Linguistic Consequences of Different Types of Linguistics Impairment due to Cerebral Damage

An Essay by
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The study of *Aphasia*,[1] language impairment due to cerebral damage, is a beautiful example of how science works. Starting from purely empirical observations, at first in clinical medicince and psychology, this field soon evolved into a vividly interacting research in many disciplines, including theoretical approaches. Our time is priviliged to witness the process of convergence, where the different approaches, theoretical buildings and hypotheses come together to form a more and more consistent picture of how the (linguistic) mind works. As I want to show in this essay, linguistic learnt a great deal from the study of cases where the linguistics abilities are *not* properly present, and this is strongly reflected in the way, linguists of today divide aphasia into different groups. I want to emphasize right from the outset that any categorization of a class of phenomena (here aphasiac cases) involves to a certain degree an *a priori* system of rules, presumptions, and assumptions, which must be carefully justified *a posteriori*.

Therefore, in the first part of the essay, I will describe the various forms of aphasia according to the more or less established system of a modular approach to linguistics abilities, i.e. the point of view that the main levels of a formal language analysis – words and the mental lexicon, syntax and grammar, messages and semantics of phrases and larger units – does more or less correspond to the mental representation and processing of language in our brain. Of course, this statement is heavily oversimplified, since these levels suggest an order or hierarchy of processing, e.g. during language comprehension, from smaller units such as phonems/graphems towards structurally more complex units. However, whether a serial or an interactive processing takes place, is still an open question, although there are strong indications that the mental lexicon is involved in all stages of language comprehension. In the second part, I will examine whether this approach and its implicit categorization of aphasia by its symptoms is a "good" one, i.e. whether it coincides with the experimental evidence on the mental organization of linguistic abilities in our brain, instead of imposing an artificial structure not supported by empirical data. This question, although always cause of much debate, has recently become increasingly important due to new insights into the

[1] In abuse of language, this word, whose meaning is *to be without (spoken) language*, is now commonly used for all kinds of language impairment and all sorts of degree. Formerly, the word *dysphasia* meaning *incorrectly (spoken) language* named impairments of lesser degree.

neurological aspects of the brain after advanced imaging devices became available which allow to glimpse directly into the working brain. These are mainly the positron emission tomograph (PET) and the nuclear magneto-resonance tomograph (NMRT), both allowing for three-dimensional images of the brain showing different degrees of bio-chemical activity. The former has a very good spatial resolution, while the latter gives higher temporal resolution.

I now turn to the description of the main aphasiac types and follow the exposition of this subject by Michael Garman:[2]

Everyone experiences at one time or another difficulties to retrive a certain word. However, when these difficulties exceed the scale of the occasional experience, it is called *anomic aphasia*. Since a general difficulty of word finding is often a common symtpom of manu forms of aphasia, it is generally difficult to diagnose whether the anomiac symptom is part of a more complex language impairment or not. Anomiac aphasia is hence used to describe cases where word finding seems to be the dominant problem. One further distinguishes between word-production anomia, word-selection anomia and different types of specific anomia, where specific sorts of words or word fields are missing. The linguistic consequences of this kind of impairment are an otherwise fluent and spontaneous speech which is often interrupted by pauses due to delayed or unsuccessful word-retrivals and which is full of circumlocutions and use of proforms (place holders). Obviously, according to the modularity hypotheses, the mental lexicon is affected. Interestingly, in many forms of anomiac aphasia, only the phonemic (output-form component) of the word is missing, but an understanding of the concept, as for instance evident from descriptions of pictures, is still present. In addition to this word-production anomia one observes a word-selection anomia with an inability to activate the lexical-form component of the word, but with also preserved general understanding of the concept. Next, there exists the form of semantic anomia, where in contrast the concept or meaning of the word is lost. As for all forms of aphasia, one tries to gain further insight into the precise extent of the impairment by diagnostic tests which besides production of free speech include general auditory comprehension, auditory-oral repetition, written language abilities such as free writing, writing to dictation, coying written sentences, and spelled-word recognition, further confrontation naming and selecting an object in response to its name. Characteristic for the anomiac aphasic is that word-finding difficulties are present in all sectors of speech comprehension and speech production by otherwise good performances. In some cases, the patient will even resist accepting the correct word when provided by the examiner. Again, as for all forms of aphasia, the diagnostic situation might be quite complex. Thus, in general it does not help to provide the patient with the first consonant or vowl of the word, but in the particular case of phonemic paraphasia, where incorrect phonems are substituted in an otherwise correct syllabic structure of the word,

