

# Syntax and Language Disorders

Martina Penke (University of Cologne, May 2011)

## 1. Introduction

Syntactic deficits are common in language disorders and have always been at the focus of research on language disorders. The investigation whether or not syntactic deficits occur in a given acquired or developmental language disorder, which syntactic structures or processes are eventually affected and how to capture such deficits in an explanatory theoretical account has dominated the linguistic research on language disorders since its very first beginnings to the present.

Interest on syntactic deficits in language disorders first focussed on *Broca's aphasia* – an acquired language disorder caused by strokes affecting left frontal brain regions. The core symptom of Broca's aphasia is an *agrammatic* spontaneous speech production which is characterised by omissions of free functional elements and a severe reduction of the length and syntactic complexity of utterances. This leads to a preponderance of very short utterances that seem to consist of a simple, linearly organised string of open-class words. As the term agrammatic already indicates, the spontaneous speech of language impaired speakers suffering from this disorder gives the impression of lacking syntactic structure. Agrammatic Broca's aphasia was therefore considered the ideal candidate for investigating syntactic deficits and has been at the focus of linguistic investigations concerned with syntactic disorders ever since the advent of modern generative syntactic theory (Chomsky 1957)

According to Chomsky's generative approach, the human language faculty is due to a specialized, domain-specific language organ situated in the brain that is part of our biological endowment and therefore genetically specified (e.g. Chomsky 1980, 2002). If this conception holds true, the language organ in the human brain should be affected by brain lesions involving this organ. An impairment of this organ due to a brain lesion should consequently result in an impairment of the language faculty. Agrammatic Broca's aphasia seemed to exemplify this theoretically predicted case of an impaired language faculty due to brain lesions. Consequently, from the late 1960's to the late 1980's the linguistic investigation of language disorders associated with syntactic deficits was predominantly concerned with the language deficits observed in Broca's aphasia, the focus of this research being on the issue whether or not Agrammatism is due to a deficit affecting syntactic competence and how to capture this purported syntactic-competence deficit in a syntax-theoretical framework.

The interest on agrammatic Broca's aphasia was also furthered by the assumption that Wernicke's aphasia constituted a syndrome mirroring the deficits observed in Broca's aphasia. Individuals with Wernicke's aphasia typically suffer from lesions to temporo-parietal brain regions and display marked language comprehension deficits and impairments in word retrieval. These symptoms were considered as indicative of a lexical-semantic deficit in Wernicke's aphasia, whereas syntactic abilities were assumed to be intact (cf. Marin, Saffran, and Schwartz 1976 and the overviews in Edwards 2005 and Wimmer 2010). The assumed dichotomy between intact lexical-semantic and impaired syntactic abilities in Broca's aphasia and spared syntactic but affected lexical-semantic capacities in Wernicke's

aphasia was taken as evidence for a dual architecture of the language system encompassing a storage component (i.e. the lexicon) and a computational component (i.e. syntax) that contains the operations to generate composite hierarchical structures such as phrases and sentences out of the stored elements in the mental lexicon (Chomsky 1965; Pinker 1999).

In the 1990 then, the discovery of the British KE family, a huge three-generational family in which about half of the members suffer from an inherited speech and language disorder (Gopnik and Crago 1991), drew the attention to developmental language disorders, specifically to the language disorder called *Specific Language Impairment (SLI)*.<sup>1</sup> Children with Specific Language Impairment display severe problems in acquiring inflectional morphology, verb movement and complex syntactic constructions such as relative clauses or passives. If the language capacity is innately specified, as suggested by Chomsky's generative approach, this capacity should in principle be susceptible to genetic disorders which affect the genetically encoded blueprints controlling the development and functioning of the brain areas that subserve this language organ. Indeed, in 2001 a point-mutation of the human gene *FOXP2* (located on the long arm of chromosome 7) was found to be related to the inherited speech and language disorder affecting the members of the KE family (Lai et al. 2001). The findings suggest that the mutation of this gene leads to an atypical development of brain areas typically associated with speech and language (Liégeois et al. 2003). This was the first case attested where a genetic defect could be directly correlated to neuro-anatomical abnormalities which in turn are held responsible for certain speech and language functions. *FOXP2* was therefore enthusiastically celebrated as the first 'language gene' to be discovered.

Although further genetic investigations have revealed that the point mutation discovered by the KE family is not regularly observed in other individuals with Specific Language impairment (e.g. SLI Consortium 2002; Meaburn et al. 2002), the evidence suggests that Specific Language Impairment has a genetic basis (cf. SLI Consortium 2002; overview in Stromswold 2001; Bishop 2009). This nourishes the hope that it might be possible to explore the genetic basis underlying the specific syntactic deficits observed in this syndrome, thereby ultimately uncovering those aspects of the language capacity that are genetically specified in our species.<sup>2</sup> Moreover, SLI is considered to display an interesting dissociation between impaired language and spared general cognitive capacities, a dissociation that has been argued to exemplify that the human language capacity is independent from other cognitive domains as assumed in the framework of Generative Grammar (e.g. Chomsky 1957, 1980) and cannot be put down to the operation of domain-general cognitive operations (see e.g. Levy 1996; Van der Lely 1997, 2005). The deficits associated with this developmental language disorder have hence attracted much research on syntactic deficits over the last 20 years.

