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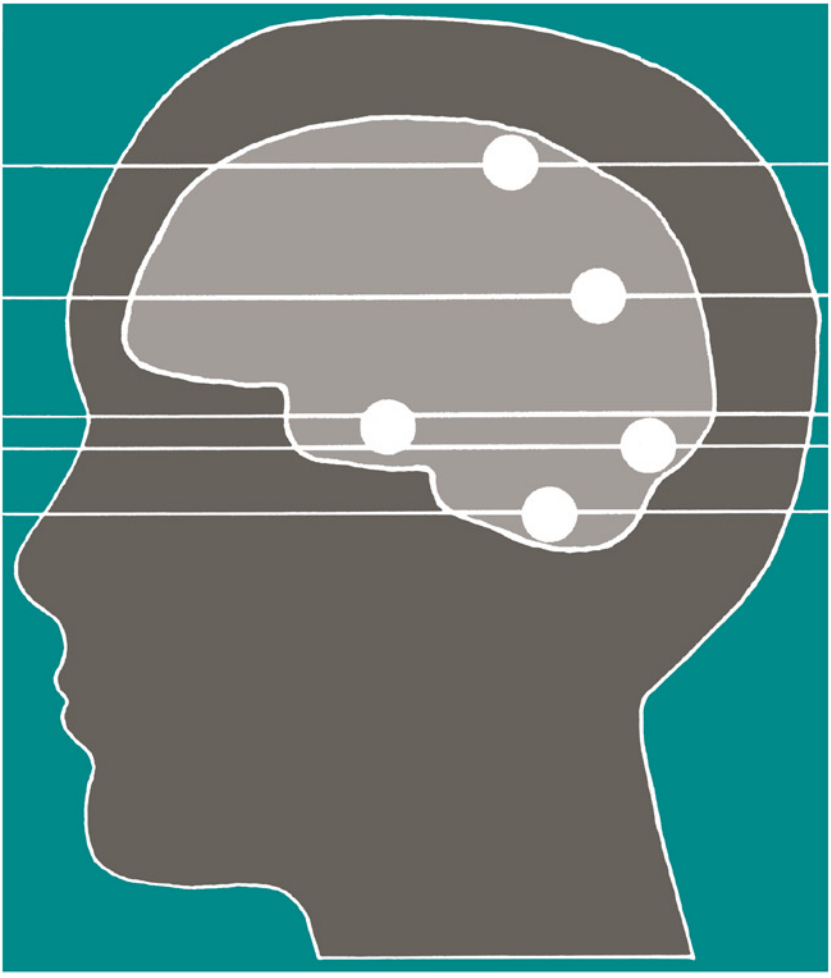

ACQUIRED NEUROLOGICAL SPEECH/LANGUAGE DISORDERS IN CHILDHOOD

Edited by Bruce E. Murdoch

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Contents

Series Preface

From being an area primarily on the periphery of mainstream behavioural and cognitive science, neuropsychology has developed in recent years into an area of central concern for a range of disciplines. We are witnessing not only a revolution in the way in which brain-behaviour-cognition relationships are viewed, but a widening of interest concerning developments in neuropsychology on the part of a range of workers in a variety of fields. Major advances in brain-imaging techniques and the cognitive modelling of the impairments following brain damage promise a wider understanding of the nature of the representation of cognition and behaviour in the damaged and undamaged brain.

Neuropsychology is now centrally important for those working with brain-damaged people, but the very rate of expansion in the area makes it difficult to keep up with findings from current research. The aim of the *Brain Damage, Behaviour and Cognition* series is to publish a wide range of books which present comprehensive and up-to-date overviews of current developments in specific areas of interest.

These books will be of particular interest to those working with the brain-damaged. It is the editors' intention that undergraduates, postgraduates, clinicians and researchers in psychology, speech pathology and medicine will find this series a useful source of information on important current developments. The authors and editors of the books in this series are experts in their respective fields, working at the forefront of contemporary research. They have produced texts which are accessible and scholarly. We thank them for their contribution and their hard work in fulfilling the aims of the series.

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Leicester and Ipswich
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Chapter 1

Acquired Childhood Aphasia: Neuropathology, Linguistic Characteristics and Prognosis

Anne E.Ozanne and Bruce E.Murdoch

Definition of Acquired Childhood Aphasia

Language disorders occurring in childhood are usually divided into developmental and acquired disorders. In general, the term 'developmental language disorder' is used to describe those language problems which are apparent from the initial stages of language development. In most cases, developmental language disorders in children have an idiopathic origin, although these disorders may also occur secondary to other conditions such as peripheral hearing loss, mental retardation, cerebral palsy, child autism, or environmental deprivation.

In contrast, acquired aphasia in childhood results from brain damage. A variety of definitions of acquired childhood aphasia have appeared in the literature. Some examples of these definitions are shown in Table 1.1. Despite the minor variations evident in these definitions, two major features of acquired childhood aphasia appear to be consistently identified. First, the onset of acquired childhood aphasia is precipitated by some type of cerebral insult. The cerebral insult, in turn, can result from a variety of causes, including head trauma, cerebrovascular accidents, brain tumours, infections and seizure disorders (Miller *et al.,* 1984). Second, most authors agree that acquired aphasia in childhood follows a period of normal language acquisition during which the child commences learning language normally, though some studies of acquired childhood aphasia have included subjects whose cerebral insult occurred before the development of language. In the present book, the demonstrable presence of a cerebral insult is taken as the major feature and discussion is included of subjects where the lesion occurred both before and after language development.

Table 1.1 Definitions of acquired childhood aphasia

Author	Year	Definition
McCarthy	1963	Childhood aphasia is language impairment occurring after language has been acquired in a normal manner
Alajouanine and Lhermitte	1965	Acquired aphasia in children is language disorganization resulting from focal cerebral lesions occurring during childhood
Hécaen	1976	Acquired aphasia in childhood refers only to disturbances of language due to cerebral lesions which have occurred after language acquisition
Carrow-Woolfolk and Lynch	1982	Acquired aphasia results from focal cerebral lesions occurring during childhood, as opposed to suspected injury occurring before or at birth
Miller *et al.*	1984	Children . . . whose normal language-learning progress is disturbed as a direct result of neurological impairment are . . . acquired aphasics

Historical Perspective

Although the first descriptions of acquired aphasia in children were reported in the late 1800s (Bernhardt, 1885; Cotard, 1868, cited in Guttmann, 1942; Freud, 1897), nearly 100 years later, little conclusive information is available to help clinicians in the management of the cases they may find in their caseloads. Little also is understood about the linguistic pathology from which to derive the most effective and efficient treatment strategies.

From the limited number of studies reported in the literature, a traditional description of childhood acquired aphasia has emerged. In the past it has often been stated that acquired childhood aphasia is rare, however, when it does occur it is characterized by an initial period of mutism followed by a non-fluent, motor type of language impairment with no accompanying comprehension deficit or other features of a fluent type of aphasia (e.g. jargon, logorrhea and paraphasia). In addition, acquired childhood aphasia was usually regarded as being transitory in nature, affected individuals making a good recovery. The stated rarity (Cotard, 1868; Denckla, 1979) was in part contributed to by the observation that children who do exhibit aphasia also showed rapid recovery of language skills. Bernhardt (1885) stated that childhood aphasia was not rare but because the aphasia was of a transient nature and rarely permanent it was often not reported. In support of Bernhardt (1885), Nadoreczny (1926, cited in Guttmann, 1942) also noted that most cases of acquired childhood aphasia recover within a few weeks.

The recovery of language in acquired childhood aphasia varies amongst children. Some children go through the normal language development process as they regain their language skills while others skip developmental stages during the recovery process (Basser, 1962; Byers and McLean, 1962). One 8-year-old girl with left hemiplegia, described by Byers and McLean (1962), initially communicated by gestures and within 3 weeks of becoming aphasic was responding verbally to simple sums. Soon after that, with her ability to write letters using her right hand, came the use of automatic speech including counting and reciting poetry. No description, however, was given of her functional language at this time. Within 3 months this child had returned to school, her only reported aphasic symptoms being a word-finding deficit.