[2]Michael Garman, *Psycholinguistics*, (Cambrdige University Press: Cambridge, 1990), Chapter 8, 'Impairment of Processing'

it sometimes does. Finally, two strange forms of anomia are visual or auditory agnosisa where a word can only be recollected from the sound or image of the object respectively, but not both. These latter forms are insofar interesting, as the corresponding lesion presumably interfers with the connections from the relevant sensory area to the assumed semantic area. Lesions resulting in anomia in a mild form do not seem to lie in specific brain regions, but may be found anywhere, even in the right hemisphere. This is supported by new NMRT experiments that show that the mental lexicon seems to be distributed over wide part of the brain, but with specific locations for specific word fields such as humans, animals, and tools.[3]

Opposed to the quite mild form of anomiac aphasia is the *global aphasia*. The diagnosis of symtpoms is simply that all testable parameters are so severely impaired that it might be impossible to test any other non-language abilities. However, spontaneous speech output is frequently not limited to the level of complete mutism. As for the linguistics consequences, it has been proposed (but not widely accepted) that global aphasia could be characterised as phonemic disintegrations. However, due to the severness of the phenomenon, it is currently impossible to decide whether this simply captures a superficial aspect. A specific linguistic impairment cannot be identified, although some cases of global aphasia evolve into more specific forms with time. Experts then speak of pseudo-global aphasia as merely very sever early stages of other more specific types of aphasia. The characteristic brain lesions corresponding to global aphasia are in the left hemisphere, and usually of a fairly large extent. In case focal damage can be determined, it is usually found on the edges sylvian fissure together with the surrounding cortex.

The probably best known type of aphasia is *Broca's aphasia*. This is, of course, due to the historic role of Broca, who first linked aphasiac symtpoms to specific forms of brain damage. The prominent symptoms are non-fluent spontaneous speech, low output rate, and articulatory difficilties. Here a brief example from a patient describing what brought him into the hospital:

> Yes ... ah ... Monday ... ah ... Dad and Peter Hogan, and Dad ... hospital ... and ah ... Wednesday ... Wednesday nine o'clock and ah Thursday ... ten o'clock ah doctors ... two ... two ... an doctors and ... ah ... teeth ... yah ... And a doctor an girl ... and gums, an I.[4]

One further observes segmental substitutions and the disruption of normal intonational patterns. Utterances tend to be very short and agrammatical. The style of utterances is often called telegraphic, because it mainly uses nouns and other content words, but omitts functional words. This hints towards an impairment

[3]Rüdiger Vaas, 'Ein Blick ins Lexikon des Gehirns', *Spectrum der Wissenschaft*, November (1996) 24-32

[4]Steven Pinker, *The Language Instinct: how the Mind creates Language*, (William Morrow and Co. Inc., New York, 1994) p. 307

within the grammatical processing. The brain damage is usually concentrated to a certain region in the left frontal lobe, which now carries Borca's name. This type of aphasia is a good example where the specific grammatical nature of the symptoms has been used to conclude that Broca's area is responsible for syntactical processing. The observation that auditoral comprehension seems to be often less affected may according to Pinker be explained by the fact that "comprehension ... can often exploit the redundnacy in speech to come up with sensible interpretations with little in the way of actual parsing." [5] This is an important point, since often one specific impairment can be compensated for by making extensive use of other unaffected faculties. Pinker also notes that some grammatical abilities survive damage of Broca's area, which contradicts the claim that this area is our grammar function module. It must therefore be said that the precise function of Broca's area is still unclear, and Pinker suggests that "perhaps the area underlies grammatical processing by converting messages in Mentalese into grammatical structures and vice versa."[6]

