APHASIA, GRAMMAR AND LANGUAGE: A HISTORICAL PERSPECTIVE

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The present review highlights various aspects of investigations of aphasia. In its historical portion it focuses on the main developments until the middle of the 20th century (major topics: localization vs. holism; Gesner, Gall, Broca, Wernicke, Lichtheim, Jackson, Marie, Head, Pick, von Monakow, Goldstein). Its contemporary aspects include linguistic and neuropsychological investigations of aphasia (esp. phonological, semantic, syntactic, pragmatic disorders). Finally, basic aspects of aphasia rehabilitation are discussed.

Keywords: *history of aphasia, phonological disorders, semantic disorders, syntactic disorders, pragmatic disorders, aphasia syndromes, aphasia rehabilitation*

1. APHASIA

Aphasia is usually defined as a language disorder following brain damage. Based on the ICIDH classification of 1980 aphasia can be conceptualized in the following way (Fig. 1).

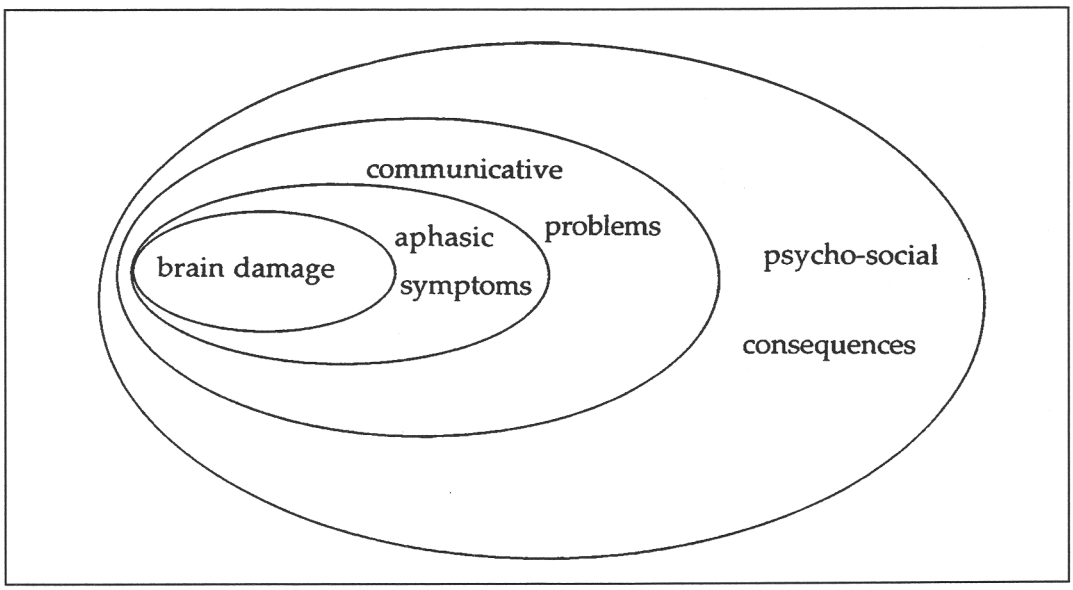


FIGURE 1. Dimensions of aphasia.

Following some sort of sudden brain damage (typically strokes or head trauma), aphasic symptoms (word finding difficulties, ungrammatical utterances, writing problems, etc.) occur, and each patient has a more or less unique pattern of symptoms at various linguistic levels and in different modalities. These symptoms usually lead to communicative problems, i.e. aphasic persons are not able to express themselves using language and are also not able to understand what is said to them. Given the central role of language for humans in every-day activities and professional life, the psycho-social consequences of aphasia are enormous. Aphasic persons lose their jobs, they become socially isolated, and they are frequently victims to depression.

Given this situation, it is clear that several scientific disciplines are necessary to investigate and understand the phenomenon of aphasia and to generate appropriate methods and approaches to language rehabilitation.

Even though we generally have a working definition of aphasia (i.e. *neurogenic language* disorder) that is understood and used across disciplines, the field of aphasiology is full of controversy. Thus Benson and Ardila (1996, 3) correctly state following: *"Aphasia is the loss or impairment of language function by brain damage. The appearance of fundamental agreement is illusory, however. Aphasia was born of controversy, has a history of ongoing disagreements about appropriate approaches and remains a contentious topic."*

At least the following aspects are often subject to debate: the underlying cause, age of onset, localization, the relation between language, communication and cognition, the question of syndromes, the question of supramodality vs. unimodality, the modelling of symptoms (see Tesak, 1997, for a summary). In this article, I will briefly outline three discussion points: (i) What causes aphasia?, (ii) Can aphasia be localized?, and (iii) Are there aphasic syndromes?

The most common causes for aphasia are strokes and traumatic head injuries. Both causes have sudden onsets, and for many colleagues 'sudden onset' is a defining criterion for true aphasia. However, also brain tumors and brain infections may cause aphasia. The question becomes even more complicated when one takes into account that degenerative brain diseases like senile dementia of the Alzheimer type may also cause language problems (see discussion in Au et al., 1988). There is some agreement to exclude degenerative brain diseases as underlying causes for 'true' aphasia despite the fact that "aphasic" symptoms do occur in these patients. Since chronic aphasia is not usually becoming worse, the term progressive aphasia has been introduced for 'aphasia' of degenerative origin (see Kirshner, 1995c, for a review). Some authors (e.g. Caplan, 1992) even use the term aphasia completely disregarding the underlying cause of the language disorder.

In relation to localization, there is some agreement and hard data exist (e.g. Lecours, Lhermitte & Bryans, 1984; Russel & Espir, 1961) that aphasia follows brain lesions of the left hemisphere, especially in the perisylvian cortex. However, to what extent various symptoms and combinations of symptoms (so-called syndromes) are systematically caused by lesions to specific regions of the left hemisphere is quite unclear. There are different reasons for this situation. First, there are various exceptions. Also right-hemispheric lesions (in right-handed persons) may lead to aphasia (see Coppens & Robey, 1992), and lefthanders, illiterates, bi- and multilinguals, speakers of tone languages, writers of non-alphabetic writings systems notoriously do not fit the general picture (see Lecours et al., 1985, for some

provocative and stimulating statements). Second, studies with CT-scans have clearly shown that correlations between symptoms (or syndromes) with lesion sites in the human neo-cortex are subject to great variation (e.g. Caplan & Vanier, 1990; de Bleser, 1988). Third, modern brain imaging techniques like PET and SPECT show that language processing cannot be restricted to the so-called cortical language centers. One of the leading PET scientists, J. Metter (1995, 206–7) summarizes the situation as follows: "The studies reviewed suggest that language requires the interaction of a number of highly integrated systems of the brain. This interaction involves both hemispheres as well as cortical and subcortical structures." Generally, the idea of subcortical involvement in language processing led to the concept of "sub-cortical aphasia" (see Cappa & Abutalebi, 1999; and Fabbro, 1997, for recent discussions) which in itself casts doubt on the original concept of aphasia as a purely "cortical" dysfunction.

