of yet, however, it appears that few investigators have applied these techniques to the study of brain organization for language of children with acquired aphasia. Papanicolaou, DiScenna, Gillespie, and Aram (1990) reported the use of the probe evoked potential paradigm (Papanicolaou & Johnstone, 1984) with a group of children with unilateral left lesions in the absence of seizure disorders. The left-lesioned children in this study displayed the normal pattern of predominantly left hemisphere engagement in a language task and right hemisphere engagement in a visuospatial task. Thus, among this group of left-lesioned subjects it appeared that language restitution and development involved intra- rather than interhemispheric functional reorganization. That 9 of the 14 children in this study were left-handed at the time of study is not consistent with Rasmussen and Milner’s (1977) findings reported with the sodium amytal procedure; however, differences may relate to the fact that in Papanicolaou et al.’s patients, lesions were focal and...
not accompanied by seizures, compared with Rasmussen and Milner’s patients who had intractable seizure disorders. Also, in the evoked potential study, the language tasks involved only a phonological target detection task, signaled by raising the index fingers, whereas the tasks used in Rasmussen and Milner’s sodium amytal study involved naming and sequential speech. Thus the task used in the evoked potential study did not require any language production and may not have tapped more anterior speech areas. Therefore, the Papanicolaou et al. findings cannot preclude the possibility, suggested by Rasmussen and Milner (1977), that speech functions among early lesioned subjects may be mediated asymmetrically by the hemispheres. Had a language production task been included, it is possible that results for that task may have been different from those for the phonological detection task.

Clearly, much remains to be learned about how language recovers among children with acquired aphasia. The few studies available suggest that both intra- and interhemispheric reorganizations occur. With the application of noninvasive techniques, such as evoked potentials, and more dynamic imaging techniques, such as positron emission topography and functional magnetic resonance imaging, greater understanding of factors related to the process of recovery from acquired aphasia in childhood hopefully will be forthcoming.

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References


Dorothy M. Aram


Introduction

In recent years, a major paradigm shift has occurred in the way that aphasia in traumatic brain injury (TBI) is considered. Previously, there was a bias toward defining the communicative impairments in both adult and pediatric populations with TBI within the narrow context of specific language disturbances. As such, language impairments were evaluated in the domains of phonology, semantics, morphology, and syntax. Disturbances in these specific language areas were identified by relying on structured language measures that had been developed for the purposes of either identifying the linguistic sequelae in adults with aphasia after stroke or determining the presence of developmental language problems in children. Current evidence indicates that the cognitive-communicative deficits in TBI are not readily detectable by structured language measures (Chapman, 1997; Hartley & Jensen, 1991; MacDonald, 1993).

As evident from the literature review that follows, the language-brain framework established by classic aphasia theory has contributed to our understanding of language deficits in TBI. Moreover, recent developments have illustrated that discourse measures provide valuable indices of communicative competence in patients with stroke-induced aphasia in addition to the performance data derived from aphasia batteries (Chapman & Ulatowska, 1994; Ulatowska & Chapman, 1994). Such developments have paved the way for using discourse procedures to characterize the communicative ability or disability in TBI. Growing evidence indicates that discourse measures are more sensitive to the cognitive-linguistic sequelae associated with brain injury in both adult and pediatric populations than are...
the structured measures of linguistic abilities (Chapman, 1997; Coelho, Liles, & Duffy, 1995; Hartley, 1995).

It is important to recognize that the cognitive-linguistic profiles in patients with TBI are distinct from the classic patterns seen in adult patients with stroke-induced aphasia or in children with developmental language delay. The distinctions between patients with TBI and adults with stroke-induced aphasia or children with developmental language problems arise largely from differences in the pathophysiology of the mechanisms of brain injury and from the diverse concomitant neurobehavioral disturbances. The management of the communicative disturbances in TBI requires an understanding of the neurological aspects of the injury; of the complex interactions between the injury mechanism, extent of brain injury, and disturbances to cognitive and linguistic-social systems; and of the clinical utility of discourse methods in elucidating the rich interplay between cognitive and linguistic abilities.

This chapter reviews the extant literature that has contributed to the current empirical and theoretical understanding of the cognitive-linguistic abilities in TBI populations. The specific issues address are

1. The pathophysiological profile of TBI.
2. The nature of the specific language disturbances identified in TBI.
3. The dilemmas in sorting out linguistic disturbances from confusion-al states.
4. Additional concomitant disturbances of speech and language.
5. The theoretical and clinical reasons for a shift in focus to discourse measures.
6. Critical issues related to recovery in communicative competence including age at injury, severity of injury, focality of lesion, and concomitant cognitive and behavioral disturbances.
7. Special considerations for management of cognitive-communicative behaviors.

Epidemiology and Mechanisms of Injury

Definitions

In contrast to the frequent occurrence of open head injuries from penetrating missile wounds (e.g., bullets, shell fragments) in casualties of war, closed head injury (CHI) predominates in civilian head trauma. The term CHI is used here to refer to head trauma in which the primary mechanism of injury is a sudden acceleration–deceleration imparted to the freely mov-
ing head. Impact of a blunt object is another common mechanism of CHI. The primary cause of CHI in many areas of the United States is vehicular accident (Kraus et al., 1984), whereas assault is a more frequent mechanism in some urban areas. Falls also are a common cause of head injury in young children.

Epidemiology

Kraus and coworkers (1984) reported incidence data based on all hospital admissions for traumatic brain injury in San Diego County. Using case ascertainment criteria, such as acute impairment of consciousness, Kraus et al. found an overall incidence of 180/100,000 population, which closely approximates previous findings reported for Olmstead County, Minnesota, over the period from 1935 to 1974 (Annegers, Grabow, Kurland, & Laws, 1980). As shown in the age- and sex-specific incidence curve (Figure 14.1), the incidence of head injury rises sharply in late childhood and reaches a peak exceeding 400/100,000 population in adolescent and young adult males. A second peak in incidence is seen in older adults, which could have an impact on rehabilitation services because of the shift in the age distribution of the general population. Although males predominate during most of the age span in hospital admissions of adults with CHI, the male–female disparity in head injury is low in young children and in adults over 70 years of age (Kraus et al., 1984).