Another characteristic of the traditional description of childhood acquired aphasia is a period of mutism immediately post-onset. This has been described as the chief characteristic of childhood aphasia by Branco-Lefèvre (1950) and Alajouanine and Lhermitte (1965). Guttmann (1942) noted an absence of spontaneous speech in 14 of his 16 cases of acquired childhood aphasia, mostly in children less than 10 years of age. Nine of the 15 right hemiplegic subjects (60 per cent) reported by Hécaen (1976) had a period of mutism lasting from 5 days to 3 months. Hécaen (1983) observed that mutism appeared mostly in children with anterior lesions (63 per cent) while only 10 per cent of children with temporal lesions showed mutism.

Some authors have suggested a psychological basis for the initial mutism shown by children with acquired aphasia (Alajouanine and Lhermitte, 1965; Byers and McLean, 1962). Reasons suggested for a psychological basis to the period of mutism include a presence of aspontaneity in written and gestural expression as well as in spoken language. In addition, when speech does return, increased incentives and encouragements are required to get the children to use words they are capable of producing. Furthermore, it has been noted that in general children tend to become silent and isolated at times of conflict or difficulties.

When speech and language do return, the traditional description of the aphasia is of a non-fluent, motor-type (Bernhardt, 1885; Carrow-Woolfolk and Lynch, 1982; Guttmann, 1942). Other features of acquired childhood aphasia reported in the literature include telegraphic speech (Guttmann, 1942) simplified syntax (Alajouanine and Lhermitte, 1965) and hesitation and dysarthria (Byers and McLean, 1962; Guttmann, 1942). Many investigators have noted that paraphasias, logorrhea and jargon are rare, or in the majority of cases, absent (Alajouanine and Lhermitte, 1965; Collignon *et al.,* 1968; Hécaen, 1976). Some authors (Alajouanine and Lhermitte, 1965; Guttmann, 1942; Poetzl, 1926), however, noted that the traditional description of acquired aphasia was most commonly seen in children less than 10 years of age while older children tended to present more like adult aphasics. Hécaen (1976) was unable to confirm this relationship between aphasic type and age, but did, however, postulate a relationship between lesion site and aphasic type. He found that children with acquired aphasia resulting from anterior lesions presented with an initial mutism while thosewith temporal lobe lesions

showed comprehension deficits in the acute stages that resolved within 1 year. Alajouanine and Lhermitte (1965) and Hécaen (1983) also noted transitory comprehension deficits in one-third of their subjects.

Another characteristic of acquired childhood aphasia noted to be present, particularly in the acute stages post-onset, is a naming deficit or poverty of lexical stock (Alajouanine and Lhermitte, 1965; Collignon *et al.,* 1968). Hécaen (1976) noted that naming disorders occurred in 7 of 15 of his left hemisphere-lesioned subjects. In three of these cases this deficit persisted long-term and was often noted in school reports.

Satz and Bullard-Bates (1981:405) carried out a comprehensive review of the literature to investigate the three aspects of the traditional description of acquired aphasia in children: its rarity, its clinical description and its rapid recovery. Overall, the findings of their review, which are described below, demonstrated a need to reevaluate the traditional description of acquired childhood aphasia. They took all subjects in published studies who met the following criteria:

1 Some speech reported before the lesion onset
2 Hand preference reported before lesion onset
3 Patient under 16 years of age before lesion onset
4 Evidence that lesion was unilateral only
5 Comparison of presence vs absence of aphasia following unilateral injury

Of 21 studies and 929 reported cases only 68 cases met the above criteria. In many studies handedness data was not provided. In other studies only cases with aphasia were included and therefore could not form part of a review on the incidence of acquired childhood aphasia.

In conclusion, Satz and Bullard-Bates (1981) stated that if the lesion is unilateral and encroaches on the speech areas then childhood aphasias are not rare. They noted, however, a lower prevalence of unilateral vascular disease in children as compared with adults. Furthermore, they concluded that if the left hemisphere is damaged the risk of language impairment is approximately the same in right-handed children as in adults. Regardless of age (at least after infancy), the risk of acquired aphasia is substantially greater following left-sided rather than right-sided lesions. Finally, it was concluded that, although rare, crossed aphasias do exist in left-handed children regardless of age. In particular crossed aphasias are especially rare after 3–5 years of age in right-handed children.

When reviewing the clinical type of aphasia Satz and Bullard-Bates (1981) concluded that the clinical pattern is predominantly non-fluent with rare or absent paraphasias and logorrhea, but disorders of auditory comprehension, naming and writing may co-exist. They stipulated that at that time it was unclear how those clinical patterns related to age and maturational effects or to the lesion, its size, cause, type, site or the time after the lesion.

The third aspect of acquired aphasia in children which Satz and Bullard-Bates(1981)

reviewed was the apparent rapid recovery of the aphasia. They concluded that: in the majority of cases spontaneous recovery is dramatic (i.e. approximately 75 per cent of cases), 25–50 per cent of cases still present with aphasia within 1 year post-onset, the recovery from aphasia is unrelated to the presence/severity of hemiparesis, and that despite recovery from aphasia, intellectual, cognitive or school achievement may still be impaired. Satz and Bullard-Bates (1981) could not identify the factors affecting recovery but suggested that these may include cause, aphasia type, lesion size or age at the time of the lesion.

Satz and Bullard-Bates (1981) did not believe that their findings supported the equipotentiality hypothesis put forward by Basser (1962) and Lenneberg (1967). These authors did state, however, that their findings were in line with studies which have noted a structural asymmetry of the brain at birth (Chi *et al*., 1977; Galaburda *et al.,* 1978) as well as those which have noted functional asymmetry in non-aphasics (Hiscock and Kinsbourne, 1978; Molfese *et al.,* 1975). Satz and Bullard-Bates (1981) noted that the results of their study, however, do not explain the dramatic recovery rate seen in children with acquired aphasia.

Since the early 1980s, therefore, it has been recognized that childhood acquired aphasia is not as rare as previously thought and despite the rapid recovery seen in some cases, 25–50 per cent of children who develop acquired aphasia still have aphasic symptoms 1 year post-onset. It is these children to whom we will now turn our attention. Several questions still need to be asked. What are the precise linguistic deficits with which children with acquired aphasia present? Do these linguistic deficits relate to specific causes? Do these deficits differ from linguistic impairments seen in children with developmental language impairment? What assessment and treatment strategies are available to clinicians working with these cases? These issues are addressed in the remainder of this chapter and in Chapter 2.

Neuropathological Substrate of Acquired Childhood Aphasia

Although acquired childhood aphasia can be caused by a similar range of disorders of the nervous system as adult aphasia, the relative importance of each of the different causes to the occurrence of language disturbances in children differs from the situation seen in adults. For instance, although in peacetime cerebrovascular accidents are the most common cause of aphasia in adults, the most common cause of acquired childhood aphasia is traumatic head injury.

Head Injury

In the majority of cases of acquired childhood aphasia reported in the literature thecause

is head injury. Head injuries can be divided into two major types: closed head injuries and open head injuries. In a closed head injury the covering of the brain remains intact even though the skull may be fractured. An open head injury differs from a closed head injury in that the brain or meninges are exposed. By far the majority of traumatic head injuries in both children and adults in civilian life are closed head injuries.

Damage to the brain following traumatic head injury may be either focal, multi-focal or diffuse in nature. In general, closed head injuries tend to produce more diffuse pathology while open head injuries are associated with more focal pathology. Brain contusions (bruises), lacerations and haemorrhages can be caused at the time of head injury from either the direct trauma at the site of impact on the skull, acceleration of the brain against the bony shelves of the skull (e.g. the sphenoidal ridge) or from contra-coup trauma that occurs when the brain strikes the skull on the side opposite the point of insult. Brain and Walton (1969) identified three different destructive forces which are applied to the brain at the moment of impact: compression or impression which forces the brain tissue together, tension which pulls the brain apart, and shearing produced by rotational acceleration and which develops primarily at those points where the brain impinges upon bony or ligamentous ridges within the cranial vault. According to Adams *et al.* (1977), the primary mechanism producing brain injury following closed head injury is diffuse axonal damage in the white matter occurring at the time of impact and caused by a shearing mechanism arising from rotational acceleration.