Another well known type is *Wernicke's aphasia*, which is again named after the man who found a link between specific symptoms and focused brain damage. Wernicke's aphasia is in some ways the complement of Broca's aphasia: The speech is fluent with an high, even abnormally high, word-output (logorrhea) of normal phrase length and well controlled intonation. The grammatical structure is more or less intact, but the speech is perceived as lacking specific content words, being more or less empty of transmitted information. Neologisms and word substitutions are numerous, combined with anomiac symptoms, and Pinker observes that such patients show few signs of comprehending the speech around them.[7] Again a brief example, where a patient answers the question what brought him to the hospital:

> Boy, I'm sweating. I'm awful nervous, you know, once in a while I get caught up, I can't mention the tarripoi, a month ago, quite a litle, I've done a lot well, I impose a lot, while, on the other hand, you know what I mean, I have to run around, look it over, trebbin and all that sort of stuff.[8]

The overall picture of the symptoms suggests that this type of aphasia constitutes a semantic disorder which affects both, input as well as output. This clearly distinguishes Wernicke's aphasia from anomia and Broca's aphasia, where comprehension is still sufficiently preserved. Pinker proposes that "Wernicke's area seems to have a role in looking up words and funneling them to other areas, notably Broca's, that assemble or parse them syntactically."[9] The site of the typical

[5]Pinker, p. 308
[6]Pinker, p. 310
[7]Pinker, p. 311
[8]Pinker, p. 310
[9]Pinker, p. 311

lesion is placed in the superior temporal lobe close to the temporal-parietal junction and extends into the infolded surface from the sylvian fissure. However, damage might be even wider spread in more severe cases. One further important distinction between Wernicke's aphasia and Borca's is that Wernicke patient are not aware of their inability of comprehension as well as the meaninglessness of their outout, while sufferers of Broca's aphasia are fully aware of their linguistic impairments. This, and Pinker's observation might suggest that the brain damage specific for Wernicke's aphasia affects in particular the internal communication as compared to the disruption of a specific function in Broca's aphasia. This, however, is a general and very difficult and by no menas self-evident question relevant for all sorts of aphasia.

Next, I discuss the so-called *conduction aphasia*. This is an even more stiking example of the effects that the disruption of connecting 'communication lines' has. Conduction aphasia seems to be caused by the damage of a very sepcific and highly focused region which connects Broca's area with Wernicke's. Many symptoms are not unlike Wernicke's aphasia with, however, more interuptions in free speech, presumably due to word-finding difficulties, and slightly less quality in intonation. But there is one very specific symptom which characterises this syndrom, namely the severe impairment of the ability to repeat spoken words or sentences. In some patients, this contrasts to the fact that the same ability with respect to written words remaines to some extent intact, underlining the highly specific nature of this symptom. This led to several suggestions about the organization of the transfer of linguistic input to output and vice versa which, however, remain to date inconclusive.

These four types of aphasia (excluding the global one) constitute the group of aphasiac syndroms that are commonly associated with focal lesions in the language are (the perisylvian region). The group of syndroms I will discuss next are usually linked to brain damage focused on the borders of the perisylvian region. Given the standard neurological model that the perisylvian region is indeed the main processing region for language, this would indicate that the following forms of aphasia are more related to impairment of connections in to or out of the language processing region.

The first member of this new group is the so-called *transcortical motor aphasia* (TM). The symptoms are broad and not unsimilar to Broca's aphasia. However, grammar is not the main issue, and patients with TM are able to correct grammatical errors in provided test sentences. The main difficulty for them is the production of speech itself, with non-fluent spontaneous speech and in particular problems in initianing articulation. Output may be repetitive or stuttering, but patients react well to sound-structure prompts and contextual hints. The impairments seem to be due to sequencing difficulties in input and output, in particular when modality changes are iinvolved (e.g. speech to writing). The similarity to Broca's aphasia makes diagnosis difficult, and there are even cases

of Broca's aphasia which during recovery pass through a state of TM. Further complications in asserting and diagnosing TM arise from a loss of the contrast between affirmative and positive (for example in response to yes/no questions) as well as uncontrolled non-verbal repetitions of answers which make the assessment of comprehension difficult.