Epidemiologic statistics vary depending on the source of information. An important epidemiologic finding in the San Diego study was that mild to moderate head injury accounted for about three-fourths of all admissions of acute head trauma (Kraus et al., 1984). In contrast, the impressions gained from rehabilitation studies about the frequency of aphasia probably reflect selection of more severely injured patients. At the other end of the continuum, available data probably underestimate the incidence of mildly injured patients, as many are treated and then released from emergency rooms.

Mechanisms of Injury

Neuropathologic investigation of the traumatized human brain (see Adams, Mitchell, Graham, & Doyle, 1977) and studies using experimental models of head injury in animals (see Ommaya & Gennarelli, 1974) have suggested that a primary mechanism of CHI is rotational acceleration of the skull, which produces shear strains within the intracranial contents. Histological study of the brains of patients dying soon after CHI has disclosed diffuse injury to the cerebral white matter, which apparently results
from shearing and stretching of nerve fibers at the moment of impact (Adams et al., 1977). Pertinent to the development of hemispheric disconnection, the corpus callosum is especially vulnerable to diffuse, mechanically induced shear strains. Ommaya and Gennarelli (1974) postulated that the severity of diffuse CHI follows a centripetal gradient; that is, the injury extends to the rostral brain stem only in cases with severe diffuse hemispheric injury. The bulk of cerebral white matter may be reduced further by delayed degeneration, which results in ventricular enlargement. Complications contributing to the severity of generalized CHI include brain swelling, increased intracranial pressure, hypoxia, and infection.

Focal lesions after CHI result from contusion of the brain surface by transient in-bending of the skull or by penetration of bone fragment in cases of depressed skull fracture, which may also produce brain laceration (Gurdjian & Gurdjian, 1976). Focal areas of ischemia are frequently present in the neocortex and basal ganglia (Graham & Adams, 1971). Stresses of the impact may cause arterial and venous tears resulting in intracerebral (see
Figure 14.2) or extracerebral hematomas. The orbital surfaces of the frontal and temporal lobes are particularly vulnerable to contusion by impaction against the bony sphenoid wing. Formation of hematomas is also common in this area. Large mass lesions may produce contralateral shift of midline structures and tentorial herniation of the temporal lobe, possibly involving the uncus and hippocampus.

Assessment of Initial Injury

Closed Head Injury

Closed head injury often produces a period of amnesia, if not loss of consciousness, immediately after impact. The acute severity of diffuse CHI is measured by the degree and duration of altered consciousness. Teasdale and Jennett (1974) developed the Glasgow Coma Scale (GCS; see Table
TABLE 14.1
The Glasgow Coma Scale

<table>
<thead>
<tr>
<th>Best eye opening</th>
<th>Best motor response</th>
<th>Best verbal response</th>
</tr>
</thead>
<tbody>
<tr>
<td>4 Spontaneous</td>
<td>6 Obey Commands</td>
<td>5 Oriented</td>
</tr>
<tr>
<td>3 To Speech</td>
<td>5 Localizes to Pain</td>
<td>4 Confused</td>
</tr>
<tr>
<td>2 To Pain</td>
<td>4 Flexion-Withdrawal to Pain</td>
<td>3 Inappropriate Words</td>
</tr>
<tr>
<td>1 None</td>
<td>3 Abnormal Flexion to Pain</td>
<td>2 Incomprehensible</td>
</tr>
<tr>
<td></td>
<td>2 Extension to Pain</td>
<td>1 None</td>
</tr>
<tr>
<td></td>
<td>1 None</td>
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14.1) for the assessment of coma. This scale consists of three components: the minimal stimulus necessary to elicit eye opening, the best motor response to command or to painful stimulation, and the best verbal response. Summation of the component scores of the GCS yields a total score, which can range from 3 to 13. Jennett et al. (1977) defined a severe acute CHI as one that results in no eye opening, inability to obey commands, and no comprehensible speech, that is, a GCS score of 8 or less for a period of at least 6 hours. Recent outcome research has typically defined a moderate CHI primarily by impaired consciousness (i.e., a GCS score from 9 to 12) which does not produce coma, whereas a mild CHI is reflected by confusion and disorientation (a GCS score from 13 to 15) with negative findings on brain-imaging studies (e.g., computerized tomography, magnetic resonance imaging).

Recent evidence raises a question concerning the contribution of acute linguistic disturbance to duration of impaired consciousness. To investigate the relationship between lateralization of focal parenchymal lesion and impaired consciousness. Levin, Gary, and Eisenberg (1989) serially assessed selected patients from the Traumatic Coma Data Bank who had unilateral intracerebral lesions of at least 15 cc until they recovered from coma. When the criterion for resolution of coma was the return of the ability to obey simple commands, patients with left-hemisphere lesions were found to have a more prolonged period (mean = 32.8 days) of impaired consciousness than had patients who sustained focal right-hemisphere insults (mean = 8.8 days). In contrast, lateralization of lesion had no effect when localization of a painful stimulus (e.g., moving an arm toward the site of supraorbital pressure) was the criterion for improved consciousness in patients who were initially comatose. The investigators interpreted these findings as evidence for the contribution of acute disturbance of receptive language to the impression of more prolonged impaired consciousness in
CHI patients with left-hemisphere lesions. An implication is that nonverbal modes of response and processing are necessary to evaluate recovery of consciousness in head-injured patients with left-hemisphere lesions.