It has often been reported that children show a striking rate of recovery following closed head injury. Some authors have suggested that one of the reasons for the good prognosis for recovery in childhood is that the degree of brain damage following head injury is less in children than in adults, due in part to the different nature of their head injuries as well as to differences in the basic mechanisms of brain damage following head injury. Most childhood head injuries result from falls or low speed accidents. Consequently many paediatric head injuries are associated with a lesser degree of rotational acceleration and therefore, presumably, with a lesser amount of brain damage (Levin *et al.,* 1982). Jamison and Kaye (1974) noted that persistent neurological deficits were only present in children injured in road traffic accidents which are, by their nature, likely to yield greater diffuse brain injury. Likewise, a study conducted by Moyes (1980) showed road traffic accidents to be the most common cause of long-term morbidity following childhood head injury. In addition, Strich (1969) suggested that the shearing strains produced by rotational acceleration in head trauma are less pronounced in smaller brains.

Although the rate of spontaneous recovery in children following closed head injury is often described as excellent, persistent long-term language disorders have been reported (Gaidolfi and Vignolo, 1980; Jordan *et al.,* 1988; Satz and Bullard-Bates, 1981) and even when specific linguistic symptoms resolve cognitive and academic difficultiesoften

remain. The speech/language disorders associated with childhood head injuries together with the mechanisms of head injury are discussed in more detail in Chapter 3.

Cerebrovascular Disorders

Several investigators have documented the occurrence of acquired aphasia with vascular disorders in children (Aram *et al.,* 1983, 1986; Dennis, 1980). Cerebrovascular accidents or strokes are spontaneous interruptions to the blood supply to the brain, arising from either occlusion of the cerebral blood vessels (ischaemic stroke) or in some cases from rupture of one of the cerebral blood vessels (haemorrhagic stroke). Although cerebrovascular accidents are much less common in children than in adults, they occur more frequently than is commonly thought and are a significant cause of morbidity and mortality in the childhood population. Banker (1961) reported that of 555 childhood autopsy cases studied by him, death was due to a cerebrovascular accident in 48 (8.6 per cent).

Virtually all the diseases of blood vessels which affect adults may at some time also occur in children (Bickerstaff, 1972; Salam-Adams and Adams, 1988). Despite this, the causes of vascular diseases of the brain in children differ from those in adults. For instance, degenerative disorders such as atherosclerosis affect primarily the middle-aged and elderly and are rare in childhood (Moosy, 1959). Some vascular diseases of the brain, such as embolism arising from subacute or acute bacterial endocardial valvular disease, occur at all ages while others, such as vascular disorders associated with congenital heart disease, are peculiar to childhood.

Acute hemiplegia of childhood is a term used by many paediatricians and neurologists to describe the sudden onset of hemiplegia in children. A wide variety of vascular diseases of the brain, including both occlusive and haemorrhagic disorders, have been described under this heading.

Idiopathic childhood hemiplegia

The most commonly reported and dramatic syndrome resulting from an ischaemic stroke in childhood is idiopathic childhood hemiplegia. This syndrome involves the sudden onset of hemiplegia as a result of a unilateral brain infarct of unknown origin and can affect children from a few months of age up to 12 years of age (Bickerstaff, 1972). According to Bickerstaff (1972), females are affected more than males in a ratio of about 3:2.

The cause of idiopathic childhood hemiplegia has been argued for many years and a variety of possible causes proposed including: polioencephalitis (Strumpell, 1884), encephalitis (Adams *et al.,* 1949; Bernheim, 1956; Brandt, 1962), venous thrombosis (Bernheim, 1956; Branch, 1962; Norman, 1962), demyelination (Wyllie, 1948),

epilepsy (Norman, 1962), and occlusion of the internal carotid artery (Bickerstaff, 1964; Duffey *et al.,* 1957; Goldstein and Burgess, 1958). Although there appears to be some agreement that arterial occlusion is the most common cause of idiopathic childhood hemiplegia the reason for the occlusion is less certain. For reasons indicated above, atheroma cannot be implicated in childhood. Studies using carotid angiograms have demonstrated the presence of thrombosis of either the common or internal carotid arteries in some cases of idiopathic childhood hemiplegia (Salam-Adams and Adams, 1988). Bickerstaff (1964) suggested that roughening of the wall of the internal carotid artery as a result of arteritis secondary to throat, tonsillar or cervical gland infection might be the causal factor in some instances. Further, in some reported cases neither angiography nor post-mortem examination was able to demonstrate the presence of vascular lesions, suggesting that in these cases an embolus may have temporarily blocked a cerebral artery and then later broken up before the angiogram was taken or the post-mortem performed (Salam-Adams and Adams, 1988).

Other vascular occlusive disorders in childhood

A number of other vascular occlusive disorders peculiar to childhood can also cause ischaemic strokes in children. These disorders include: vascular disease associated with congenital heart disease, arteritis (inflammation of an artery) of various types, sickle cell anaemia, vascular occlusion associated with irradiation of the base of the brain, moyamoya, and strokes associated with homocystinuria and Fabry's angiokeratosis.

Ischaemic strokes associated with congenital heart disease occur most frequently in the first 2 years of life, corresponding to the stage when congenital heart disease has its greatest frequency (6 per 1000 live births) (Salam-Adams and Adams, 1988). Banker (1961) reported that of the childhood cerebrovascular accident cases examined by him, 28 per cent were associated with congenital heart disease, making it the single most common cause of cerebrovascular accidents in his study.

Various types of arteritis, including that associated with lupus erythematosus and occurring secondary to infections in the tonsillar fossa and lymph glands in the neck, have also been reported to cause ischaemic strokes in children (Bickerstaff, 1964; Davie and Cox, 1967; Salam-Adams and Adams, 1988). Lupus erythematosus is a diffuse inflammatory disease that involves the kidneys, skin, haematological system, the central nervous system and occasionally the liver. It is more common in females than males with a ratio of about 10:1. Although the average onset is around 30 years of age, symptoms can occur in the first decade of life (Bell and Lastimosa, 1980). Neurological complications have been reported to occur in up to 75 per cent of patients (Tindall, 1980), with seizures being the most common single neurological symptom. Hemiplegia secondary to cerebral arteritis, which is either transitory or permanent, occurs in approximately 5 per cent of patients with lupus erythematosus.

If permanent neurological loss occurs it is correlated with obstruction of one of the major extracranial or intracranial blood vessels. A case of lymphadenitis (inflammation of the lymph nodes) in the region of the carotid bifurcation and extending to involve the artery was described by Schnüriger (1966, cited in Bickerstaff, 1972). Bickerstaff (1972) reported damage to the carotid artery near its passage past the tonsillar fossa possibly resulting from arteritis due to a throat infection.

Both arterial and venous occlusions leading to cerebral infarction have been observed in children with sickle cell disease, an inherited blood disorder occurring primarily in Negroes (Salam-Adams and Adams, 1988). Likewise, cerebral infarcts have been reported subsequent to cobalt radiation of the base of the brain for treatment of a variety of neoplastic disorders in children, including craniopharyngiomas and pituitary adenomas.

Another vascular disorder of childhood that may cause vascular occlusion of the internal carotid artery is moyamoya disease. Patients with this condition present with headache, seizures, stroke-like episodes, visual symptoms and mental retardation as well as, in some cases, a movement disorder, a gait disturbance and/or a speech deficit. Typically the symptoms are bihemispheric. Moyamoya disease is characterized by the presence of a network of fine anastomotic blood vessels at the base of the brain called a rete mirabile. The aetiology of moyamoya disease is uncertain. There have been some suggestions that the condition is a congenital disorder involving retention of the embryonal rete mirabile. Alternatively, the network of anastomoses may be the consequence of an acquired disorder involving occlusion of the carotid arteries. It is possible, therefore, that several types of arterial disease in childhood may lead to moyamoya.