The relation of language, communication, and cognition in aphasia is less clear than it may seem. Whereas standard teaching would claim that the primary problem in aphasia lies in language processing, that consequently communicative problems are secondary in nature, and that aphasia per se leaves cognition untouched (e.g. Goodglass, 1993; Wallesch & Kertesz, 1993; Huber et al., 1983), each of this aspects is under discussion. Within a therapeutic perspective it is well understandable that aphasia is conceptualized as a disorder of communication rather than a problem with linguistic rules and structures (e.g. Carlomagno, 1994). Others broaden the definition of aphasia by incorporating cognitive aspects, e.g. Chapey (1994c) writes: "[A]dult aphasia is defined as an acquired impairment in language and the cognitive processes that underlie language caused by organic damage to the brain [...] such as recognition, comprehension, memory and thinking." Consequently, primary and secondary aphasia have to be differentiated: in primary aphasias linguistic processes per se are affected, and in secondary aphasias language problems arise from deficits in attention, memory, or cognition (see Caplan, 1992, 16).

Standard teaching in aphasiology assumes that aphasic symptoms come in predictable bundles, in so-called aphasic syndromes (e.g. Goodglass, 1993; Huber et al., 1983; Kirshner, 1995b). Well known is the classification into Broca's aphasia, Wernicke's aphasia, global aphasia, anomic aphasia, conduction aphasia and various transcortical aphasia types. However, the syndrome approach is under heavy criticism. Among other things, it is not clear whether certain symptoms are a necessary prerequisite for classification into a specific syndrome, e.g. there exist Broca's aphasics without agrammatism (Goodglass, 1993, 217), even though agrammatism is supposed to be the main feature of Broca's aphasia. In addition, certain symptoms like agrammatism seem to be themselves syndromes rather than simple symptoms (see Menn & Obler, 1990; Tesak, 1991). Consequently, the assumption of homogeneity of aphasic groups (e.g. Broca's aphasics) could not be methodologically upheld (e.g. Caramazza, 1984; 1986), and thus group studies of aphasics are theoretically worthless (e.g. Tyler, 1987). Another, related problem is that similar linguistic surface phenomena may be caused by different psychopathological reasons.

2. HISTORY OF APHASIOLOGY

The history of aphasiology is long and fascinating (for details and surveys see Caplan,

1987; Howard & Hatfield, 1987; Whitaker, 1998; Jacyna, 2000; Tesak, 2001). The first reports on aphasic persons date back to the first written medical reports in Ancient Egypt. The Greek and Roman period saw many case descriptions of apoplectic patients with aphasia, alexia, and agraphia. The common interpretation was that the patients were supposed to have a memory deficit, and this deficit causes problems in word finding and speaking. In this classical period it was already hypothesized – though by a minority only – that the human brain was responsible for the higher functions. The ventricles were supposed to be of special importance – the fourth ventricle in this view was responsible for memory functions. Given the idea that aphasia is memory-related, early localizationism could state that aphasia results from lesions in the posterior areas of the head/brain.

During the Middle ages the cell doctrine

FIGURE 2. Medieval cell doctrine (after a picture from 1490).

was dominant – different cells were held responsible for different functions. The physical localization of the cells is hard to establish, thus in many medieval pictures the cells are depicted in different ways on the skull (see Figure 2 for an example).

The period from the Renaissance to the 17th century brought new knowledge in all scientific disciplines. Anatomy in general and brain anatomy made rapid progress. Leonardo da Vinci (1472–1519), Andreas Vesalius (1514–1564), Thomas Willis (1626–1675) and René Descartes (1596–1650) have to be mentioned in this context. Leonardo investigated the form of human ventricles, Vesalius and Willis were milestones in brain anatomy, and Willis also postulated some kind of localizationism: e.g. the cortex was held responsible for memory functions. Descartes finally developed the idea that the human body functions like a hydraulic machine.

During the period from the Renaissance to the 17th century many case studies were published. In the 15th and 16th centuries, Antonio Guainerio (see Finger, 1994, 19), Nicolo Massa (see Howard & Hatfield, 1987, 22), and Francisco Arceo (Finger, 1994, 372) write about various traumatic and cerebro-vascular cases of aphasia. Johannes Schenck (1530–1598; see Luzzatti & Whitaker, 1996) describes at least 16 cases of language disorders due to traumatic brain injury. Schenck rejects the cell doctrine arguing that there are cases known to him that have the fourth ventricles intact and still no memory (or language) disorder. Schenck also distinguishes between dysarthric and aphasic patients. Johannes Jakob Wepfer (1620–1695, see Luzzatti & Whitaker, 1996) and Johann Schmidt (1624–1690, see Benton & Joynt, 1963) also describe interesting cases, among them a case of alexia without agraphia.

In the 18th century, scientific endeavors flourish in all fields, and descriptions of

aphasic patients increase both in quality and quantity. Famous case descriptions stem from Olof Dalin (1708–1763, see Benton & Joynt, 1960, 211f.), Giambattista Vico (1688–1744; see Denes & Barba, 1998) and Johannes Gesner (1738–1801; see Gesner, 1789). Gesner is probably the first to clearly understand the phenomenon of aphasia with its communicative and psycho-social impact. Interestingly, Gesner calls the problem "language amnesia", which makes clear that Gesner assumes a selective memory deficit underlying the aphasic language problem.

The 19th century saw the birth of aphasiology as a medical discipline. On the basis of Franz Josef Gall's (1764–1828) discovery of cerebral localization at the beginning of the century (see summary in Tesak, 2001, 47–52), phrenology led to virtually hundreds of case studies in the first half of the 19th century that tried to prove that the language faculty is seated in the frontal regions of the brain (see Williams, 1894).

In France, the case of language localization was taken up by Jean-Baptiste Bouillaud (1796–1881; see e.g. Bouillaud, 1825). Bouillaud's son-in-law, Ernest Auburtin (1825–1893; see Stookey, 1963) started the famous debate on language localization in Paris in 1861, during which Pierre Paul Broca (1824–1880; see Schiller, 1992) presented his famous cases of Leborgne (Broca, 1861a; 1861b) and Lelong (Broca, 1861c). In these aphasic cases, lesions in the third frontal convolutions were established post mortem (see Figure 3 for the famous brain of Leborgne). Consequently, the seat of the "articulate language" was assumed to reside in that region, which later was called Broca's area.