In the same way as Wernicke's aphasia might be seen as the complement to Broca's, the so-called *transcortical sensory aphasia* (TS) is somehow the complement to TM. Speech is fluent with many paraphasias and neologisms. The main difference to Wernicke's aphasia is that repetition abilities have been spared, and it is striking that TS patients tend to faithfully repeat everything the examiner has said (echolalia) to such an extent that it must be viewed as a defect. As with Wernicke's aphasia, auditory comprehension is impaired, and, analogous to TM, a difficulty with yes/no questions can be observed. Also, the echolalia and lost comprehension lead to the fact that nonsense sequences are repeated as faithfully and readily as meanginful ones. The TS symptoms, in particular the echolalia, distinguish it clearly from Wernicke's aphasia, caused by a – within a limited span – differentially spared auditory repdroduction.

The most severe aphasia of this group is the so-called *mixed transcortical aphasia*, which is a combination of both former types. Although Broca's area, Wernicke's area and their connections seem to be intact, they are completely cut of from the rest of the cortex like an island (isolation syndrom). These patients never speak spontaneously and the only form of observed speech is echolalia without any hint of comprehension. As with global aphasia, brain damage is usually wide spread. However, patients show signs of non-verbal comprehension, especially in response to written language. It is not clear, whether this is achieved on the level of graphem/phonem conversion or with the help of a more direct access to visual faculties, or with a mixture of both.

For completeness, I also mention the so-called *Pure Word Deafness* as discussed by Pinker:[10] This rather strange phenmenon does not affect reading and speaking, but just the understanding of spoken words. Although patients clearly can hear environmental sounds in a normal way, "words are as meaningless as if they were from a foreign language."

There is one more group of linguistic impairments which I would like to discuss. These encompass *alexia* and *agraphia*. The former concerns reading, the latter writing, and both mark a distinction in input versus output. Both are commonly divided into two subcases, depending on whether the site of lesion is frontal or parieto-temporal. It is striking that the overall symptoms for frontal alexia and agraphia are in correspondence to each other and align themselves to Broca's aphasia, while the symtpoms for parieto-temporal alexia and agraphia, again in mutual correspondence, align themsevles to Wernicke's aphasia or forms of anomic aphasia. It should be noted that historically the main focus in the description and definiton of aphasiac syndroms was towards disorders where speech

[10] *Pinker, p. 313*

is primarily affected. This reflects the belief that speech is somehow more central and more basic than abilities as reading and writing. However, modern psycholinguists tend to test all modalities of language. There even are indications that for example alexia may be related to impairment of the phonological processing, as discussed by Shaywitz.[11]. According to this model, speech impairment is simply less obvious, since it may be better compensated by semantic means of comprehension. Thus, modern psycholinguistics offers us the view that the brain does not so much distinguish between different modalities of language, but treats the common universal structures on an equal footing. This is supported by the fact that a completely alien language system, namely the sign languages of the deaf, seems to have precisely the same mental representation and organization as a spoken language. A very nice account of this may be found in Sacks' book,[12] which also notes that deaf people may suffer from virtually identical forms of aphasia with respect to their sign language. This is also observed by Pinker,[13] and fits his theory that our brain represents language universally in its own Mentalese relatively independent of the concrete ways of language input and output.