Confusion and anterograde amnesia (i.e., the inability to consolidate information about ongoing events) usually persist for a varying duration after the patient emerges from coma (Russell & Smith, 1961). The duration of posttraumatic amnesia (PTA) may range from a few minutes after mild CHI that produces no coma to several months following severe CHI. The duration of PTA is assessed directly by questioning the patient concerning orientation and recent events (Levin, O'Donnell, & Grossman, 1979) and is estimated retrospectively by inquiring about the period for which the patient has no remembrance (Russell & Smith, 1961). Focal brain lesions (e.g., hematomas) may occur in the presence of relatively mild or moderate diffuse CHI, as reflected by the period of coma and PTA.

Missile injury causes tearing of the scalp, depression or fracture of the skull, and possibly wounding of brain tissue in the track of the foreign body (see Figure 14.3). A small shower of bone fragments is often project-

FIGURE 14.3. Gunshot wound of the right frontotemporal region visualized by computerized tomography. Note the path of the bullet and bone fragments which traversed to the left temporal area. Wernicke's aphasia with jargon persisted for 18 months postinjury in this woman.
FIGURE 14.4. Chart indicating the center of injury for missile wounds to the left hemisphere that caused aphasia. Cases with foreign bodies in remote regions of the brain were excluded from this map. Localization of missile wounds on the outline of a normal lateral skull was based on skull x-rays and surgical findings. From Russell and Espir (1961).

ed into the brain from the point of impact; the extent of dural penetration and loss of brain tissue are indexes of injury severity (Newcombe, 1969). As a consequence of dural penetration, posttraumatic seizure disorder is more strongly associated with aphasia secondary to missile wounds than in cases of CHI (Russell & Espir, 1961).

To determine the locus of lesion, Russell and Espir (1961) used surgical findings and lateral and anteroposterior skull x-rays to chart the entry wound and missile track on a lateral sagittal diagram of the hemisphere (see Figure 14.4). Verification of lesion localization by postmortem data suggested that this was a fairly accurate method. Although missile wounds tend to be more circumscribed than diffuse CHI and produce little or no coma, metal fragments can spread far from the primary locus of injury. Furthermore, Mohr et al. (1980) found that missile injury that produced a language disorder was frequently associated with a period of unconsciousness, suggesting that diffuse effects were contributory. Missile injury that results in aphasia also commonly produces motor and/or sensory deficit contralateral to the dominant hemisphere; this association is stronger than in the case of CHI (Levin, Grossman, & Kelly, 1976).

From this summary of the pathophysiology of head injury, we may in-
fer that clinical data concerning the extent of focal brain injury and the severity of diffuse cerebral disturbance are pertinent to the assessment of posttraumatic aphasia.

SPECIFIC LANGUAGE DISTURBANCES IN TBI

One distinctive feature of acute aphasia after CHI is the predominance of anomia (Heilman, Safran, & Geschwind, 1971). Fluent speech is often associated with verbal paraphasia and circumlocution; comprehension and repetition are relatively spared, whereas naming is markedly defective, especially to confrontation. Anomic errors include semantic approximation (e.g., "snout" for tusks of an elephant), circumlocution (e.g., "to make music" for pedals of a piano), and concrete representation (e.g., "orange" for a circle).

Wernicke's aphasia is the second most common language disorder after CHI. Although an acute picture of fluent paraphasic speech, poor comprehension for oral and written language, and impaired repetition has been described in CHI cases with left temporal lesions (Heilman et al., 1971; Stone, Lopes, & Moody, 1978; Thomsen, 1976), restoration of comprehension may be rapid after a hematoma resolves or is surgically removed (cf. Stone et al, 1978).

We describe a case of transient Wernicke's aphasia that occurred after a relatively mild diffuse injury (as reflected by the GCS) concomitant with a suspected left-hemisphere mass lesion that was not directly visualized by computerized tomography (CT). The patient was a 17-year-old right-handed student who sustained a closed head injury in a motorcycle accident. When admitted to the neurosurgery service on the day of injury, he had a GCS score of 11 and no focal motor or sensory deficit. CT showed compression of the left lateral ventricle, which resolved during the course of hospitalization. The patient's speech was fluent at a rate faster than normal and was contaminated by jargon (e.g., "ruby baby"). Comprehension was grossly impaired, and the patient's mood was characterized by excitement and agitation. Throughout the first 2 weeks of his hospitalization, he was grossly disoriented and continued to exhibit Wernicke's aphasia. Stereotyped phrases and expletives represented the primary verbal output. The patient's orientation began to improve and reached a normal level by 3 weeks postinjury. Although a clinical interview showed substantial improvement in his comprehension; the Multilingual Aphasia Examination (MAE) given the fourth week postinjury disclosed defective visual naming (e.g., he described a rectangle as a "long square"), inability to repeat sentences presented orally, and decreased word finding. Follow-up assessment 6 months postinjury revealed total recovery of language (see Figure 14.5).
Open Head Injury

Most published studies of aphasia after missile wounds to the brain are based on detailed observations of servicemen who were treated at the Military Hospital for Head Injuries in Oxford during and after World War II (Newcombe, 1969; Russell & Espir, 1961; Schiller, 1947). Mohr et al. (1980) and Ludlow and coworkers (1986) extended this research to include servicemen who sustained penetrating head injuries in Viet Nam. These au-
thors have frequently described linguistic disturbance characteristic of Broca’s aphasia, which is typically seen after occlusion of the left middle cerebral artery.