When it was established that aphasic persons also have problems in comprehension (by Bastian, 1869; and Meynert, 1866; see Whitaker & Etlinger, 1993), it was postulated by Carl Wernicke (1848–1905; see Kleist, 1970) in his famous 1874 book "Der aphasische Symptomencomplex" that there exists another language center whose function is to comprehend words. Its seat is assumed to lie in the first temporal convolution of the left hemisphere. Wernicke, together with Ludwig Lichtheim (1845–1928), finally developed the following classification scheme: cortical sensory aphasia, subcortical sensory aphasia, transcortical sensory aphasia, cortical motor aphasia, subcortical motor aphasia, transcortical motor aphasia, conduction aphasia (see Tesak, 2001,

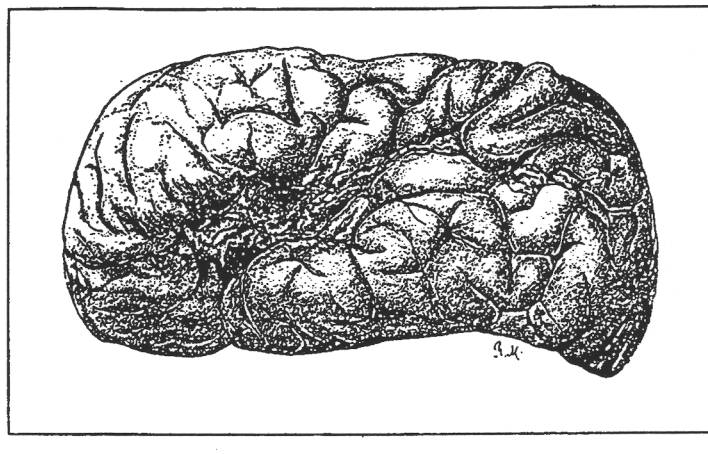


FIGURE 3. The brain of Broca's 1861 patient Leborgne.

101ff.). With the work of Gall, Broca, Wernicke and Lichtheim, the so-called classical aphasia doctrine was established. Within the work by Jean-Martin Charcot (1825–1893; see Brais, 1993) this paradigm finds its place, and Figure 4 depicts the assumed lesion sites of various aphasic disorders (agraphia, motor aphasia, sensory aphasia, alexia) according to his theory.

Even though the classical aphasia doctrine dominated the last third of the 19th century, it was opposed from the beginning on by various aphasiologists, prominent among whom were Hughlings Jackson, Constantin von Monakow, Pierre Marie, Henry Head and Kurt Goldstein.

One of the early critics, John Hughlings Jackson (1835–1911; see Critchley & Critchley, 1998), whose importance was only to be recognized in the 20th century, pointed out that the sentence or proposition was the central aspect in language: "Speaking is not simply the utterance of words. The utterance of any number of words would not constitute

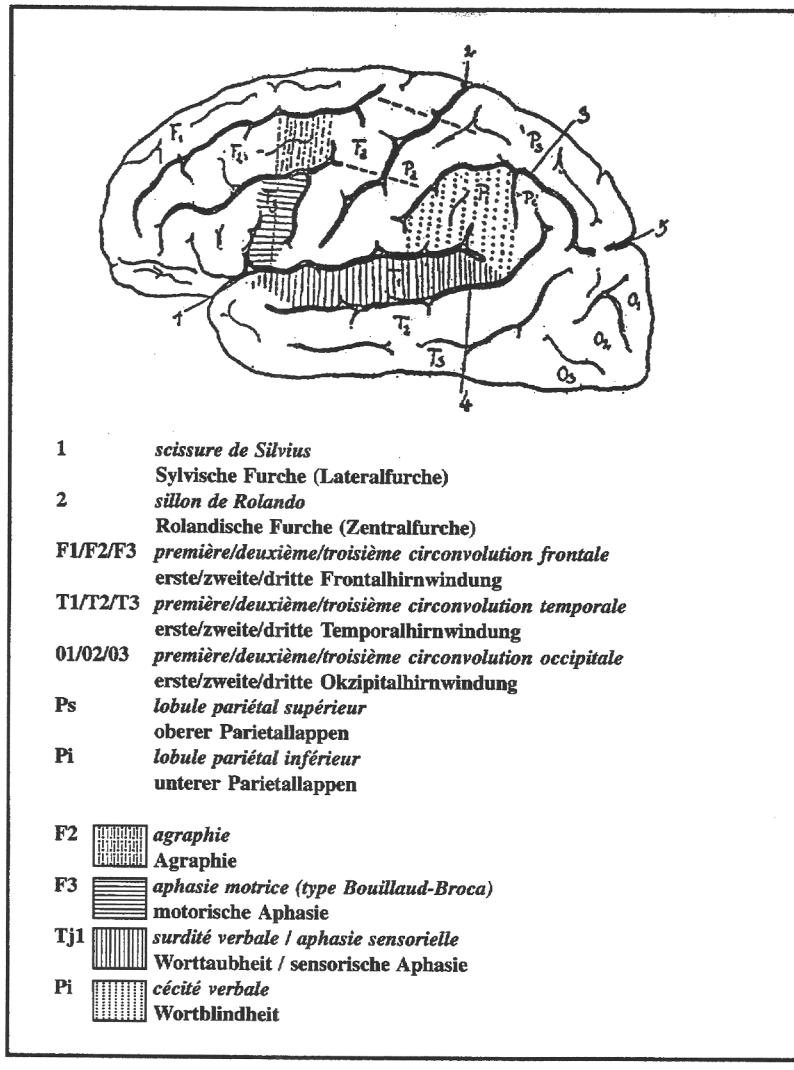


FIGURE 4. Localization of aphasia after Charcot.

speech. Speaking is 'propositionising'" (Hughlings Jackson, 1874/1958:130). Consequently, the "[l]oss of speech is the loss of power to propositionise" (Hughlings Jackson quoted after Schulte 1994:152). With this, one of the weak points of the approach of the Wernicke type is attacked, namely the fixation on words as the main aspect of language.

Jackson also distinguished between volitional propositional speech and automatized non-propositional speech. An aphasic person has the problem of "not being able to propositionise in any way", but (s)he "has not lost the automatic use of words" (Hughlings Jackson, 1874/1958:133). Thus aphasic persons may use social formulae, curse, sing and recite overlearned material. In relation to localization, Hughlings Jackson is an opponent of the strict localization of the language faculty, which in itself is doubtful as a concept: "I think, then, that the so called 'faculty' of language has no existence" (Hughlings Jackson 1866/1958:123) and "I have never acceded to the opinion that speech is to be localized in any one spot" (Hughlings Jackson quoted after Critchley & Critchley, 1998: 98). Early support for anti-localizationist position in Great Britain came from Frederic Bateman (1824–1904) who wrote one of the first monographs on aphasia (see Bateman, 1870).