The division of the types of aphasia into groups as presented above encodes more than one hundred years of research. It is an attempt to group these disorders with respect to their main level of linguistic impairment such as lexicon, grammar or message comprehension and to align this with the pathological findings of neuro-physiologists suggesting a certain amount of localisation of these linguistic facilities. However, even after one hundred years, this can only be seen as a very preliminary attempt. The full statistics of aphasic patients presents a much less clear cut ensemble than the above descriptions might suggest. For example, in tne percent of the cases with a Wernicke area lesion, Broca aphasiac symptoms occur, and vice versa. Also, Pinker reports[14] that the neuro-surgeon George Ojemann eletrically stimulated highly localised (a few millimeters across) different sites in the brain of concious people and found that he was able to disrupt a single function like repeating or completing a sentence, naming an object or reading a word. However, these sites were widly spread with the main bulk of the distribution within the perisylvian regions, but unfortunately not the whole distribution within it. Even worse, different people showed different sites for the same function. A very surprising result in this direction is a strong gender specific difference in the localisation of the phonological decoding while reading words. In male persons it is solely concentrated in the gyrus frontalis of the left hemisphere (where also Broca's area resides), while female persons also activate the right gyrus frontalis.[15] This might explain why female sufferes of a left-sided

[11] Sally E. Shaywitz, 'Legasthenie – gestörte Lautverarbeitung', *Spektrum der Wissenschaft*, January (1997) 68-76

[12] *Oliver Sacks,* Seeing Voices: Journey into the Worlf of the Deaf*, Harper Perennial: New York, 1990*

[13] Pinker, p. 302

[14] Pinker, p. 315

[15] Shaywitz, p. 74

stroke often show less severe forms of linguistic impairments, and why there are statistics that girls are less prone to dyslexia than boys. As Pinker points out:

> A very gross anatomy of language sub-organs within the perisylvian might be: front of the perisylvian (including Broca's area), grammatical processing; rear of the perisylvian (including Wernicke's and the three-lobe junction), the sounds of words, especially nouns, and some aspects of their meaning. ... There are not smaller patches of brain that one can draw a line around an label as some linguistic module – at least, not today. But ... there must be portions of cortex that carry out circumscribed tasks, because brain damage can lead to language deficits that are startingly specific.[16]

These one hundred years of research were much driven by the debate of "localisationism", stating that language (and other) brain functions can be pin-pointed down to specific well defined regions of the brain. Modern imaging methods such as PET scans or NMRT pictures, which for the first time allowed us to see active regions of the healthy and living brain – in contrast to the deductions made from lesions of damaged brains after autopsy – show that the situation might be far from being that simple. For Pinker, it is conceivable that "Some kinds of linguistic knowledge might be stored in multiple copies, some of higher quality than others, in several places."[17]

Therefore, an interpretation of aphasiac syndroms in linguistic terms faces two main obstacles: Firstly, even a seemingly simple task of naming an object involves many mental functions such as recognizing, looking up its entry in the mental dictionary, accessing its pronuncation, articulating it, and perhaps also monitoring the output for errors by listening to it. Obviously, a naming problem could arise at any of these stages. Secondly, the link between a deficit and its manifastations may be highly complex, giving rise to more than one manifestation in unexpected parts of the language system. Linguists and psychologists try to overcome these problems by highly sophisticated tests involving, to name just one example, tasks like pluralisation of fake-words. However, even the most sophisticated tests have their limits since they are constructed according to certain linguistic or psychological models, and hence always superimpose their assumptions onto the measurements to an unavoidable degree – a well know fact in the theory of statistics: As long as we don't know how the brain works, our tests will possess intrinsic shortcomings, and if we do know how the brain works, we no longer need tests.

To illuminate the range of difficulties of a linguistic interpretation, I will consider one paricular example, namely the adult acquired reading disorder following brain

[16]Pinker, p. 313
[17]Pinker, p. 316

damage. One distinguishes two patterns, the so-called *deep dyslexia* and the so-called *surface dyslexia*. The former often produces errors semantically similar to the stimulus word, e.g. 'father' is read as 'Dad', 'Christmas' becomes 'turkey'. If the error is of semantical nature, it is often preceeded by a faulty visual perception of the stimulus word, such as 'sympathy' is read as 'orchestra' via the perception as 'symphony'. In contrast, surface dyslexia produces phonologically similar errors to the stimulus words, which even might result in non-lexical, i.e. non-existent, words. Semantic misreadings are a consequence of the errorneous phonologic perception, e.g. 'begin' is phonologically perceived as 'begging' and hence understood as 'collecting money when you ask someone'.[18] This latter form is quite similar to dyslexia as observed in children.