Russell and Espir (1961) obtained information on localization of injury by separately studying aphasics who had circumscribed left-hemisphere wounds without foreign bodies in remote areas of the brain (see Figure 14.4). In contrast to the rare occurrence of nonfluent agrammatic language disturbance after CHI, Russell and Espir (1961) reported that 12% of aphasics with missile wounds had Broca’s aphasia, which was typically associated with right-sided weakness and a focal injury to the frontal or Rolandic area. In a related study, Schiller (1947) linked a disturbance of articulation, inflection, and rate of speech with a wound at the foot of the precentral convolution. He observed that agrammatism, disturbed prosody, and perseveration were present in patients with left frontotemporal missile wounds. Russell and Espir found focal missile wounds in the dominant parietal lobe to result frequently in a global aphasia, although small posterior parietal lesions resulted in specific anomia, alexia, and agraphia. Similarly, Mohr et al. (1980) noted that parietal injury was more likely to produce aphasia than was a focal wound of any other lobe. Global aphasia with jargon, prolonged posttraumatic amnesia, and residual memory deficit have been observed during the early stage of recovery from penetrating injury of the left temporal lobe (Russell & Espir, 1961). Focal temporal wounds damaging the optic radiations resulted in a visual field defect in addition to global aphasia. Impairment of reading was common in these patients.

Russell and Espir (1961) analyzed the occurrence of aphasia after unilateral brain wounds separately for right- and left-handers. The authors defined handedness in terms of preference for a majority of motor skills. As anticipated from other sources of data concerning cerebral dominance, only 1% of right-handers became aphasic after right-hemisphere wounds, whereas 17% of left-handers became aphasic after right-hemisphere wounds. Also consistent with other lines of evidence for cerebral dominance, unilateral left-hemisphere wounds more frequently produced aphasia in right-handers (65%) than in sinistrals (38%). The overall figures for aphasia after unilateral injury of either hemisphere were 37% for right-handers and 27% for left-handers. Although these figures are incompatible with the concept that sinistrals are more likely to become aphasic, the comparative data presented by Russell and Espir included right-handed patients with lesions outside the speech territory. Of the right-hemisphere wounds that resulted in aphasia in sinistrals, the frontal or parietal lobes were involved in all cases.

In summary, the pattern of aphasia observed after missile wounds to the brain conforms fairly well to the localization of language in patients with
cerebrovascular disease (see Chapter 3 for background). The localizing significance of missile wounds is greatly enhanced by identifying patients in whom there is no evidence of additional brain penetration by fragments in remote areas (see Figure 14.4).

**Language Disturbances versus Confusional States**

**PERIOD OF POSTTRAUMATIC AMNESIA**

As mentioned in the review of mechanisms of injury, the early postcomatose stage of recovery from CHI is typically characterized by an amnesic condition during which the patient is confused. Reduplicative paramnesia (Benson, Gardner & Meadows, 1976)—that is, the mistaken identification of a person, place, or event for one previously experienced—confabulation, and profound impairment of memory may be misinterpreted as signs of language disorder. The distinction may be particularly difficult in a patient whose fluent speech is disconnected and perseverative. Confused, nonaphasic speech after CHI was evident in a patient studied in Galveston.

A 24-year-old, right-handed woman was transferred from a community hospital to the University of Texas Medical Branch 3 hr after she sustained a CHI in a motor vehicle accident on 28 January. The GCS score was 8 when she was initially examined. The cerebral ventricles and cisterns were poorly visualized on CT, suggesting the presence of diffuse cerebral swelling. Although she obeyed commands after 4 days, her disorientation persisted until 5 March. Spontaneous speech during the confusional period was continuous, rambling, and disorganized in this fearful, agitated woman. When queried on 27 February regarding the reason for her hospitalization, she responded, “Something that came up natural being born somewhere born somewhere in here.” Later in the day the patient elaborated that she was in the hospital “to have a baby” and that the year was 1952 (she was born in 1953). Reminiscent of the patients described by Weinstein and Kahn (1955), during examination the following day, she commented that she was hospitalized because of “being stabbed.” This statement was interpreted as a reference to her intravenous tubes. Assessment of language on 19 April, however, disclosed completely normal findings on the MAE.

Analysis of this patient's utterances provided little evidence of consistent paraphasic errors, particularly when she was asked structured questions that focused on specific objects rather than on expository material. Repeated questioning within the limits of her short attention span disclosed no evidence of receptive impairment similar to that found in patients with Wernicke's aphasia. Administration of tests of naming and
word finding to this patient during PTA would likely have yielded defective scores. Weinstein and Kahn (1955) described patients with brain damage of diverse etiologies, including diffuse cerebral disturbance, who exhibited misnaming that was qualitatively atypical for aphasia. Anomic errors were frequently associated with objects that bore a relation to the patient's illness and frequently occurred during a period of disorientation, confabulation, and denial of illness. The authors observed that, in contrast to patients with classical anomic aphasia, patients exhibiting nonaphasic misnaming frequently showed no evidence of groping for words in their spontaneous speech nor did their naming necessarily improve when correction was offered.

Conversely, the presence of paraphasic errors in conversational speech after CHI may be misinterpreted as evidence for disorientation and confusion. This condition is likely to be found in CHI patients with mass lesion or depressed skull fracture involving the left hemisphere. In such cases, a multiple-choice format of testing orientation may be useful, as well as relatively nonverbal tests during the early stages of recovery.

**POSTCONFUSIONAL STAGE OF RECOVERY**

Few studies of aphasia during the early stages of recovery from CHI have concurrently assessed orientation. Consequently, there is a possibility that PTA had not completely resolved at the time language was evaluated.