Complications of certain hereditary metabolic diseases may also occasionally cause occlusive vascular disease in children. Two such conditions where this may occur include homocystinuria and Fabry's disease. Both of these conditions result from enzyme deficiencies and both, amongst other effects, may cause structural damage to the blood vessels leading to thrombosis. Homocystinuria, resulting from a lack of cystathionine-synthetase, manifests as mental retardation. Ischaemic strokes arising from either arterial or venous thrombosis may be experienced by persons with this disorder in late childhood, adolescence or adult life. Likewise, Fabry's disease, a sex-linked disorder affecting males and resulting from a deficiency in galactosyl hydrolase, may also cause structural changes in the blood vessels leading to thrombosis and stroke (Adams and Lyon, 1982).

Brain Haemorrhage in Childhood

Spontaneous intracranial haemorrhage is much less common in children than in adults. Two major types of cerebral haemorrhage occur in childhood: one type

occurring secondary to haematological diseases such as leukaemia, sickle cell disease, haemophilia and thrombopenic purpura, the second type occurring secondary to vascular abnormalities such as arteriovenous malformations. It is noteworthy that haemorrhage resulting from rupture of saccular (berry) aneurysms is rare in childhood (Bickerstaff, 1972). In addition, the majority of arteriovenous malformations manifest as brain haemorrhage or in some other way during the third decade of life. Only approximately 10 per cent of arteriovenous malformations cause haemorrhage or other problems in childhood.

Details of the nature of the linguistic deficits associated with childhood vascular lesions are included in the discussion of the linguistic features of acquired childhood aphasia later in this chapter.

Tumours

Intracerebral tumours are a recognized cause of acquired aphasia in children (Alajouanine and Lhermitte, 1965; Brown, 1985; Carrow-Woolfolk and Lynch, 1982; Hécaen, 1976; Hudson *et al.,* 1989; Rekate *et al.,* 1985; Van Dongen *et al.,* 1985). Neoplasms of the posterior cranial fossa (i.e. those involving the cerebellum, pons, fourth ventricle and cisterna magna) occur more frequently in children than supratentorial tumours (Gjerris, 1978; Matson, 1956; Segall *et al.,* 1985). Cerebellar astrocytoma is the most common type of posterior fossa tumour in childhood, occurring most frequently in children between 5 and 9 years of age (Cuneo and Rand, 1952; Matson, 1969). Fortunately cerebellar astrocytomas are also highly curable and have an excellent prognosis following surgical removal. Next to cerebellar astrocytoma, medulloblastoma is the most frequent tumour type involving structures within the posterior cranial fossa in children. Generally these latter tumours involve children at a younger age than do cerebellar astrocytomas, their maximum incidence being within the age range of 3–7 years (Delong and Adams, 1975). Although initially cerebellar astrocytoma and medulloblastoma may present in a similar manner the prognosis of these two tumours types is quite different. While astrocytoma has a very favourable prognosis medulloblastoma has a poor prognosis for recovery. The third most frequently encountered tumour of the posterior cranial fossa is the ependymoma. These neoplasms grow from the floor of the fourth ventricle and affect children of a similar age to the medulloblastoma, and like the latter tumour type, ependymoma has a poor prognosis with a high proportion of tumours recurring after surgical removal.

Considering their location in the central nervous system (CNS), it would not be expected that posterior fossa tumours would cause language problems. A number of secondary effects, however, are associated with these tumours which can, in

some cases, lead to language deficits. Many posterior fossa tumours either originate from or invade the fourth ventricle. As a result hydrocephalus may occur following obstruction to the flow of cerebrospinal fluid (CSF). Subsequent compression of the cerebral cortex can then lead to malfunctioning of the central speech/language centres. In addition, radiotherapy (see Ch. 9) administered after surgical removal of posterior fossa tumours to prevent tumour spread or recurrence, has been implicated in the occurrence of language disorders in both adults and children (Broadbent *et al.,* 1981); Burns and Boyle, 1984; Duffner *et al.,* 1983; Hudson *et al.,* 1989; Meadows *et al.,* 1981).

The effects of intracranial tumours on language functions in children are discussed in further detail in Chapter 8.

Infections

Infectious disorders of the CNS are recognized as a significant cause of acquired childhood aphasia. Van Dongen *et al.* (1985) reported that of 27 children with acquired aphasia referred to their clinic over a 4-year period, 15 per cent had a history of infectious disease. Similarly Van Hout *et al.* (1985) noted that 38 per cent of their child cases with acquired aphasia had an infectious disorder.

Although the brain and its membranous coverings can be infected by the same range of microorganisms as other organs of the body, in the majority of cases of acquired childhood aphasia with infectious disease reported in the literature, the infectious disorder involved has been herpes simplex encephalitis. Two antigenic types of herpes simplex virus (HSV) are now recognized, type 1 (HSV-1) and type 2 (HSV-2). HSV-1 is responsible for almost all cases of sporadic encephalitis in children and adults. HSV-2, which causes congenital herpes, produces aseptic meningitis in the adult and encephalitis in the neonate.

HSV appears to involve primarily the basomedial portion of the frontal and temporal lobes and may present as an intracranial space-occupying lesion. As a result of the destructive nature of the lesions associated with infectious disorders, and in particular herpes simplex encephalitis, the language disorder associated with this disease tends to be more severe than in acquired aphasia resulting from other causes. For instance, Van Hout and Lyon (1986) attributed the severe language disorder they observed in a 10-year-old boy to the destructive bilateral damage to his temporal lobes caused by herpes simplex encephalitis.

Communication disorders resulting from infectious disorders in childhood are discussed in Chapter 4.

Convulsive Disorder

Acquired aphasia with convulsive disorder was first described by Landau and Kleffner (1957). In this condition the child's language deteriorates in association with the epileptiform discharges seen in their electroencephalogram. In some cases the language deterioration is either preceded, accompanied or followed by a series of convulsive seizures (Van de Sandt-Koenderman *et al.,* 1984). Although seizures do occur often, they are not the defining feature of this syndrome.

Onset of the Landau-Kleffner syndrome is usually between 2 and 13 years of age, with the initial loss of language function occurring most frequently between 3 and 7 years of age. Males are affected twice as often as females (Cooper and Ferry, 1978; Msall *et al.,* 1986). Although the cause of Landau-Kleffner syndrome is unknown, several hypotheses on the pathogenesis of this disorder have been proposed. Some authors have postulated that the language regression results from functional ablation of the primary cortical language areas by persistent electrical discharges (Landau and Kleffner, 1957; Sato and Dreifuss, 1973). The characteristics of the language disorder shown by children with Landau-Kleffner syndrome are discussed later in this chapter.

Linguistic Characteristics of Acquired Childhood Aphasia

The presence of methodological limitations in many studies have prevented the identification of definitive profiles of the linguistic characteristics of acquired childhood aphasia. These limitations, often resulting from restrictions on the number of subjects available, include factors such as: the inclusion of a diverse range of causes of acquired aphasia, the use of non-specific methods of lesion localization (e.g. the identification of brain lesions on the basis of the presence of a hemiplegia), the failure to exclude cases with possible bilateral brain damage (e.g. occurring after head trauma or infection), and the inclusion of subjects of different ages before and after the onset of brain damage.

Before the publication of the review by Satz and Bullard-Bates (1981) most reports of the speech and language characteristics of acquired childhood aphasia had been of a descriptive nature with very few standardized speech and language assessments being reported. In the recent studies more objective measures of speech and language performance have been used. In addition, more accurate methods of lesion localization such as computed tomography have also been utilized. Despite this, the problem of groups of mixed pathology and diverse age ranges still exist.

Auditory Comprehension Deficits

As stated earlier, the first reports of acquired childhood aphasia found no auditory comprehension deficits present in any subjects. More recent studies, however, have reported the presence of transitory comprehension deficits. For instance, Hécaen (1976, 1983) and Alajouanine and Lhermitte (1965) observed transitory comprehension problems in one-third of the children with acquired aphasia that they examined. Guttmann (1942) reported 'disturbed auditory comprehension' in two out of his 14 subjects with acquired childhood aphasia. In one of these cases, after a temporoparietal lesion, the comprehension deficit was the major symptom of the aphasia. Guttmann (1942) warned, however, that comprehension difficulties could be 'disregarded' unless expressive language deficits were also present.