Anti-localizationism is often mentioned in one breath with holism. This is not quote correct, since holism (in relation to the brain) simply states that the whole brain interacts in accomplishing tasks like language. For example, the Swiss neurologist Constantin von Monakow (1853–1930) on the one hand assumed that the core functions can be localized, but on the other hand held the whole brain responsible for the higher functions (e.g. von Monakow, 1905).

Holism in relation to the person means that a specific problem affects the whole person. As Kurt Goldstein (1878–1965) puts it: "In pathological cases like in aphasia we do not only have a person with altered language, but also a person ... with other symptoms. One never should look at symptoms isolated from the ill person" (translated from Goldstein 1927/1971:164). And even Goldstein, "the epitome of holistic ... aphasiology" (Howard & Hatfield, 1987:45) has often localizationist opinions (e.g. in Goldstein, 1948).

Clear anti-localizationist positions were rigorously put forward by Pierre Marie (1853–1940) and Henry Head (1861–1940). Marie launched a debate on aphasia in 1906 when he published a paper with the provocative title "The third left frontal convolution plays no role for language function" (Marie, 1906). The "Paris medical community was shocked with Marie's article" (Brais, 1992:693) and the classical aphasia doctrine became seriously weakened as the leading paradigm in the consequent debate (see Brais, 1992, and Leischner, 1992, for summaries).

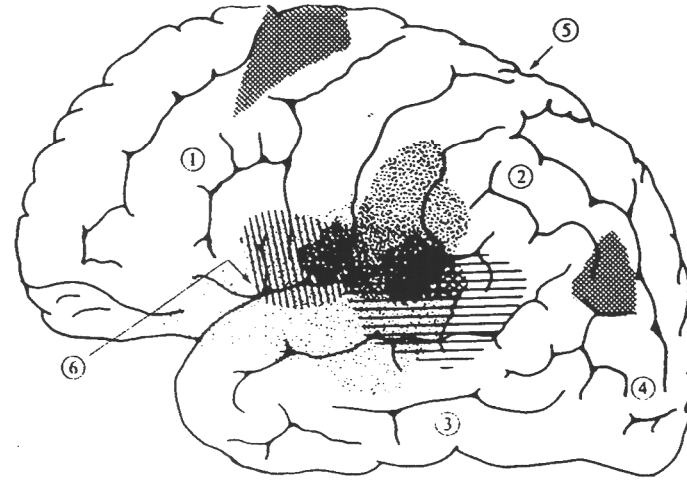
Head re-introduced the works of John Hughlings Jackson into the aphasiology of his time and made Jacksonian views known to mainstream neurology, and its impetus for holism cannot be overestimated. Head's own, two-volume work "Aphasia and Kindred Disorders of Speech" appeared in 1926. It is the peak of anti-localizationist and holistic opinions in aphasiology in the 20th century. In relation to the classical doctrine in aphasia (represented by Bastian, Broca, Wernicke and others), Head writes, that "[m]ost of the observations [by the diagram-makers...] failed to contribute anything of permanent value to the solution of the problems of aphasia" (Head 1926/I:65).

The classical aphasia doctrine was also attacked from another side. Taking up arguments from Hughlings Jackson, it was argued (i) that aphasia cannot be understood

simply from anatomical study alone and (ii) that the focus on words and problems in word processing is far too limited. Eminent in this respect is the work by neurologist Arnold Pick (1851–1924; e.g. Pick, 1913, see Kertesz & Kalvach, 1996). He argued for the inclusion of contemporary linguistics and psychology into the investgation of aphasia, and he was also interested to overcome the fixation on single words within the classical aphasia doctrine, thus forcefully arguing for inlusion of disorders of sentences as main topic of aphasiology. And indeed, the first 30 years of the 20th century witnessed a highly interesting and sophisticated debate on grammatical disorders in aphasia (for a survey see de Bleser, 1987). Contributors, among others, are Karl Kleist, Max Isserlin, Arnold Pick, Karl Heilbronner, and Erich Salomon.

By 1930, holism and anti-localizationism dominated aphasiology, and the situation remained unchanged through World War II. At the same time, fascism terminated a fruitful discussion of aphasia in central Europe, and many leading aphasiologists had to emigrate (e.g. Goldstein, Isserlin), lost their positions or did not survive (Gelb, Forster). After World War II, the classical Wernicke-Lichtheim model was revived through the Bostonian neurologist Norman Geschind, who helped to (re-)launch the most influential scheme of neo-connectionism whose basic principle can be shown in Figure 5. In it, various aphasia syndromes are depicted with their presumed lesion localization.

This approach is still influential, especially within medicine. Test Batteries like the BD-AE (Boston Diagnostic Aphasia Examination, Goodglass & Kaplan, 1973), the AAT (Aachener Aphasie Test, Huber et al., 1983)



The lateral surface of the brain and its major anatomical landmarks, with a summary of the lesions of various aphasia types. 1. Frontal lobe; 2. Parietal lobe; 3. Temporal lobe; 4. Occipital lobe; 5. Rolandic fissure; 6. Sylvian fissure.

FIGURE 5. Classic aphasia doctrine in contemporary clinical aphasiology (after Kertesz & Wallesch, 1993: 126).

or the WAB (Western Aphasia Battery, Kertesz, 1982) are based on the Wernicke-Lichtheim-Geschwind model, and many aphasiologists in clinical contexts deal with terms like Broca's aphasia on a daily basis. However, the anatomically based syndrome approach to aphasia is unsatisfactory due to several reasons. As already mentioned, syndromes are rather vague categories that can only be used as rough and superficial descriptions of the patients' behavior, and the correlations between localization and syndromes are difficult to prove. de Bleser (1988, 182) states after she studied the lesion sites of aphasia syndromes: "No correlations [...] were exceptionless. In some cases, as for Broca's aphasia, the exceptions were as frequent as the expected rule."

After World War II, aphasiology became a truly interdisciplinary enterprise. Besides medicine, linguistics, philosophy, psychology and therapy were involved. Presently, linguistic and neuropsychogical investigations are a major force in gaining a better understanding of aphasia, even though through the use of new brain-imaging technology a return of brain oriented aphasiology seems likely.