Both forms of adult dyslexia share the property that they seem to porduce semantic misreadings. From the superficial diagnostic findings one might conclude that dyslexia is due to a semantic disability. However, dyslexic persons often exhibit strong if not exceptional good semantic performances (as mentioned above, because they are well trained in compensating for their disablities on this level). Deeper research reveals that the problem has to do with the access of the mental lexicon. And here it gets complicated again: There exist several competing theories on how the mental lexicon is accessed. Dyslexia may be fairly well explained by models proposing a graphem/phonem conversion such that the phonetic equivalent of a read word is the real input for the mental lexicon matching process. The deep dyslexic patient, however, cannot use this path due to a blockage of this conversion process. He/she has to use other means of access, such as a direct access via the global visual perception. Direct access does not allow for a point-by-point analysis of the input so that the exactly matching word cannot be found. Since the relevant semantical field has been activated by the (possibly incomplete and errorneous) global visual perception, another word out of this activated field may come up. The seemingly semantical disorder turns out to be a result of the impairment of the graphem/phonem route. Thus, some researchers call it phonemic dyslexia. In surface dyslexia, the graphem/phonem route is preserved, but faulty. This accounts for the typically phonological errors of these patients that often sound like semantic errors when they are asked to explain the meaning of the stimulus words.

Although this analysis sounds fairly plausible, it is full of implicit and not strictly proven assumptions. For instance, this interpretation makes heavy use of theoretical models on access to the mental lexicon, e.g. the direct access model whose construction is based on knowledge of simple pattern recognizing neural nets (which in turn were designed to explain visual perception). Also, the model of graphem/phonem correspondence rules, underlying the above interpretation, is still a proposal, known as the phonological recording hypothesis[19] which, however,

[18] Examples taken from Garman, p. 545

[19] Alan Garnham, *Psycholinguistics: Central Topics*, (Routledge: London, New York, 1994) p. 57

recently received some experimental support through the research on dyslexia in children.[20]

Another problem of this kind is *agrammatism*, as it is found most prominently in Borca's aphasia and TM. Traditionally, agrammatism has been understood as a loss of grammatical functioning. Although it has been recognized that not only grammatical but also phonologica factors might be involved in the impairment (remember that due to recent NMRT studies, phonological decoding takes place in virtually the same brain region where Broca's area resides). As an alternative hypothesis Kean[21] suggested that a single phonological deficit could account for all the characteristics of agrammatism. According to Kean "the manifested linguistic deficits of Broca's aphasia can be accounted for only in terms of the interaction between an impaired phonological capacity and other intact linguistic capacities." In her opinion, a range of different grammatical structures may converge on unitary phonological forms. The phonem /boys/, for instance, is the same for the three terms 'boys', 'boy's' and 'boy+is'. Therefore, if a Broca aphasic says 'boy play grass' instead of 'the boys are playing on the grass', this might not be due to omitting the grammatical structuring function words or word-suffixes, but due to omitting simply all elements, which are not themselves words but are attached to words. These elements are called clitics by Kean. Note that according to Kean determiners are treated as clitics and not as function words by Broca patients. This phonological approach towards Broca's aphasia is, of course, not free of problems and controversy. Without going into more details, the important lesson can be drawn that agrammatism is still a challanging issue. The phonological hypothesis is at least able to explain some of the effects that were previously attributed to agrammatism. In response, grammatical approaches have been updated and refined. Nevertheless, a basic issue still awaiting explanation is the often noted co-occurence of agrammatism and impaired articulatory sequencing. One might add one further remark that according to Chomsky's approach,[22] language is understood to be made out of a hierarchy of building units, starting from the phonems, and sets of rules yielding the admissable composite building blocks of the next level in the hierarchy. Such sets of rules are formal grammars, and from this point of view agrammatism is not essential different from phonological defects.