Of the 32 children studied by Alajouanine and Lhermitte (1965) only ten were noted to have auditory comprehension deficits. These deficits were 'marked' in only four of the ten cases. In all these cases by 6 months after the lesion, comprehension had returned to 'near normal' with difficulties encountered only on the most difficult tasks. One year post-onset no auditory comprehension difficulties were noted in any of the ten subjects. Alajouanine and Lhermitte (1965) had divided their 32 subjects into two age groups. In the youngest age group (5–9 years old) nearly all (eight out of nine cases) had comprehension deficits in the initial stages, while in the second age group (10–15 years old) only two subjects of the 23 had auditory comprehension deficits. Comprehension deficits from this study, therefore, appear more common in children who acquired a brain lesion before 10 years of age.

Hécaen (1983) reviewed 26 children with acquired aphasia between 3½ and 15 years of age and found no support for the difference between the number of cases with comprehension difficulties in children above and below 10 years of age noted by Alajouanine and Lhermitte (1965). Although Hécaen observed that there were more cases of auditory comprehension deficits in children with left-sided lesions who were less than 10 years of age (i.e. six of 14 cases—42 per cent) than in children older than 10 years of age (six out of twenty cases—30 per cent) this difference was not statistically significant. Neither was there any significant difference in the number of cases with comprehension impairments in relation to sites of lesion (i.e. fronto-Rolandic or temporal) nor cause of lesion (i.e. haematoma, trauma or tumour/abscess) in the cases studied by Hécaen, although he noted a trend for more cases of comprehension impairment following an anterior lesion or a rapid onset.

The cause most commonly associated with auditory comprehension disorders in children with acquired aphasia is convulsive disorder (Landau and Kleffner, 1957; Rapin *et al.,* 1977; Miller *et al.,* 1984; Worster-Drought, 1971). The nature of these auditory comprehension deficits will be discussed later in this chapter.

Early studies reporting the nature of comprehension deficits in children with acquired aphasia usually used subjective and/or descriptive notes on the deficits often

without reference to the developmental level of competence expected in the child. Later studies, while using objective assessment procedures (usually versions of the Token Test), tended to cite unitary scores. Only in the most recent publications are the descriptive and standardized assessment methods combined in an effort to understand the nature of the auditory comprehension disorders. These early descriptions contained in case studies demonstrate the variable nature of auditory comprehension deficits seen in children with acquired aphasia. Oelschlaeger and Scarborough (1976:283) described a 10-year-old child who was 'unable to comprehend auditorily any speech stimuli' following a fall from a horse. After therapy, 1 year post-onset she still presented with significantly impaired receptive and expressive language skills but was able to answer questions on current activities appropriately.

Severe comprehension difficulties post-onset (e.g. inability to identify parts of the face) were also described by Pohl (1979) in a 6-year-old child after a suspected occlusion of the middle cerebral artery. Four months post-onset, comprehension had returned to normal while expressive language was at a one to two word level. Visch-Brink and Van de Sandt-Koenderman (1984) described four cases with differing degrees of comprehension impairment immediately post-onset. The first case scored 37 of a possible 61 on the Token Test (De Renzi and Vignolo, 1962) soon after a subdural empyema. This score improved to 44 correct, 6 weeks later and to 53 of 61 correct 5 months post-onset, at which stage the child's expressive language was characterized by mild syntactic difficulties and resolving paraphasias. The second case recovered comprehension and expressive skills within 14 days of a head trauma. Before recovery mild comprehension difficulties, telegraphic speech and paraphasias were evident. The other two cases presented with severe comprehension deficits. One of the two cases also had long runs of unintelligible speech and showed very slow recovery in both areas of language while the other presented with 'empty speech' characterized by strings of disconnected words then periods of adequate speech, paraphasias and paragrammatisms.

Formal assessment results in individual cases of children with auditory comprehension deficits as part of their aphasia, also emphasizes the variable nature of the deficits and the recovery process reported above in the descriptive studies. Van Hout and Lyon (1986) described a 10-year-old boy with a severe comprehension deficit as part of a Wernicke's aphasia following herpes-simplex encephalitis. In the early stages the child scored below five standard deviations from his age-matched test mean on the Gaddes and Crockett (1975) norms of the Spreen-Benton Neurosensory Centre Comprehensive Examination for Aphasia (NCCEA) (Spreen and Benton, 1969). Eight months later he was scoring at a 6-year age level for syntactic comprehension but could only identify an object by name 60 per cent of the time. Another 10-year-old child, described by Dennis (1980), presented on the Identification by Name and Sentence subtests of the NCCEA at below the 6-year age level 2 weeks after a cerebrovascular accident. Three months after the accident, Identification by Name was at an age-appropriate level but

Identification by Sentence was still at the original 6-year age level. Descriptive notes stated that 3 months after the cerebrovascular accident the subject understood single words and short commands. Test results indicated that comprehension and expressive skills were developing in parallel.

Two cases with subcortical lesions were reported by Aram *et al.* (1983). Only one of the cases had comprehension deficits. These deficits resolved within 2 months. Initially the subject (7 years old) had exhibited an 18 month delay on the Peabody Picture Vocabulary Test (Dunn, 1965) and a 2½ year delay on the North Western Syntax Screening Test (NWSST): Receptive (Lee, 1971). The authors suggested that the language deficit, including the moderate comprehension impairment noted in one subject but not in the other, was related to the site of the subcortical lesion.

Van Dongen *et al.* (1985), however, described differing degrees of comprehension impairment in three girls with similar lesion sites, all of which involved Wernicke's area. All three subjects presented with a fluent aphasia. The cause in two cases was trauma, and both of these showed rapid recovery. One of the head trauma subjects had normal conversational comprehension 13 days post-onset. The results of her Token Test, however, revealed auditory comprehension deficits as she scored 28 correct of a possible 61 on the test. By 2½ months post-onset, both conversational and test performance comprehension were within normal limits. A faster recovery period was noted in the second head trauma subject. Her Token Test results were 52 of 61 and her conversational comprehension was within normal limits 12 days post-onset. Initially she had presented with marked comprehension impairments both on the test results (i.e. 13 of 61 correct) and in conversation. She also presented as anomic in spontaneous speech. The third subject described by Van Dongen *et al.* (1985) had a suspected haematoma and 2 months later developed convulsions. Although before the onset of convulsions she scored 6 of 10 correct on the easiest part of the Token Test, once the convulsions had commenced this subject could not be assessed using formal test procedures, even though she could understand simple commands. Her expressive language at this stage was described as being comprised of simple words spoken in a telegraphic style. No improvement was noted over the next 2 years.

Studies, using either descriptive or standardized assessment procedures, therefore, have demonstrated a range of auditory comprehension deficits in children with acquired aphasia. These findings are contrary to those of the early reports which either suggested that comprehension disorders did not exist in cases of acquired childhood aphasia or they were of a transitory nature. More recent studies have compared groups of children with acquired aphasia either with a group of non-brain-injured controls or have compared groups of right and left hemisphere-lesioned subjects. Most of these latter studies have served to emphasize the persistent nature of auditory comprehension deficits found in some children with acquired aphasia.

Van Hout *et al.* (1985) studied 16 children with acquired aphasia between 2 and 13 years of age. They reported that the three youngest subjects (all 2 years old) presented

according to the classical description of childhood aphasia, while the two oldest subjects (both 13 years old) presented with a Broca's aphasia. The remaining 11 subjects did not fit either pattern. They divided these 11 subjects into three groups based on their recovery from paraphasia. The first two groups whose paraphasias resolved either within days or months presented with moderate comprehension deficits, while the third group who had paraphasias lasting more than 1 year post-onset, presented with severe comprehension impairments. The authors noted that the most common cause in this last group was infection. Decreased receptive language skills or global aphasia were also described as the initial communication deficits in the four children with infections described by Cooper and Flowers (1987). Initial comprehension deficits, however, were also noted in their subjects with anoxia, closed head injury and tumours.