3. LINGUISTIC AND NEUROPSYCHOLOGICAL INVESTIGATIONS OF APHASIA

The first linguist to investigate aphasic language was probably Heymann Steinthal (1871), and researchers like Pick (1913) had demanded to introduce linguistic knowledge into aphasiology, but only after World War II linguistics became a major theme within aphasiology. The Russian neuropsychologist Alexander R. Luria (1902–1977) relied heavily on the linguistic work by Roman Jakobson (1896–1980), whose *Kindersprache, Aphasie und allgemeine Lautgesetze* (1941) gave a major impetus to the field (after its translation into English in 1968) (see Goodglass & Blumstein, 1973c). Important for the field was the so-called Chomskyan revolution in linguistics, which also influenced aphasiology (cf. Newmeyer, 1980). Noam Chomsky developed generative grammar (Barsky, 1997), in which linguistics is understood as part of psychology or biology. Thus, interest in biological aspects of language became a natural part of linguistic investigation and, consequently, interest in psycho- and neurolinguistics grew. Psycholinguistics and the related field of neuropsychology tried to develop models of the human language processing machinery. Cognitive Neuropsychology (CN) developed within neuropsychology (see Shallice, 1988). CN tries to model various cognitive phenomena using models of normal behavior (cf. Ellis & Young, 1991; Shallice, 1988). One strong line of argumentation within CN comes from data obtained from brain-damaged patients. In addition, external evidence was welcomed within the Chomskyan paradigm. This meant that corroborating evidence from, e.g., aphasia was sought. A number of contemporary linguists in aphasiology work within a generative paradigm (see Grodzinsky, 1990).

Neuropsychological and (psycho-)linguistic investigations of aphasia have two goals. On the one hand, one tries to understand pathological language and language processing, on the other hand, neurolinguistic data is used to understand normal human language in a better way.

In the following, I will restrict my discussion in two ways (for more extensive and in-depth surveys see Blanken et al., 1993; Caplan, 1992; Fabbro, 1999; Stemmer & Whitaker, 1998; Kirshner, 1995a). First, I will discuss spoken aphasic language only, even though it must be clear that aphasic persons do also have problems in comprehension,

writing and reading. (The topic of written language will be briefly mentioned in the section on language therapy.) Second, I will deal with selected aspects only. For example, I will not discuss repetitive phenomena (perseverations, stereotypes, etc.) (see Wallesch, 1990, for a review).

3.1. Phonological and semantic disorders

At the word level, aphasic persons have problems with the phonological structure of words (see Kohn, 1993, for an overview). Phonemes can be substituted, deleted, added, misordered or a phonological feature may be changed. The result is named phonological (phonemic) paraphasia. Phoneme chains that are not conventionalized in a given language are called (phonological) neologisms. The problem for classifying segmental errors is that sometimes the target word is easy to recognize even though the phoneme chain per se does not exist as a word (e.g. [epl > epe]. Other neologisms, e.g. [he:pa] for "lion" cannot be related to the target form. Thus it is common practice to call phonological neologism that are recognizable phonological paraphasias, and if they are not recognizable they are phonological (or abstruse) neologisms. Goodglass (1993) uses a more formal criterion: more than 50% correct = paraphasia, less than 50% = neologism. However, this type of classification depends on the hearer's knowledge about the intended target. In repeating words, the target is known, usually also in naming. But in spontaneous speech, the hearer is often unaware of the intended target word and thus is often not able to judge the percentage of correctness. If the phonologically deviant but target-related phoneme chain is a real word, and if there is no semantic relation, it is called a formal paraphasia (e.g. computer > communist).

Interestingly, segmental errors seem mainly to be restricted to content words, while function words are less vulnerable to phonological problems. This phenomenon can be seen in its extreme in so-called phonological jargon where (often) normal syntactic markers are mixed with phonological neologisms, i.e. sound/phoneme-chains that are not meaningful in the target language.

Semantic disorders (see Gurd & Marshall, 1993, for a survey) lead to semantic paraphasias, which are common in many aphasic persons (and also in patients with Alzheimer dementia). In such cases the target word is substituted by a semantically related word, i.e. the erroneously produced word has some relation in meaning (table > chair, Volvo > Fiat). Various sources of semantic errors have been discussed, mainly along the lines degradation of the semantic network vs. access problems (see below).

Category-specific semantic deficits are of special interest, and there the most commonly reported dissociation concerns living vs. nonliving things, i.e. aphasic persons may be able to name all living things, but none that are inanimate, or vice versa. Sometimes more selective deficits are reported for categories like animals and fruits. Another topic of discussion is whether semantics is modality-specific or not. In the first case, each language modality (speaking, comprehension, reading, writing) would have its own semantic network. This would help to explain why in certain patients semantic deficits can mainly be observed in one modality. On the the other hand, semantic problems in aphasic patients are usually present in a supramodal manner (i.e. in all modalities). Thus many models assume a central semantic system for language.

Other interesting phenomena on the word level are the following. Different word classes are subject to different breakdown patterns in aphasia (see Friederici & Saddy, 1993),

and generally open word classes and closed class items are subject to dissociations. It also seems that morphological processes like inflection, derivation and compounds can be selectively damaged (cf. Badecker & Caramazza, 1993, for a survey).

In explaining word level phenomena the so-called logogen model (Morton, 1970; Marshall & Newcombe, 1973) has been very influential and it exists in various variants (Butterworth, 1993; Blanken, 1991b; Kotten, 1997; Nickels, 1997). The model is designed to represent the mental architecture of single-word processing in all modalities (speaking, repeating, naming, comprehension, reading aloud, reading comprehension, written naming, writing to dictation, copying, etc.). The part of the model that deals with speaking single words can be seen in Figure 6 (after Nickels, 1997).