Clinical examination of language in consecutive CHI admissions at the Boston City Hospital by Heilman et al. (1971) yielded 13 cases of aphasia, including 9 patients with anomic aphasia and 4 cases of Wernicke's aphasia. Aphasics accounted for 2% of the Boston series, a base rate close to that obtained in a previous study of consecutive CHI admissions (Arseni, Constantinovici, Iliescu, Dobrota, & Gagea, 1970). In the Boston study, the authors defined anomic aphasia as a fluent aphasia in which the patient demonstrates verbal paraphasia for all kinds of material, especially to confrontation. Wernicke's aphasia was defined as a fluent aphasia with paraphasia, impaired comprehension for spoken and written language, and poor repetition. Broca's aphasia was defined as nonfluent aphasia with relatively intact comprehension. No patient had a Broca's aphasia or exhibited a total disruption of language. Heilman et al. (1971) excluded patients with intracranial surgery (other than evacuation of subdural hematoma). This strategy of patient selection, combined with the relatively high proportion of falls relative to motor vehicle accidents, may have restricted patients with mass lesions, thereby resulting in fewer aphasic cases compared with those of other neurotrauma centers. Heilman et al. (1971) distinguished the anomia in their CHI patients from nonaphasic misnaming
(Weinstein & Kahn, 1955). In contrast to the narrow range of anomic errors (e.g., related to illness) in cases of nonaphasic misnaming, the anomic CHI patients described by Heilman et al. exhibited diverse naming defects in spontaneous speech and writing.

As shown in the following case, we have also been able to distinguish anomic aphasia from nonaphasic misnaming by delaying evaluation of language until the injured patient recovers to a normal level of orientation. This case was a 17-year-old student who was transferred to the University of Texas Medical Branch 3 hours after a motor vehicle accident. The GCS score on admission was 8. Although the patient obeyed commands on the day of admission, delayed neurological deterioration was reflected by the development of a right hemiparesis and evolution of a Wernicke's-type aphasia. Three days postinjury a partial left temporal lobectomy was performed with evacuation of an intracerebral hematoma. The patient remained confused for a month after injury but exhibited gradual improvement of receptive language. Administration of the MAE two months after injury disclosed findings consistent with an anomic aphasia. There were frequent errors of circumlocution (e.g., she described an island as “a place where you fish”), semantic approximation (e.g., she described the trunk of an elephant as a “nose”), and a tendency to substitute names of concrete objects for geometric designs (e.g., she described a triangle as “the thing you use when you play pool”). As shown in Figure 14.6A, the patient’s long-term recovery of language was complete, except for a subtle residual anomic disturbance that was evident only under testing conditions.

Anomic aphasia may also persist after resolution of PTA in patients with severe diffuse CHI who evidence no other focal neurologic signs. In a study of 26 CHI patients without mass lesions who had been in a coma for a least 24 hours, Thomsen (1975) found that aphasic symptoms were present during the first 2 or 3 weeks after injury in 12 cases. Verbal paraphasia (i.e., substitution of inappropriate words) and anomia were the most common defects; receptive impairment and dysgraphia were also frequently observed, whereas agrammatisms and other symptoms suggestive of Broca's aphasia were rarely seen.

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FIGURE 14.6. (A) Baseline and follow-up language profiles of a 17-year-old student with residual anomia that was initially accompanied by a receptive impairment after surgical evacuation of a left temporal intracerebral hematoma. (B) Baseline and follow-up findings in a 19-year-old student which show persistent impairment of expressive and receptive language associated with cognitive deficit and progressive ventricular enlargement. He sustained a severe diffuse injury (coma = 21 days) complicated by bifrontal subdural hematomas. VN, Visual Naming; TNR, Tactile Naming, Right Hand; TNL, Tactile Naming, Left Hand; SR, Sentence Repetition; COWA, Controlled Word Association; WD, Writing to Dictation; WC, Writing–Copying; TOKENS, Token Test; ACWP, Auditory Comprehension of Words and Phrases; RC, Reading Comprehension. From Levin, Grossman, Sarwar, and Meyers (1981). Reproduced with permission by the publisher.
Expressive Tests

Receptive Tests

A

Expressive Tests

Receptive Tests

B
The following is a case that closely resembled the series of diffuse head injuries described by Thomsen. A 20-year-old, right-handed man was admitted to the hospital with a severe CHI (GCS score = 5) but no focal motor deficit. The CT scan suggested generalized brain swelling without a mass lesion. Baseline assessment 4 months after injury disclosed anomic errors that deteriorated into jargon (e.g., the handle of a fork was described as a “forkline” and the posterior aspect of the leg was described as a “negline”). Circumlocution was evidenced by his response when the examiner pointed to the pedals of a piano: “If you want a different sound, push them down.” Word finding was defective on a test of letter–word association.

Clinical examination of language has disclosed a broad range of language defects in CHI patients. Thomsen (1976) characterized the findings in a series of patients with left-hemisphere mass lesions as “multisymptomatic aphasia.” She used this term to describe patients who exhibited anomia, agraphia, and impaired comprehension; one-third of the patients in her series had global or receptive aphasia. Anomic aphasia was less common, and there were no cases of Broca’s aphasia. Posttraumatic dyslexia and dysgraphia have also been reported by other authors (de Morsier, 1973).

Additional Concomitant Disorders of Speech and Language

Posttraumatic disorders of speech and language may also include mutism, stuttering, echolalia, palilalia, and dysarthria.

MUTISM

Total abolition of speech and language may occur after termination of coma in patients capable of following commands during the transition between spontaneous eye opening and recovery of orientation. As previously described, transient mutism is characteristic of aphasia after head injury in children.

Prolonged if not permanent speechlessness is observed in adults who are persistently vegetative or exhibit akinetic mutism (Cairns, 1942; Plum & Posner, 1980). The akinetic type is a form of subacute or persistent mutism with little or no vocalization. Behaviorally this condition is distinguished from the vegetative state by its immobility. The features common to both conditions include apparent wakefulness with restoration of the sleep–wake cycle and inability to demonstrate cognitive function through interaction with the environment. When akinetic mutism is a sequel to CHI, diffuse cerebral injury is to be suspected.