The assessment of Cooper and Flowers' (1987) 15 brain-lesioned children (between 10 and 18 years of age) was carried out 3 to 12 years post-onset. When compared with a control group without brain injury the experimental group performed significantly poorer on all assessments of language comprehension. Despite performing significantly poorer on the assessments than a group of controls, Cooper and Flowers (1987) emphasized the heterogeneity of the group pointing out that one subject showed no impairment on any language test while three subjects showed impairment on all expressive as well as receptive tests. For this reason they gave individual assessment profiles for all 15 brain-injured subjects. Nine subjects scored greater than two standard deviations below the age mean on the Peabody Picture Vocabulary Test-Revised (Dunn and Dunn, 1981). Of these nine subjects two subjects scored more than four standard deviations below the mean while another three subjects scored greater than seven deviations below. On the shortened version of the Token Test (De Renzi and Faglioni, 1978) seven of the 15 subjects scored greater than two standard deviations below the mean; however, Cooper and Flowers (1987) claimed that only one of these could be described as having a moderate comprehension deficit. As a group, the brain-lesioned subjects did not perform significantly different from the controls on Parts 1–5 of the test; however, there was a statistically significant difference between the scores of the experimental and control groups on Part 6 of the test. This would indicate that as a group, the brain-lesioned subjects had no more difficulty than the control group with the increase in syntactical information load but had more difficulty with increased linguistic complexity.

The proposed difficulties with syntactical complexity is supported by the individual case profiles published by Cooper and Flowers (1987) which showed that their brain-injured subjects had most difficulty with the items containing the instructions 'in addition to, if, between' and the item 'touch the blue circle *with* the red square'. Only one subject had difficulty with the earlier parts of the test. Six of the 15 subjects reported by Cooper and Flowers (1987) had difficulties with the Processing Spoken Paragraphs subtest of the Clinical Evaluation of Language Function (CELF) (Semel

and Wiig, 1980). In all cases the authors noted that the subjects appeared to comprehend the questions asked but were unable to recall the appropriate information. Overall three subjects presented with deficits in all three areas of comprehension assessed; two had problems in lexical and auditory language comprehension; two had problems in lexical and comprehension of contextual language while four subjects had one area only of deficit, two in lexical comprehension and one each in auditory language and comprehension of contextual language. No relationships between these results and the side, site, size or cause of the lesion nor of the age at injury or after the lesion could be drawn as the subjects differed too widely on all these variables. The study, however, showed conclusively that after a mean length of time post-onset of 8 years, residual comprehension deficits were still shown by all but three of Cooper and Flowers (1987) 15 subjects.

Another series of studies have investigated the influence of the side of lesion on comprehension abilities in children with acquired aphasia. Most of these studies have included children who received their brain lesions perinatally or within a few months of birth. Therefore any deficits found when brain-lesioned children are compared with a control group would support the notion of persisting deficits of auditory comprehension.

Twenty-eight children with left hemisphere lesions and 25 with right hemisphere lesions were assessed using the Token Test 2 years post-onset (Vargha-Khadem *et al.,* 1985). A significant difference was found between the side of the lesion, with the children who had left hemisphere lesions performing more poorly than those with right-sided lesions. The Token Test scores did not correlate with the WISC-R digit span subtest score, leading the authors to conclude that this was a linguistic not a memory deficit. The scores of the children with right hemisphere lesions did not differ from the control group. Vargha-Khadem *et al.* (1985) also divided the side of lesion groups into three subgroups based on age at the time of the lesions, i.e. prenatal, early postnatal (2 months to 5 years) and late postnatal (5–14 years). There was a trend that the later the injury occurred the greater was the impairment of auditory comprehension.

Significant differences between the auditory comprehension skills of children with right and left hemisphere lesions were also found by Rankin *et al.* (1981). Lexical comprehension measured using the PPVT showed that right hemiplegics performed more poorly than left hemiplegics. This pattern of performance was repeated on the measures of syntactic comprehension using the NWSST: Receptive (Lee, 1971) and the Token Test, i.e. the right hemiplegics performing less well than the left hemiplegics. A closer examination of the components of the Token Test showed that the left hemiplegics showed a progressive decline in correct scores across the five parts of the test while the right hemiplegics showed no errors in Part 1 of the Token Test but their performance deteriorated across Parts 2–4 with the increase in information load. The right hemiplegics, however, improved their performance on Part 5 which

showed they did not have difficulty with syntactic complexity *per se*. This is in contrast to the findings of Cooper and Flowers (1987).

Aram *et al.* (1985) compared the comprehension skills of children with right and left unilateral brain lesions. Each group of brain-lesioned children was matched to an appropriate control group. Some children had acquired their brain lesions in the first few months of life while others were 18 months to 6 years of age at the time of the lesion. On the PPVT all left-lesioned children scored within the normal range but five of eight scored lower than their control subjects. Similarly, the right-lesioned subjects performed more poorly than their controls. As a group, the left-lesioned subjects performed higher than the right-lesioned subjects. On the NWSST: Receptive the left-lesioned subjects performed less well than their controls while there was no difference between the right-lesioned group and their controls.

In another study using the Revised Token Test (McNeil and Prescott, 1978), Aram and Ekelman (1987) assessed the effect of unilateral brain lesions on auditory comprehension skills. In addition they also assessed the effect of the site of lesion and the age at lesion onset on those skills. Seventeen children with left lesions and 11 children with right-sided unilateral lesions between 6 and 17 years of age were assessed. Significant differences between the right-and left-lesioned groups and their respective controls were found for all ten subtests of the revised Token Test. As a whole the left-lesioned group performed similarly to their controls on subtests one to three and subtest nine. In subtest ten the left-lesion group appeared to use a memory strategy to enable them to remember the instructions. Aram and Ekelman (1987) concluded that the left-lesioned group presented with memory difficulties which affected their performance as the information load increased. The right-lesioned group, however, made more errors than their controls but there was no difference in the number of errors with the increase in information or linguistic loading. The authors suggested that this reflected the more impulsive nature of the right-lesioned group. In relation to site of lesion, there was a trend for more errors on the Token Test in the children with left retro-Rolandic rather than pre-Rolandic lesions. No difference in performance was noted in the site of the right hemisphere lesions or the presence or absence of subcortical lesions. In general, the earlier the left hemisphere lesions occurred (i.e. less than 1 year of age) the poorer the performance on the Revised Token Test. The reverse was true, however, for right hemisphere lesions. The results for the left hemisphere lesions were in contrast to the results of Vargha-Khadem *et al.* (1985) who found greater deficits the older the child was at the time of the lesion.

In conclusion, auditory comprehension deficits do exist in children with acquired aphasia, particularly when the brain-lesioned subjects are compared with a group of matched controls. These deficits, while of a subtle nature, occur even when the lesions are acquired at an early age. The extent of the comprehension impairment is variable, as is its recovery. The differences found between right and left hemisphere-lesioned subjects would suggest that side of lesion may influence the level and type of

impairment. Further research into the aetiology of acquired aphasia is required to determine the influence of cause, size and site of lesion and time post-onset. Certainly, the evidence to date does not support the historical description of auditory comprehension deficits being rare and only of a transitory nature in children with acquired aphasia.

Production Deficits

Syntax

In the traditional description of acquired childhood aphasia, as mentioned previously, the aphasia was described as being of a non-fluent type with simplified syntax (Alajouanine and Lhermitte, 1965) or telegraphic speech (Guttmann, 1942), Alajouanine and Lhermitte (1965) noted that children with acquired aphasia tend to use simplified syntax rather than producing syntactical errors *per se.* Consequently, they stated that the syntax used by children with acquired aphasia does not resemble the agrammatism seen in adults. Despite the description of simplified syntax from the earliest reports (Bernhardht, 1885) few studies have given detailed descriptions of the syntax used by children with acquired aphasia.

Cooper and Flowers (1987) examined 15 brain-injured children who had acquired neurological damage between 2 and 12 years of age and were at least 1 year post-onset at the time of the study. As a group these brain-injured children scored significantly poorer than a matched control group on the Producing Formulated Sentences subtest of the CELF (Semel and Wiig, 1980). Cooper and Flowers (1987), however, observed a wide variety of language skills within the brain-injured group. Of the 15 subjects, only one achieved a score greater than two standard deviations below the test mean. By examining the case descriptions supplied by Cooper and Flowers (1987) it appears that most syntactical errors were made on the items which required complex sentence structures (i.e. 'because', 'if', 'herself'). Cooper and Flowers (1987) also provided examples of syntactically correct complex sentences produced by these same subjects. These correct sentences, however, tended to have a more stereotypic quality (e.g. 'If I tell you, do you promise to keep it a secret? I'll tell my parents about what has gone on at school. I will tell her I'm hungry'). Some acquired aphasia subjects produced agrammatical sentences (e.g. 'Himself and herself is a boy or is a gal'.) while others produced simple or incomplete sentences (e.g. 'Because today is rainy and wet') perhaps suggesting a semantic rather than syntactic impairment.