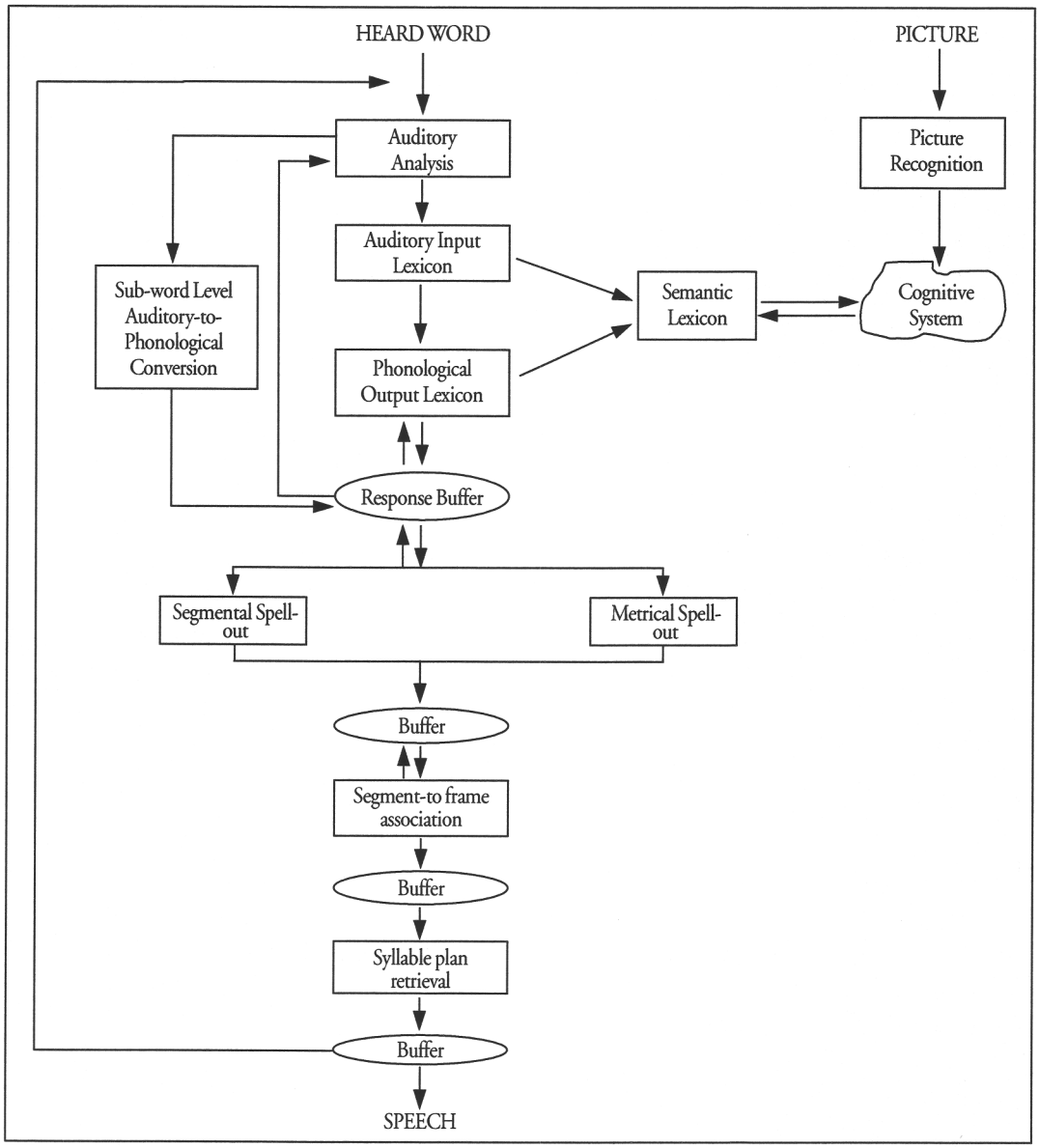


FIGURE 6. Part of the logogen model (after Nickels, 1997:94).

As can be seen, the model is composed of various sub-lexicons (for auditory input, semantics, phonologcal output) and processing routes that are used to explain different error types on the word level. Inability to access the semantic lexicon via the auditory input lexicon leads to problems in auditory comprehension. Should the problem be related to the word form (i.e. phonological access or storage problems), closely related entries in the input lexicon may be selected and then sent forward into the semantic system (e.g., the target /sneik/ may be understood as /sneil/). Moreover, semantic access problems lead to semantically-related misunderstandings (/kau/ will be understood as /pig/). However, in such cases, output will not be affected, but it would be affected, should the problem for the aphasic person be within the semantic lexicon. Then both comprehension and production will be erroneous at the semantic level. Within the semantic lexicon, various structuring principles have to be assumed to explain e.g. category-specific disorders.

The phonological output lexicon is the long-term-memory store for the phonological representation of words (including both segmental information on individual phonemes and suprasegmental metrical and syllabic information). A disorder within the output lexicon will lead to phonological paraphasias both in naming and spontaneous speech. Repeating will not be affected with non-words since non-words per definition are not represented within the lexicon and they have to use the auditory-phonological conversion route. Whether the repetition performance on words will be affected depends on the route the speaker chooses; should the phonological output lexicon be involved (which in normal processing is usually the case), then performance will be affected.

The response buffer and the buffer following segmental and metrical spell-out can also be subject to aphasic problems. In such cases, phonological deviations will affect all spoken performance, be it naming, repeating or spontaneous speech.

Generally, the model-oriented approach has become an important, highly productive and fairly complicated approach within aphasiology. A major force within this paradigm is the use of detailed single case studies. It also has to be added that the discussion of disorders in the cognitive neuropsychological domain often takes place outside any localizationist considerations, i.e. in most cases it is not assumed that certain functional elements of the models can be precisely localized in the brain.

More recently, connectionist network-modelling has gained some ground within aphasiology. These "networks compute via the parallel co-operative and competitive interactions of a large number of simple neuron-like processing units" (Plaut & Shallice, 1994, 7f) and thus are unlike box-and-arrow-models like the logogen model. (For further discussion of connectionist network models of language production see Schade, 1999; Plaut & Shallice, 1994.)

3.2. Syntactic disorders

Breakdown of syntactic abilities is a frequent phenomenon in aphasic persons, i.e. grammatically well-formed structures are not produced when intended and conversationally required (Benson & Ardila, 1996). Structures are aborted, incomplete or blended. Widely two major forms of syntactic breakdown are accepted: so-called agrammatism and so-called paragrammatism. Both disorders are syndroms (bundles of symptomes) rather than unitary symptoms. Agrammatism has been of special interest to linguists for decades and it still is a major field of lin-

guistic aphasiology (see Kean, 1985; Whitaker, 1997).

Agrammatic output is characterized by following symptoms (see Menn & Obler, 1990): overrepresentation of open-class items (nouns, verbs, adjectives); selective disturbance of closed class items (determiners, pronouns, prepositions, etc.) and inflectional morphology; nouns lack case markings, verbs lack inflection; low number of pronouns and determiners; structural simplicity; lack of complex sentences and subordination; problems with verbs (omission, underrepresentation in contrast to nouns, lack of inflection). Due to a skeletal form of agrammatic utterances, the term "telegraphic language" has been applied to agrammatism, but this is a misnomer, since telegrams and agrammatism share superficial similarities only (Tesak & Niemi, 1997).

Cross-linguistic research (Menn & Obler, 1990) has shown that impairment of grammatical morphology seems to be the hallmark of agrammatism regardless of the language. However, language specific aspects have to be taken into account to describe the agrammatic pattern(s) in different languages; e.g. the issue of omission vs. substitution of grammatical morphemes. In morphologically poor languages like English omission seems to be the dominant feature through deletion of closed class items, whereas in languages like Finnish also substitution of grammatical morphemes may be observed in agrammatism (Niemi et al., 1990).