The above clitics hypothesis has also been disputed by scientists who relate the apparent agrammatism of Broca's aphasics to particular losses of certain parts of the mental lexicon (thus relating it to forms of anomia), which just happen to contain precisely the closed-class words which carry grammatical structure. Closed-class means that such a class of words resists its extension by new words, while open classes readily welcome new members. That closed-class words (or function words) are organized in a different way as open-class words (or content

[20]Shaywitz, p. 68-76, see also the remarks by John Horgan, ibid. p. 76
[21]as reported by Garman, p. 457
[22]Pinker, 21-24, 38-42, 84

words) can be inferred from measuring the access times of words in dependency of the general statistical distribution of their usage frequencies in ordinary speech. While open-class words are accessed the faster the more common they are, closed-class words are all accessed in approximately the same time. The mental lexicon approach agrees with the clitics hypothesis insofar, as the closed-class words not only are the ones which carry syntactical structure, but at the same time are the ones which are phonologically 'uninteresting' or insignificant.[23] The approach via the mental lexicon traces the high rate of absence of verbs in the speech of Broca aphasics back to a similar loss of part of the mental lexicon. Verbs again are different from normal content words, since they also intrinsically carry syntactical structure. However, it remains an open question whether the impaired retrival of verbs is the cause or the effect – a general question applicable to most of linguistic interpretations of aphasiac speech.

Unfortunately, we are not yet finished with Broca's aphasia, since there still remains the linguistisc interpretaion due to cognitive linguistics. Deane[24] proposes a construction of syntactic structure according to four fundamental links, the R-link, involving referential restrictions; the C-link, involving co-occurence restrictions; the P-link, involving predication; and the S-link, involving modification of lexemic and conceptual meaning. After revieweing recent clinical evidence stating that Broca aphasics have lesions in more extended brain regions than Broca's area alone, and that the latter is not an area for syntactic processing but a motor center, responsible for programming movements in the face, mouth, and throat, he concludes that his cognitive model is in agreement with the clinical evidence and that therefore the agrammatism in Broca aphasia is mainly caused by patterns of synactic disturbances attributable to the absence of R-links. Broca aphasics avoid such words which depend on R-links, since their concept of R-link is destroyed due to cerebral damage. The main difference of this model to the above mentioned ones is that the syntactical structure is transferred away from a processing brain region and towards a manifestation in form of communication tracks directly corresponding to the different categories of links. Therefore, Deane proposes an alternative to the (strictly) modular and autonomous approaches to the question of universal grammar (Pinker and Chomsky call such a grammar generative grammar). He emphasizes the importance of interconnections within the brain, ultimately and implicitly taking a more holistic point of view than the other discussed approaches. His model would imply strong connections between the human language faculty and general properties of human cognition, i.e. between linguistic and non-linguistic modes of thought. But what this quite recent cognitive linguistics approach also shows is that even very old questions as on the modularity versus holistic organization of the brain are far from being dealt

[23]For an exposition of the mental lexicon approach see Jean Aitchison, *Words in the Mind: an Introduction to the Mental Lexicon*, (Balckwell: Oxford, Cambridge (MA), 1994), in particular pp. 106-109

[24]Paul D. Deane, *Grammar in Mind and Brain: Explorations in Cognitive Syntax*, (Mouton de Gruyter: Berlin, New York, 1992), pp. 281-293, 299

with.

Grammar and its corresponding deficits is also an issue in Wernicke's aphasia, where traditionally so-called *paragrammatism* is diagnosed. This means that the usually hyper-fluent speech of such a patient does possess all the grammatical function words and suffixes (as opposed to Broca's aphasia), but in an incorrect way. However, when a Wernicke aphasic is tested together with a normal test subject, many features of their speech are similar. The differences are firstly a poor turn-taking ability of the aphasic, derived from an impaired self-monitoring, and secondly much unintelligibility, most of which is linked to noun-based neologisms instead of entity nouns, hence constituting a defect with respect to the mental lexicon rather than to specific grammatical functions. The deployment of other lexical elements and structural patterns, however, seems to be not different from the one of a normal person, i.e. paragrammatism could not be found. This might be a singular event of this particular experiment, but it emphasizes the importance of a comparison between aphasiac language and suitable control data, "if untoward assumptions about the 'ungrammaticality' of spontaneous-speech samples are not to cloud the issue."[25] It might be worthwhile to note in this context that it is also quite non-trivial to obtain precise data of aphasiac speech, because many aphasics are only tested at a time where first steps of recovery have been taken.