A series of studies by Aram and co-workers are the only ones reported which have systematically looked at productive syntactic skills in children with unilateral brain lesions. It should be noted, however, that most of Aram's subjects acquired brain lesions early in life, before they began talking. It was claimed by Aram and her co-

workers that children who suffer left hemisphere lesions shortly after birth are at risk for syntactical impairment. Their reasons for this claim was the observation of significant differences between the syntactical abilities of appropriately matched controls and children with left hemisphere lesions and between children with either left or right hemisphere lesions.

Rankin *et al.* (1981) found three right hemiplegic children to be markedly below three left hemiplegic children on the NWSST. A later study by Aram *et al.* (1985) comparing eight children with right hemisphere lesions and eight with left hemisphere lesions and two appropriate control groups showed that the children with right hemisphere lesions did not differ from their controls on any syntactic measure except for mean length of utterance. The children with left hemisphere lesions, however, differed from their controls on all measures of syntactical skill including the NWSST and measures based on a spontaneous language sample (e.g. Mean Length of Utterance (MLU) and a Developmental Sentence Score (Lee, 1974)). Examination of individual scores of the children with left hemisphere lesions showed that no subject in this group scored higher than the 18th percentile on the NWSST: Expressive.

An extension of this study by Aram *et al.* (1986) undertook more detailed analyses of the spontaneous language samples by the eight left-and eight right-lesioned subjects. These analyses showed that the left hemisphere-lesioned subjects performed more poorly than their controls on most measures of simple and complex sentences while the right-lesioned group were similar to their controls in most measures of syntactical ability. The two lesioned groups differed from each other on measures of MLU, mean number of interrogative reversals and 'wh'-questions, the percentage of complex sentences attempted, the percentage of complex sentences correct, the number of embedded sentences attempted and the percentage of embedded sentences correct. The right-lesioned subjects differed from their controls on MLU, percentage of all sentences correct, the percentage of simple sentences correct, the total number of main verbs used, the mean number of negatives used and the grammatical markers in error (GME). The only syntactical measures on which the left-lesioned subjects did not differ from their controls were the total number of negatives used, and the use of pronouns or conjunctions. In addition it was noted that while the left-lesioned group performed more poorly than their controls on most measures of syntax, they did use more simple sentences reflecting their reduced use of complex sentences. Aram *et al.* (1986) suggested that these findings are indicative of a developmental immaturity on the part of the left-lesioned subjects. At the same time the right-lesion group produced a small percentage of their simple sentences correctly when compared with their controls, even though they attempted the same number of simple sentences. Both of these counts were different between the left-lesion group and their controls. The obviously poorer performance on the left-lesioned subjects when compared with their controls and when compared with the right-lesioned group and their controls

is indicative of a susceptibility for expressive syntactic impairments in children with unilateral left hemisphere lesions.

Semantics

Two outstanding aspects of expressive semantics are commonly noted in the classical description of acquired childhood aphasia. The first of these is the presence and often persistence of naming disorders while the second is the absence or rarity of paraphasias, jargon and logorrhea. As Carrow-Woolfolk and Lynch (1982:334) state 'once speech re-emerges children may have name-finding difficulties but they do not display the paraphasic or misnaming symptoms characteristic of adults'. (Receptive semantics has been discussed under comprehension.)

Naming disorders in children with acquired aphasia have been described by Hécaen (1983) as frequent and persistent, and are often noted in school reports. He found 15 of his 34 subjects with acquired aphasia (i.e. 44 per cent), between 3½ and 15 years of age, to have naming disorders. No significant difference was found in the number of subjects with naming disorders when examined for site or cause of lesion or for the age of the subject. In an earlier study of 15 subjects, Hécaen (1976) found naming disorders in seven cases, three of which showed persistent problems.

Cooper and Flowers (1987) reported a significant difference between the scores achieved by children with acquired aphasia (between 10 and 18 years of age) and those achieved by a non-brain-damaged group on the Boston Naming Test (Kaplan *et al.,* 1976), including latency of response, and the Producing Word Associations subtest of the CELF. As their subjects were assessed between 3 and 12 years post-onset their findings support the fact that naming difficulties can be of a persistent nature in children with acquired aphasia.

Van Dongen and Visch-Brink (1988) also reported severe naming disorders in their six left hemisphere-lesioned children with acquired aphasia in the initial stages. The recovery from the naming disorder, however, differed between a head-injured group and a non-head-injured group. In the head-injured group recovery was complete within 6 months while the non-head-injured group could not complete a naming test 1 month post-onset and recovery was still not complete 1 year post-onset. Meanwhile six subjects with a right hemisphere lesion had scores on the Boston Naming Test within normal limits immediately post-onset. The complete recovery of naming abilities in the head-injured population is in contrast to the findings of Jordan *et al.* (1988, 1990) (see Ch.3).

Differences in naming abilities between subjects with right and left hemisphere lesions and control groups have been investigated by several other authors. Also using the Boston Naming Test, Kiessling, *et al.* (1983) found that the mean scores for their right and left brain-lesioned children were lower than those for a sibling control

group. This difference, however, was not statistically significant, neither was the difference between the Boston Naming Test scores achieved by children with right compared with left brain lesions. Two studies using the Oldfield and Wingfield Object Naming Test (Oldfield and Wingfield, 1964) did, however, find significant differences between controls and brain-lesioned children. Woods and Carey (1979) reported lower scores for children sustaining a left-sided brain lesion after 1 year of age when compared with a non-lesioned control group. Although a difference between children sustaining a brain lesion before 1 year of age, when compared with controls, was not found by Woods and Carey (1979), such a difference was found by Vargha-Khadem *et al.* (1985) who also used the Oldfield and Wingfield Naming Test. This difference was evident in Vargha-Khadem *et al.* (1985) prenatal, early postnatal (i.e. the lesion occurring between 2 months and 5 years of age) and late postnatal (i.e. 5–14 years of age) left hemisphere-lesioned groups as well as the early postnatal right hemisphere-lesioned group. The results of their study also indicated that the naming disorders were more marked the later the age at which the lesion occurred.

Rankin *et al.* (1981) compared the language abilities of three right and three left hemiplegic children between 6 and 8 years of age and found no differences between the two groups on the Naming Fluency subtest of the Boston Diagnostic Aphasia Examination (Goodglass and Kaplan, 1972). Two studies by Aram and co-workers, however, found slightly different results. Using the Expressive One-Word Picture Vocabulary Test (EOWPVT) (Gardner, 1979), Aram *et al.* (1985) found that both right and left hemisphere-lesioned children between 18 months and 8 years of age performed more poorly than their appropriate controls; however, this difference was only statistically significant for the left-lesioned subjects. Aram *et al.* (1985) noted the wide variation in individual scores in both the lesioned groups. A greater number of subjects with unilateral brain lesions (n=32) between 6 and 17 years of age were assessed by Aram *et al.* (1987) to try to clarify the results of the previous studies. Two measures of lexical retrieval were used: the Word Finding Test (Wiegel-Crump and Dennis, 1984) and the Rapid Automatized Naming Test (RAN) (Denckla and Rudel, 1976). The results indicated that children with left hemisphere lesions required a longer latency to respond than their controls. The type of lexical access on the Word Finding Test affected the results. For example, rhyming cues produced more errors and longer latencies than semantic or visual cues, however, this was also the general pattern seen in children developing normally and the control groups used in the study. The left hemisphere-lesioned group were also slower to respond for the RAN, having greater difficulty naming objects and colours than letters and numbers. The right hemisphere-lesioned subjects, on the other hand, produced more errors than their controls but did not require a longer latency. In fact the right-lesioned group responded faster than their controls on all access conditions of the Word Finding Test. Aram *et al.* (1987) suggested that a speed-accuracy trade-off occurred with the right-lesioned group. In addition, the right hemisphere-lesioned subjects had more errors

that could be attributed to visual processing difficulties. These results lead Aram *et al.* (1987) to postulate that left hemisphere-lesioned subjects have lexical retrieval problems while the errors seen in the right-lesioned group could be attributed to impulsivity or visual processing difficulties. Aram *et al.* (1987) also looked at the effects of lesion site and age at time of lesion on naming disorders and concluded that various sites of lesions in the left hemisphere can produce word access problems and that there was no clear relationship between degree of lexical retrieval impairment and age at lesion onset.