The challenging question to linguists has been from the beginning whether elements affected in agrammatic spontaneous speech have an inherent connection. A major interest in agrammatism has come from Chomskyan linguistics and it has generated a series of theoretical accounts of agrammatism (see Penke, 1998, for an overview). A well-known hypothesis is the trace deletion hypothesis (TDH) by Grodzinsky (1990) in the GB framework, where it is postulated that traces are absent from agrammatic structural representations (see summary and critique in Kolk, 1998). Minimalist program hypothesis (Chomsky, 1995) is the Tree Pruning Hypothesis (TPH, Friedmann and Grodzinsky, 1997) that tries to relate the deficit in tense inflection, in the selective deficit in subordination production, and in the deficit in producing WH-questions. On this account agrammatic deficit is due to a deficit in the Tense node; and the structures above mentioned depend on information from nodes higher up in the hierarchical syntactic structure. Thus, they are all impaired.

Several facts are problematic for linguistic-descriptive accounts: (i) variability of error patterns, (ii) dissociations of error patterns within and between tasks, (iii) changing linguistic theories. Consequently, many contemporary approaches focus on processing deficits rather than structural descriptions, since processing approaches seem to be more adequate to deal with variable agrammatic "behavior". Several research groups favor the so-called "limited capacity" approach, i.e. the assumption is that agrammatic speakers are unable to build up syntactic structures due to either temporal or spatial restrictions in the language processing machinery (see Kolk, 1998, for an overview).

Finally, a few researchers discuss the option that agrammatic surface behavior is influenced by communicative and social aspects (Heeschen and Schegloff, 1999), and indeed, it seems that individual agrammatic speakers consciously simplify their spoken output.

In contrast to agrammatism, paragrammatism has received little attention in the linguistic literature (see de Bleser & Bayer, 1993). Paragrammatism is a grammatical deviation that violates the normative rules of morphosyntactic convention, and it is char-

acterized by substitutions of grammatical morphemes, by blending and overlapping sentence structures (...when I saw the woman gave me bread...), and by sharing the same constituents (...not even in the hospital they could not help...). (Huber & Schlenck, 1988). Sentential structures tend to be long and complex. Paragrammatism may result from (i) overuse of grammatical elements (particularly connectors) with a decrease in lexemes, (ii) an erroneous selection of grammatical elements, (iii) an absence of defining limits in sentences and utterances (and correlated often with an excessive verbal output). It remains unclear whether paragrammatism is a purely linguistic or rather a cognitive deficit (related to disordered monitoring mechanisms). Things become even more complicated since paragrammatism is often associated with semantic and phonological paraphasias that may lead to so-called jargon (fluent speech production, not making sense to listeners). In the case of phonological jargon, syntactic frames are filled with neologisms in the positions of lexical items whereas grammatical morphology seems intact. An interesting, process-oriented explanation for paragrammatic behavior is the loss of inhibition during production so that competing structures are put forward to production, which leads – under the necessity to produce only one item at a time – to substitutions of grammatical morphology and sentential blends (Huber & Schlenck, 1988).

Paragrammatism is often conceptualized as the opposite to agrammatism but both phenomena share certain features (esp. concerning the use of grammatical morphemes related to syntactic processing) so that there seems to be an overlap between agrammatic and paragrammatic symptoms (Wallesch & Kertesz, 1993). Moreover, looking into other languages than English it becomes more apparent that there is an overlap of symptoms, mainly concerning the use of grammatical morphemes related to syntactic processing (e.g. in German, Finnish).

3.3. Pragmatic disorders

Pragmatics is a wide field and for many it can be defined similar to the following: pragmatics deals with the use of language in context and discourse; it also deals with intentions of speakers and thus incorporates even interlocutors (see Levinson, 1983). Topics of pragmatics range from the relation between linguistic and non-linguistics signs, over deixis, reference, anaphora (all of which deal with the connection between language and the outer world) to conversational analysis (CA) dealing with turn taking/turn exchange, construction of texts and narrative discourse, conversational sequences, openings and closings, and repairs in conversation (see aphasia-related overview in Perkins et al., 1999).

Pragmatics remains an outlier within the tradition of classical aphasiology, since aphasic persons are per definition unimpaired when it comes to pragmatic skills, i.e. aphasic persons know the rules for communication (e.g. turn taking) and have the usual communicative intentions. The aphasic problem is supposed to be found at the level of linguistic means. Pragmatic problems in isolation such as in Alzheimer patients and patients with right hemisphere lesions are not usually labeled aphasic (Joanette & Ansaldo, 1998). Communicative and discourse problems are usually connected to non-aphasic neurological populations only. However, this position may not be justified, since pragmatics and grammar are deeply interrelated like two sides of one coin (see Lesser & Milroy, 1993). Thus it seems that linguistic aphasiology will have to broaden its focus. Researchers like Gibbs (1999) stress the point that pragmatic aspects are integrated

into language processing and thus they are not merely a special level on top of language processing per se. Thus pragmatic impairment is on its way to become an integral part of the definition of aphasia. In addition, recent research shows several problematic pragmatic domains for aphasic persons (see Stemmer, 1999; Paradis, 1998).

In reviewing communicative aspects of aphasia, Feyereisen (1993) discusses two standpoints in relation to gestural and other non-linguistic expression modes: on the one hand, some authors report equally disturbed linguistic and non-linguistic expression, on the other hand, aphasics were observed to overuse the non-linguistic channel (probably in order to compensate for the linguistic deficit).

Aphasic narrative discourse seems to preserve semantic content and textual macrostructure (whose disturbance often is considered cognitive rather than linguistic in origin), but several deviations from normal discourse can be observed: word ratios are different (e.g. verb/noun ratios) (see Berko-Gleason et al., 1980); sentences are syntactically less rich (length, complexity) (see Ulatowska et al., 1981); there are more deictics than usual (Dressler & Pléh, 1988); anaphoric pronouns are often used without clear reference, which points towards deficits in the use of referential systems (see Cardebat, 1987); such lexical items are reduced that are used to express the narrative message (Berko-Gleason et al., 1980).

Conversational discourse has only recently received attention within neurolinguistics (for an overview, see Chantraine, Joanette & Cardebat, 1998). Within spoken conversations, repair is particularly relevant to aphasic persons, since in aphasic communication difficulties routinely emerge to the extent that repair processes are much more frequent than in normal, non-aphasic conversation. The most important type seems to be collaborative repair where both the speaker and the hearer contribute to successful problem solving. Perkins et al. (1999) describe various repair patterns and the complicated interconnection between the trouble source, linguistic deficits, cooperativeness of interlocutors and individual aspects. Generally, self-repairs are more frequent in clinical situations than in conversations with non-aphasic family members. Other-initiated repairs are more frequent in familial situations, where family members are often routinely engaged in repairs (Lesser & Milroy, 1993). (For an overview on aphasia and repairs, see Lindsay & Wilkinson, 1999.)