This final remark also hints to another question. Linguistic impairment has often been seen as a breakdown of language proceeding in the reverse order as language was first acquired during childhood. This is called the *regression hypothesis* "variously associated with Ribot, Jackson, Freud, and most recently, Jakobson. [Although] this hypothesis has exerted a great deal of intellectual appeal in the history of aphasia research," mainly from the psychological direction, it does not withstand close scrutinity. Of course, comprehension of syntactically complex sentences develops late and is particularly vulnerable in aphasia, but this is only a very superficial point of view. Children acquiering language, and aphasics fighting for their recovery, have a different degree of awareness of their problems, have to deal with different obstacles, and use different strategies. Even more fundamental, the different clinical forms of aphasia with their corresponding specific sites of brain lesion resist alignment with levels of language acquisition. For example, semantic intentions seem to appear prior to adequate syntactic expressions in the development of language acquisition. From this does not follow that the telegrammatic style of Broca aphasiac speech, which at least can be understood, is at a more regressed stage than the grammatically elaborate and complex but meaningless speech of a Wernicke aphasic.[26]

[25] Garman, p. 463

[26] See the essay collection *Language Acquisition and Language Breakdown: Parallels and Divergencies*, edited by Alfonso Caramazza and Edgar B. Zurif, (Johns Hopkins University Press: Baltimore, London, 1978), citation from pp. ix-x

Returning to paragrammatism, cognitive linguists like Deane[27] do not hold with the above mentioned view that it might not exist, but suggest that, in his particular model, the interpretation of C-links is compromised which leads to a violation of constraints on syntagmatic combination in turn leading on the syntactic level to paragrammatism. Claiming that Wernicke's area is presumably responsible for auditory analysis of speech and for storing and recalling the auditory forms of words, Dean proposes that the general pathology of Wernicke's aphasia is mainly due to a reduction of the processing capacities of the system resulting in a general lowering of auditory acuity. Compared to the traditional view, Wernicke's aphasia appears not as a semantic impairment, but as a phonological disorder, once more demonstrating the wide variety of possible linguistic interpretations of the same aphasiac symptoms.

I hope that I was able to demonstrate the complexity of linguistic consequences of cerebral damage and the difficulty to assess the precise linguitic impairment due to a brain lesion with the few examples I have discussed here, particularly Broca's aphasia and the different interpretations of its agrammatism. Part of this difficulty stems from the process how we gain our knowledge, which proceeds in steps and circles, e.g. by defining a certain form of aphasia by its apparent linguistic symptoms, which then in turn have been used to associate corresponding mental functions with the damaged brain regions. As in most empirical sciences, knowledge can only progress by trial and error, and continuous testing of hypotheses in experiments. However, what makes linguistics so difficult is the fact that good experiments are extremely tricky to design. I want to conclude with a phrase by Wilhelm von Humboldt anticipating Chomsky: language "makes infinite use of finite media". There is no limit to the number of possible sentences we can communicate. Therefore, the concept of infinity, and hence complexity, is inherent to language. It seems then only natural that our understanding of what language is and what its relation to our mind, is nothing more than first glimpses. Although modern science already has started to probe language and the mind by other means than language itself, and although empirical evidence from PET scans and NMRT images gives us some feeling for how the brain works, the infinitude of language will elude us for quite a time to come. Although we may learn a great deal from the language of aphasic persons about language in general, we become aware at the same time how difficult it is to grasp the complexity of language in linguistic terms. It might very well be that all we ever will achieve is Gödel, Escher, Bach – an eternal golden braid.

[27] Deane, pp. 294-295

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