The second issue relating to expressive semantics arising out of the classical descriptions of acquired childhood aphasia is the absence or rarity of logorrhea, jargon and paraphasias. Generally this stems from the notion that the aphasia seen in children is of a non-fluent motor type. Several authors, however, have documented fluent aphasias in children (Van Dongen *et al.,* 1985; Van Hout *et al.,* 1985; Van Hout and Lyon, 1986; Visch-Brink and Van de Sandt-Koenderman, 1984). Initially it was felt that these fluent aphasias were only shown by children who had received their brain injury after 10 years of age (Alajouanine and Lhermitte, 1965; Guttmann, 1942).

Visch-Brink and Van de Sandt-Koenderman (1984) disputed the term 'rare' being applied to paraphasias shown by children with acquired aphasia. They approached the problem of rarity from two perspectives; first that paraphasias are not observed in many subjects with acquired childhood aphasia, and second that individual cases of children with acquired aphasia who do show paraphasias produce only a small number of these errors. Both uses of the term 'rare' did not apply to children with acquired aphasia they claimed. Visch-Brink and Van de Sandt-Koenderman (1984) pointed out that despite Guttmann's (1942) claim that paraphasias do not exist, two cases described in his article did show paraphasia. They also noted that Hécaen (1983), who had also described paraphasias as rare, had identified three children using paraphasias. In all cases, Hécaen claims the paraphasias resolved. The three children who did show paraphasias represented 8 per cent of the subjects with left hemisphere lesions. (It should be noted that the other aphasic symptoms described by Hécaen were present in 35–63 per cent of subjects.) There was no difference in the number of subjects showing paraphasias based on lesion site or cause, however, the paraphasias were only shown by children less than 10 years of age. Seven subjects showing phonemic paraphasias were present in Alajouanine and Lhermitte's (1965) study of 32 subjects. In contrast to Hécaen's group all these latter subjects were older than 10 years of age. No examples of logorrhea, verbal stereotypes or perseverations were noted in any subjects. These earlier reports of low incidence of paraphasia are in marked contrast to the findings of Van Hout *et al.* (1985) which showed positive semiology in all 11 subjects studied by them. (Five subjects had been previously excluded as they presented with non-fluent type of aphasia.) All 11 subjects examined by Van Hout *et al.* (1985) between 4 and 10 years of age showed verbal or phonemic paraphasias. In addition, perseverations and stereotypes were also frequently produced.

The types of paraphasic errors seen in children with acquired aphasia have been documented by several authors. For instance Cooper and Flowers (1987) noted the types of errors made on the Boston Naming Test by their subjects. Eight of 15 of their subjects scored more than two standard deviations below the mean on the test. Four of these subjects made semantically related, phonological or descriptive errors, one of them making comments about his difficulty in retrieving the words. Two other subjects made only semantically-related errors while another made phonological and descriptive errors. One subject with a left hemiplegia after anoxic encephalopathy also, at times, misinterpreted the picture (e.g. bat/paddle). As all these subjects were at least 2 years post-onset these errors would indicate persistent paraphasias. Other authors have also noted long-term paraphasic errors. In fact Van Hout *et al.* (1985) divided their 16 subjects between 2 and 13 years of age into three groups based on the rate of recovery of their paraphasias. In the first group recovery from paraphasias was noted within days. Initially the incidence of paraphasias was low (i.e. 3 per cent semantic paraphasias and 22 per cent phonemic). The resolution of the two types of paraphasias in the first group was either synchronous or the semantic ones disappeared before the phonemic. The second group whose paraphasias resolved within months of the brain lesion differed from the first group in that the two kinds of paraphasias did not resolve simultaneously, the semantic paraphasias always resolving first. Also this group was slightly older in age. The third group, whose paraphasias persisted, had a higher incidence of paraphasias (40 per cent) and the phonemic errors resolved before the semantic. The duration of the coma for this group also appeared longer and their comprehension skills were more severely affected.

Van Hout *et al.* (1985) also noted perseveration and stereotypes in their subjects with acquired aphasia. They further felt that the semiology of some of the child cases and the anatomical correlations appeared to be similar to those observed in adults. The major differences between child and adult cases were: first the rapid rate of recovery in most child cases, and second the development of negative symptoms (e.g. lack of spontaneity and the presence of naming difficulties which became more apparent as the positive signs of paraphasias decreased). Van Dongen and Visch-Brink (1988) also charted the resolution of the paraphasic errors in their head-injured and non-head-injured groups of aphasic children. For the head-injured group neologisms were always present on the Boston Naming Test in the acute stages. The neologisms decreased with every succeeding examination (up to approximately 18 days post-onset). This decline, however, was not evident for the literal or verbal paraphasias, although all errors had disappeared by 6 months post-onset. In contrast, for the non-head-injured group, one case never used neologisms and the distribution of neologisms and paraphasias were irregular over time. This low and irregular occurrence of paraphasias was despite a more severe form of aphasia than seen in the head-injured group.

Rapid recovery of paraphasias was also reported in a 9-year-old girl after a stroke (Dennis, 1980). At 2 weeks post-onset this subject showed phonemic and semantic

paraphasias and some random misnamings on visual and tactile naming tasks placing her at an age equivalency of 6 years on the NCCEA (Gaddes and Crockett, 1975). Three months after the stroke, naming difficulties had resolved to a mild level of impairment. Similarly, encephalitis in a 10-year-old produced a Wernicke's aphasia (Van Hout and Lyon, 1986) with speech limited to a few stereotyped utterances 2 weeks post-onset. Three weeks after emergence from coma, 15 per cent of this latter subject's spontaneous speech consisted of neologisms. This rose to 45 per cent 10 days later. Meanwhile, verbal paraphasias were evident in 20–30 per cent and phonemic paraphasias in 3 per cent of his utterances. Van Hout and Lyon (1986) suggested that recovery, which showed an increase in semantic paraphasias and then the development of circumlocutory speech, was consistent with that described in cases of adult sensory aphasia (Buckingham and Kertesz, 1974).

There is considerable evidence, therefore, to suggest that naming difficulties do persist in children with acquired aphasia. Examples of fluent aphasia containing neologisms and paraphasias are also more common than first thought (e.g. paraphasias were observed in all of Van Hout *et al.*'s (1985) 11 subjects). The recovery from this fluent type of aphasia, however, can vary. It may resolve totally, paraphasias may persist, or as the paraphasias decrease there may be an increase in aspontaneity of speech and word-retrieval problems.

Many different reasons have been postulated, especially by Van Hout *et al.* (1985), for the inconsistency between the incidence of paraphasias in recent studies when compared with the traditional descriptions of acquired aphasia in childhood. One reason relates to the time after the lesion as paraphasias often resolve quickly (i.e. within days) and if the child is not assessed during that period paraphasias will not be noted. Second, since earlier reports based the presence of a brain lesion in children on the presence of a hemiplegia, an unusually high number of children with anterior lesions could have been described. Third, the method of assessment may affect the identification of paraphasias. If brain-injured children are reluctant to speak then paraphasic errors may only be elicited on formal assessments such as confrontation naming.

Pragmatics

To date, no study has systematically assessed the pragmatic skills of children with acquired aphasia. The reason for the lack of research in this area is partly historical in that pragmatics is an area of language that has only recently been recognized as an important area of research. A suggestion of the presence of pragmatic problems, however, can be gleaned from earlier writings which suggested that soon after the onset of aphasia or in connection with the mute phase, children with acquired aphasia are reluctant to communicate (see Historical Perspective). For instance, Cooper and Flowers (1987) noted that one of