Geschwind (1974) distinguished between nonaphasic and aphasic
mutism. The aphasic type, which was thought to occur rarely in adults with CHI (cf. de Morsier, 1973), is accompanied by linguistic errors in writing. Nonaphasic mutism is associated with acute onset of right hemiplegia; writing is normal and there are no signs of aphasia when speech is restored. Following Bastian (1898), Geschwind referred to this condition as aphemia rather than aphasia. In such cases mutism may arise from focal lesions, often involving the basal ganglia. In the following paragraph, we describe a case of subcortical mutism.

In a prospective study of patients admitted to neurosurgery services in Houston and Galveston, posttraumatic mutism was present in nine patients (nearly 3% of the series) despite recovery of consciousness and communication through a nonspeech channel (Levin et al., 1983). CT scans revealed subcortical lesions situated primarily in the putamen and internal capsule of four patients, and four of the remaining five without subcortical lesions had left-hemisphere cortical injury. The patients without subcortical injury visualized by CT exhibited a longer duration of impaired consciousness consistent with diffuse brain injury and showed more long-term linguistic deficits. The four patients with basal ganglia lesions included two children and an adolescent, a finding consistent with other evidence that basal ganglia lesions in CHI may be more common in the pediatric age range.

STUTTERING

Published studies suggest that stuttering is a more common sequel of penetrating missile wound than of CHI (Peacher, 1945). De Morsier (1973), however, noted a fluency disorder in more than half of his series of CHI patients, including four cases with posttraumatic stuttering. Helm, Butler, and Benson (1978) implicated bilateral injury in patients with acquired stuttering after CHI.

ECHOLALIA AND PALILALIA

Echolalia is the repetition of words spoken by others, whereas palilalia is the automatic repetition of one’s own words. Echolalia may follow a period of mutism in cases with diffuse cerebral dysfunction (CHI) or may occur in patients with transcortical motor aphasia, that is, disturbed expressive and receptive language with preserved repetition. Apart from generalized cerebral disturbance, these disorders have been associated with large frontal lesions. According to Geschwind (1974), echolalia and palilalia are uncommon in patients with lesions primarily involving the perisylvian region of the dominant hemisphere.

Stengel (1947) distinguished between the automatic and mitigated forms of echolalia. The former is parrot-like, with no elaboration of the in-
Mitigated echolalia is the questioning repetition of words spoken by others, often with a change of personal pronoun. Stengel postulated that mitigated echolalia may facilitate comprehension in patients with receptive language disturbance. Accordingly, the transition from automatic to mitigated echolalia may be a sign of clinical improvement that parallels the developmental sequence in children. Stengel also observed that the mitigated type may be confined to social conversation and less evident when the patient is directly questioned by an unfamiliar speaker.

Thomsen (1976) reported three cases of echolalia in a series of 50 patients with severe CHI. Of the two echolalic patients with left-hemisphere mass lesions, one initially had a global aphasia and the other had minimal spontaneous speech. The third patient, who sustained a severe diffuse CHI with residual hydrocephalus, evidenced echolalia and palilalia. In contrast to the general association of echolalia with impoverished spontaneous speech (see Geschwind, 1974), Thomsen commented that the patient with diffuse CHI talked incessantly without monitoring the amount of output. We also studied a patient who developed a similar echolalia after CHI.

An 18-year-old, right-handed student was brought to the emergency room of the University of Texas Medical Branch shortly after an automobile accident on 11 May. Initial examination disclosed a GCS score of 6, fixed and dilated pupils, and a right hemiparesis. A CT scan on the day of injury was normal. She slowly improved and eventually followed commands on 9 June. After transfer to the Del Oro Rehabilitation Hospital in Houston on 16 June she remained confused and disoriented until 26 June. During this period, the patient's spontaneous speech changed from an overall impoverishment to a greater-than-normal flow in which automatic echolalia was prominent. Observations by neuropsychologists during the course of rehabilitation showed a transition to mitigated echolalia which resolved by the middle of July. Repetition was most evident in the presence of persons familiar to the patient. An aphasia examination on 16 July disclosed intact spontaneous speech and relatively normal naming. Echolalia had resolved, but repetition of sentences and verbal associative fluency were markedly impaired. Comprehension of complex commands on the Token Test was also defective, although the patient could read and comprehend single words and phrases. Further progress in rehabilitation was complicated by her disinhibited behavior, a finding in agreement with Stengel's (1947) interpretation of echolalia as a failure of inhibitory control.

In summary, echolalia and palilalia are infrequent sequelae of CHI that are found in cases with severe diffuse CHI or with large mass lesions in the dominant hemisphere. The absence of any reference to echolalia and palilalia in several studies supports the contention that they rarely occur.
after CHI (Levin et al., 1976; Najenson, Sazbon, Fiselzon, Becker, & Schechter, 1978; Sarno, 1980).

**DYSARTHRIA**

Sarno (1980) defined dysarthria as a speech disorder arising from pathology in the motor speech system that is evident in defects of the acoustic aspects of the speech stream (i.e., articulation, resonance, stress, and intonation). The severity of dysarthria varies from articulatory imprecision to completely unintelligible speech. Dysarthria may be caused by a lesion of either the central or the peripheral nervous system. Peacher (1945) reviewed the cases of dysarthria recorded by U.S. Army hospitals during World War II. Of the injuries producing dysarthria, which were primarily missile wound, 69% involved a lesion of the peripheral nerves. Trauma to the facial nerve was the most common site of lesion, although Peacher did not distinguish between central and peripheral facial nerve injuries.