4. APHASIA THERAPY

Aphasia rehabilitation has become an important topic due to the fact that many individuals with strokes and head injuries become aphasic. For recent reviews on aphasia therapy, see Howard & Hatfield (1987), Holland & Forbes (1993), Paradis (1993), Springer (1997), Estabrooks & Holland (1998), Blomert (1998), Tesak (1999). Within aphasia therapy one distinguishes between various broad approaches in relation to the four dimensions of aphasia as depicted in Figure 1 above. I will briefly discuss these separately below:

First, *medical intervention* has to be considered. Usually, brain damaged patients have to be treated in a number of ways, e.g. in trauma centers or stroke units. Since it seems that the human brain is flexible and "repairable" (see Stein et al., 1995), language-targeted pharmacology has in recent years gained some ground, and "numerous investigators have demonstrated a beneficial effect on aphasia from a variety of pharmacological treatments" (Minura et al., 1995). Usu-

ally the effects stem from unspecific influences on the neurotransmitter system in the human brain, but presently "we are aware of no neurochemical agent that has yet been rigorously proven to ameliorate specific language signs or symptoms" (Minura et al., 1995, 477). In relation to language recovery, it seems that language therapy that is supported by various pharmacological means may be more beneficial than the same treatment without pharamocological support.

At the second level we may speak of *traditional speech and language therapy*. Language-oriented approaches try to restore linguistic skills: words and sentences, reading, comprehension of words and sentences are the goals for therapy. This approach usually makes use of linguistically structured therapy materials to stimulate the patients to produce selected linguistic targets. The therapist uses cues, prompts and various other means to support the patient. More recently, linguistic and neuropsychological modeling has been applied to structure-related therapy goals. And, as Lesser (1993, 258) claims, this "has considerable potential for aphasia therapy", since "it has contributed a theory-based rationale for planning intervention and for selecting which of the multiplicity of materials and well-tried techniques should be appropriate for a particular patient at a particular time".

However, linguistic and model-oriented approaches start out (and end) with the idea that language (or more restrictively: grammar) per se is the problem in aphasia (cf. the hypothesis of external modularity of grammar). But as we know from numerous studies and reports from aphasic persons, linguistic ability per se and communicative everyday functioning do not correlate directly. Sometimes, aphasics are communicatively more successful than one would expect on the basis of the purely linguistic (grammatical) skills in the patient, and sometimes the situation is the opposite.

Consequently, *pragmatic and communication-oriented approaches* focus on communicative skills and communicative success rather than on grammar and correct structures per se (see Carlomagno, 1994, for an introduction). Since in most chronic aphasic cases premorbid language skills cannot be assessed, it really makes sense to consider the question of how a person can be enabled to reach his/her communicative goals irrespective of the means the persons use. Thus themes likes strategies, compensation, nonverbal skills, and – of course – the communication partner come into focus. An influential approach in this respect is PACE, an acronym for Promoting Aphasics' Communicative Effectiveness (see Davis & Wilcox, 1985). Its basic principles are: 1) there should be an exchange of new information, 2) there is a free choice of communicative channels (writing, gesturing, drawing, etc.), 3) feedback is based on communicative adequacy, and 4) the roles of participating communicators may change (see Glindemann & Springer, 1995, for a discussion of PACE).

Since communication is a co-operative process, not only the aphasic person has to be trained in successful communication, the training of conversation partners becomes necessary to help aphasic persons to communicate (see Kagan & Gailey, 1993). Typically, the communication partners are taught speaker and hearer strategies to enhance their chances of to be understood and to understand. Such strategies could include, e.g. repetition of important communicative themes, keeping an eye contact and reducing the background noise.

Fourth, in addition to intervention related to medicine, language and communication, *psycho-social intervention* is becoming an important factor since the psycho-social se-

quelae are enormous for aphasic persons (see Lafond et al., 1993, Gainotti, 1997, Code, 1999, Williams, 1996, Lyon, 1998). Herrmann et al. (1993, 190) summarize the situation: "Social isolation, deprivation, and changes in social status have been reported as frequent social consequences of aphasia." Especially depression seems very frequent in aphasia, and "prevalence studies indicate that 40–50% of stroke patients may develop depression during the acute period, and an additional 30% may develop depression at some time during the first two years" (Williams, 1996, 306).

In addition to the aphasics' problems, family members may also suffer. As Boisclair-Papillon (1993) puts it: "Without any doubt, the family of the person with aphasia also reacts to the illness and undergoes behavioral and attitudinal changes." It is undoubtedly true that "[a]n individual's aphasia is a family problem" (Davis, 1983, 290). Within this perspective the help of psychologists, family therapists, social workers, and related professionals becomes an integral part of aphasia rehabilitation. Lyon (1998) provides many useful suggestions to partners (friends, spouses, relatives, care givers, etc.) of aphasic persons. Recently, the self-help movement has gained influence worldwide and aphasic centers and self-help groups become more and more important to supplement public, medical and individual care of aphasic persons.

Generally, treatment efficiency and the efficacy of aphasia therapy have come focal areas of interest (see Wertz, 1993; 1995; Holland & Thompson, 1998). Due to economic considerations, Health Care systems all over the world ask for efficacious and efficient treatment. Thus recently it has become an important task within the field to understand how therapy works. However, as a logical prerequisite, the aphasic language processing (and grammar) as well as the communicative and psycho-social aspects of aphasia have to be understood.

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AFASIA, KIELIOPPI JA KIELI: HISTORIALLINEN NÄKÖKULMA

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Katsausartikkelissa kuvataan afasian tutkimuksen eri piirteitä. Historiallisessa katsauksessa keskitytään ennen 1950-lukua tapahtuneeseen edistykseen, jossa yksi keskeinen teema on liittynyt "lokalisationismiin" ja "holismiin". Keskeisiä henkilöitä ovat olleet mm. Gesner, Gall, Broca, Wernicke, Lichtheim, Jackson, Marie, Head, Pick, von Monakow ja Goldstein. Nykyisessä afasiatutkimuksessa on painottunut erityisesti fonologisten, semanttisten, syntaktisten ja pragmaattisten häiriöiden kielitieteellinen ja neuropsykologinen kuvaus. Katsauksessa lopussa kuvataan afasiakuntoutuksen nykytilannetta.

Avainsanat: afasian historia, fonologiset häiriöt, semanttiset häiriöt, syntaktiset häiriöt, pragmaattiset häiriöt, afasian oireyhtymät, afasiakuntoutus