Investigators of speech disorder after CHI have frequently reported dysarthria in patients with focal mass lesion of the left hemisphere (Alajouanine, Castaigne, Lhermitte, Escourolle, & De Ribaucourt, 1957; de Morsier, 1973; Thomsen, 1975) and in cases of diffuse cerebral injury (Sarno, 1980, 1984; Sarno, Buonaguro, & Levi, 1986; Thomsen, 1976). Dysarthric patients are frequently hemiparetic or may be quadriplegic. Serial assessment of language after severe CHI has suggested that dysarthria often accompanies aphasia during the early stage of recovery from CHI, and may persist after restoration of language. This dissociation is illustrated in a patient who was admitted to the University of Texas Medical Branch.

A 33-year-old, right-handed carpenter sustained a severe CHI in a motorcycle accident on 17 December. Evaluation in the emergency room shortly after injury disclosed a GCS score of 4. A CT scan showed a large left parietotemporal epidural hematoma, which was evacuated on the day of admission. Although he progressively improved and followed commands on 20 December, a left facial palsy and right hemiparesis remained. The combined aphasia and severe dysarthria rendered his speech unintelligible. By the first week in January, the patient's language and speech disorder partially resolved, although he continued to evidence anomia and impaired comprehension. A CT scan 10 months postinjury disclosed a large hypodense area at the site of the operated hematoma and a small hypodense area in the genu of the left internal capsule which was interpreted as a small lacunar infarct. He was transferred to a rehabilitation center prior to neuropsychological evaluation but returned a year later for testing. Despite frequent articulatory defects, expressive and receptive lan-
guage skills had uniformly recovered, as reflected by normal scores on all subtests of the MAE.

In contrast to this patient's case, Sarno (1980, 1984) and Sarno et al. (1986) reported that subclinical language deficit (e.g., decreased word fluency) was present in all dysarthric patients in a series of CHI cases. The findings in the Galveston patients suggest that the correspondence between language skills and motor speech varies depending on the interval between injury and assessment.

We may conclude from these studies that assessment of communicative disorder after CHI should include evaluation of dysarthria. The tests for articulatory agility and rating speech characteristics that are included in the Boston Diagnostic Aphasia Battery (Goodglass & Kaplan, 1983) are brief and useful for this purpose.

Cognitive–Communicative Deficits beyond Aphasia

The administration of standardized examinations for aphasia has yielded a characteristic profile of language disturbance in adults after CHI. This strategy has disclosed that language processing deficits on testing in the absence of clinical manifestations of classical aphasia is a common finding in CHI (Sarno, 1980, 1984; Sarno et al., 1986). Moreover, quantitative and qualitative assessment of discourse has facilitated the study of long-term recovery in CHI (cf. Levin, Grossman, Sarwar & Meyers, 1981).

Structured Language Measurement

Profiles of language disorder after CHI have been developed using the MAE (Benton, 1967; Benton & Hamsher, 1978) and the Neurosensory Center Comprehensive Exam for Aphasia (NCCEA; Spreen & Benton, 1969). The MAE evaluates expressive language on subtests of naming pictures of objects (Visual Naming), Sentence Repetition, Digit Repetition, and retrieving words beginning with a designated letter (Controlled Word Association). Benton and Hamsher (1978) included a spelling test in their 1978 revision of the MAE. Comprehension of oral language is evaluated by the Token Test and by a receptive test in which the patient points to the picture corresponding to a word or phrase presented orally (Aural Comprehension of Words and Phrases). Reading comprehension is tested using a similar format. The NCCEA (Spreen & Benton, 1969) includes similar tests, in addition to tests of naming objects presented tactually (Tactile Naming),
construction of sentences (Sentence Construction), identification of objects by name, oral reading, writing names, writing to dictation and copying, and articulation. Both examinations yield a percentile score based on normative data for each subtest; the manual for the NCCEA also provides percentile scores based on the performance of aphasics. Gaddes and Crockett (1973) published normative data for children on the NCCEA. The Boston Diagnostic Aphasia Test (Goodglass & Kaplan, 1983) also provides a profile of language abilities. It incorporates tests for articulation, repetition of automatized sequences, and rating of spontaneous speech.

We administered portions of the MAE and NCCEA to a consecutive series of patients with CHIs of varying severity (Levin et al., 1976). In this study, injury that produced no neurological deficit or loss of consciousness longer than a few minutes was designated as Grade I; Grade II referred to an injury producing a coma of not longer than 24 hr; and Grade III designated an injury that resulted in a period of coma exceeding 24 hr. A language subtest score that fell below the second percentile of the normative population was considered defective. Whereas clinical examination of spontaneous speech disclosed evidence of aphasia in only eight patients (16% of the series), nearly one-half of the patients were impaired in naming objects (Figure 14.7). Word-finding difficulty (Controlled Word Association) and impaired writing to dictation were also common expressive defects in this series. In contrast, Figure 14.7 shows that repetition of sentences was well preserved. Nearly one-third of the patients had difficulty in comprehending complex oral commands on the Token Test. The results provided strong support for the presence of higher-level deficits in manipulating the system in apparently nonaphasic CHI patients, including cases with injuries of moderate severity.

Sarno et al. (1986) elucidated the characteristics of language and speech disorder after CHI in a study of 125 CHI patients who were rendered comatose for periods ranging from 15 min to 6 months. These patients were referred to the Rusk Institute for Rehabilitation Medicine in New York. On the basis of clinical evaluation and administration of subtests of the NCCEA (median injury–test interval of 45 weeks), the authors classified the patients into categories of grossly obvious aphasia, dysarthria with language deficit reflected by test scores ("subclinical" aphasia), and language deficit without dysarthria. They found that the proportion of patients with each category of language disturbance was approximately equal. The aphasic group (n = 37) consisted of 19 patients (51%) with fluent aphasia, 13 (35%) with nonfluent aphasia, and 5 (14%) with global aphasia. As depicted in Figure 14.8, aphasic patients had linguistic test scores that fell below the other groups. Most aphasics had defective scores on all four lan-
guage subtests that were administered, whereas 56% of the subclinical patients failed at least two of the four tests. The patients with dysarthria had word fluency and sentence repetition scores that fell below the pure subclinical group. No CHI patients in this series, however, obtained completely normal scores.

Figure 14.8 shows the test results of the CHI patients in Sarno et al.'s (1986) study transformed into percentile scores for an aphasic population. Accordingly, any score below the 90th percentile is impaired in relation to normal subjects. The mean scores indicated reduced word fluency in the subclinical groups, although visual naming is also compromised. Consistent with the results of the Galveston study, Sarno’s subclinical patients without dysarthria had adequate sentence repetition, whereas the dysarthric patients exhibited difficulty on this task. Impaired comprehension of complex oral commands was also found in patients without obvious
aphasia. In a discussion of the results obtained in the Galveston study and in her own investigation of language disturbance after CHI, Sarno and colleagues (1986) concluded that sophisticated, neurolinguistic measures were necessary to detect the nature of the deficits in this population to guide effective clinical judgment.

FIGURE 14.8. Mean percentile rank of aphasic and subclinical language disorder groups on subtests of the Neurosensory Center Comprehensive Examination of Aphasia. ———, Aphasic; ——, dysarthric and subclinical; ——, subclinical. Note that the percentile scores are based on results obtained in an aphasic population. From Sarno, Buonaguro, and Levita (1986). Reproduced with permission by the author and publisher.
Disparity between Discourse and Results on Structured Tests

Recent studies have shown that head injury and other etiologies of acquired brain damage can produce a residual impairment of discourse ability despite recovery of performance on structured language tests as described in the previous section. Consistent with description of residual speech following severe head injury as being tangential, if not confabulatory, several studies have reported diminished efficiency of communication in the discourse of CHI patients (Chapman 1995; Ehrlich, 1988; Mentis & Prutting, 1987; Novoa & Ardila, 1987; Penn & Cleary, 1988; Wyckoff, 1984). The studies by Chapman (1995), Ehrlich (1988), Mentis and Prutting (1987), and Wyckoff (1984) were confined to CHI patients, whereas the other investigations included other etiologies. Techniques used in studies of discourse include asking patients to generate stories in response to a series of pictures (e.g., comic strip), to relate a personal story, to retell a story, and to generate a procedural narrative such as how to buy groceries (Biddle, McCabe, & Bliss, 1996; Coelho, et al., 1995; Wyckoff, 1984).

An important methodological aspect of discourse approaches is that they offer ways to evaluate the ability to manipulate larger units of language than can be realized through examining competence at the single-word or sentence level. It is now widely recognized that the ability to utilize the formal aspects of the language system (e.g., semantics, syntax) does not necessarily correspond with the ability to process discourse-level information (Chapman, Watkins, et al., 1997).

Discourse in Adults with TBI

In a study that analyzed the discourse responses for two narratives (in response to a comic strip and retelling a story) and one procedure (explaining how to buy groceries), Wyckoff (1984) compared the findings obtained in 11 survivors of severe CHI (including five patients who were considered to be aphasic according to a standardized battery of language tests) with a group of normal subjects. The discourse of the head-injured patients was characterized by slow speech, in which a greater percentage of syllabic utterances were dysfluencies, and by generally diminished productivity. The head-injured patients produced about one-half to two-thirds of the amount of accurate content relative to the discourse of normal speakers. Moreover, the discourse of head-injured patients contained fewer cohesive ties between utterances, a feature exemplified by failure to provide a ref-
Wyckoff concluded that discourse analysis could potentially identify difficulty in functional communication, which would otherwise be overlooked by traditional, standardized language tests. At the same time, she suggested that further study utilizing spontaneous conversation with another speaker could potentially identify additional deficits, such as difficulty in topic maintenance.

In a study of 10 nonaphasic CHI patients who returned for linguistic examination at least 6 months posttrauma, Ehrlich (1988) analyzed the stories that they recited in response to the “Cookie Theft” picture from the Boston Diagnostic Aphasia Examination (Goodglass & Kaplan, 1983). The only difference that emerged in the comparison of narratives by these patients with those of normal adults was a reduction in the amount of information per unit of time. This pattern reflected the tendency of CHI patients to utter lengthier, slower spoken language.

More detailed analysis of discourse in CHI adults has involved assessment of cohesion using the technique developed by Halliday and Hassan (1976): two items are cohesively related (i.e., a cohesive tie) if the interpretation of one of the elements in a text depends on reference to another (based on grammar and vocabulary). This methodology has shown that head-injured survivors frequently fail to provide the speaker with a referent. Fewer cohesive ties per communication were found in the discourse of survivors of severe CHI compared with that of normal subjects (Hartley & Jensen, 1991; Liles, Coelho, Duffy, Robert, & Zalageus, 1989; Wyckoff, 1984). Mentis and Prutting (1987) studied cohesion in the discourse of three patients who had sustained a severe CHI at least 1 year earlier. In common with the patients studied by Ehrlich (1988), these CHI survivors were nonaphasic according to clinical examination and to their scores on a conventional aphasia test battery. A conversation and narrative language sample (e.g., description of the patient’s work) with a familiar partner were videotaped. Although the number of cohesive ties was similar in the conversation of the two groups, the normal subjects had a greater number of cohesive ties in their narratives compared with the CHI patients, who exhibited no variation under the two conditions. The investigators postulated that word retrieval problems contributed to the reduced number of cohesive ties in the narratives of the head-injured patients.

In addition to cohesive analysis, Liles and colleagues examined complete episodic structure in the narrative discourse of adults with TBI. These researchers found that adults with TBI produced fewer complete episodes compared with normal control subjects. Other studies have verified the finding of incomplete narrative structure in adults with TBI manifested by omission of critical information (Biddle, et al., 1996; Glosser & Deser, 1990; Hartley & Jensen, 1992).