Meanwhile research on syntactic deficits has also taken other language disorders as seen in Down's syndrome, Parkinson's syndrome or Autism into focus. Today, there is hardly any acquired or developmental deficit syndrome associated with language impairments for which a deficit affecting the syntactic domain is not discussed. Such syndromes include developmental disorders such as Specific Language Impairment, Williams syndrome, Down's syndrome or Autism, and acquired language deficits such as aphasic language disorders (Broca's aphasia, Wernicke's aphasia) or degenerative brain diseases (Parkinson's disease, Alzheimer's disease, Huntington's disease).

The wealth of research that has been performed in this area up to today precludes any in-depth discussion of the investigations conducted and the discussions led – present and past – for every single language disorder. I will, therefore, try to single out some major issues that have shaped the field during the last 40 years. Controversies with respect to syntactic deficits have centred on the question whether or not the symptoms related to a given language disorder are due to a deficit affecting syntactic competence and if so, how to capture this deficit in a theoretical framework. Many issues first arose with respect to Broca's aphasia, the first language disorder to attract linguistically-based research, and therefore, research on Broca's aphasia will be at the focus of this article. Nevertheless, the article tries to achieve a broader coverage by referring to related research conducted on other language disorders when possible and by taking up issues and findings that are specific to single language disorders. In addition, the appendix gives short descriptions of the most relevant language disorders that have been associated with syntactic deficits, sketches the relevance of these disorders for the field and gives references to publications where the reader can find more detailed information on the respective syndromes.

### **1.1. Why investigate syntactic deficits?**

In investigating language disorders the aim is twofold. A first goal is to describe and explain which aspects of the language faculty are impaired or retained in a given language disorder, i.e. we want to learn about the impaired language system. Such knowledge could then provide the basis for effective therapeutical intervention. Most research in language disorders has naturally concentrated on this goal. However, although the task is set out precisely enough in theory, accomplishing this task has turned out to be quite intricate in practice. To this day, there is not a single language disorder for which a general consensus regarding the nature of the observed language deficits has been obtained among researchers (see section 2.).

A second goal of the investigation of syntactic deficits is to learn about the structure of the human language capacity by investigating cases where this capacity seems to break down. Here, the idea is that a thorough study of the syntactic deficits associated with language disorders might provide insights into the structure and organization of the normal language system. Since Chomsky (1957) introduced the generative approach into the field of linguistics, the subject matter of investigation for a generative linguist is the abstract linguistic knowledge of an individual, i.e. her/his language competence or I-language. This knowledge is accessed when we produce respectively comprehend utterances or when we judge their grammaticality. The application of this abstract system of knowledge in language production, comprehension or judgement results in performance data. The abstract system of grammatical knowledge is not directly accessible to investigation, it remains a 'black box'. Insights in our language competence can only be gained by the analysis of performance data reflecting the application of this knowledge, i.e. when we produce or comprehend speech utterances or judge their grammaticality. Note, however, that grammatical competence is only *one* factor determining performance, with other factors such as e.g. processing capacities, social, pragmatic or discourse factors affecting it, too. As linguistic competence is a 'black box' which does not allow for direct observation, the crucial task for generative linguists is to find evidence for the content of this 'black box' within performance data. The deficits observed in individuals with language disorders constitute one type of performance data that can be used to draw inferences about language competence. The value of erroneous forms was probably first noticed in speech-error research. A linguistic investigation

of speech errors revealed that such errors were not random, but that they were constrained by the architecture of the language system and only involved elements of the same category and level of representation (Fromkin 1971; Shattuck-Hufnagel 1979). A similar logic applies to erroneous forms produced in language impairments, the idea being that we can learn about the human language faculty by investigating what factors constrain the errors that occur in language impaired individuals (cf. Fromkin 1997 for overview).

The assumption that the investigation of syntactic deficits can provide insights into the structure of the human language faculty rests on four axioms on the nature of the human language capacity and its breakdown following brain lesions. According to the assumptions of *autonomy* and *modularity* (Fodor 1983) the human language faculty is autonomous from other cognitive domains or modules (i.e. it is domain-specific) and consists of task-specific and independent submodules carrying out different types of computations on different types of representations. According to the *fractionation* assumption (Caramazza 1984) brain damage can result in the selective impairment of specific submodules of the language capacity. The *transparency* assumption (Caramazza 1984) states that the components unaffected by the brain lesion will continue to function as normal such that the output of the language system will directly reflect the impairment of the affected component. The function of the impaired module in the unimpaired language system can, then, be directly inferred from the symptoms observed in the language impaired individual. Although the validity of these assumptions has been controversially debated (see Kosslyn and Van Kleeck 1990; Caramazza 1992; Levy and Kavé 1999; Penke 2006: §2.3 for discussion), they nevertheless have proven very fruitful in linguistic investigations of language disorders.<sup>3</sup> The major justification for adopting these axioms comes from the observation that results obtained from the analysis of language deficits have been supported by converging results coming from theoretical linguistics and from data obtained with other psycho- or neurolinguistic methodologies (cf. Caramazza 1992; Penke 2006: §2.3).

In pursuing the goal to draw inferences about the normal language faculty by investigating language disorders, the investigation of *selective deficits*, i.e. of deficits that selectively affect an entity X but spare a different entity Y, has drawn particular attention. The observation that a language disorder affects linguistic entity (i.e. an element, structure or operation) X while sparing others is seen as evidence that the affected entity is represented in the mind/brain in a way that is distinct from entities that are unaffected by this language disorder. For example, it has been suggested that agrammatic Broca's aphasia is due to a syntactic deficit sparing the semantic component, whereas Wernicke's aphasia might be associated with a deficit of the semantic component sparing syntax (cf. Marin, Saffran, and Schwartz 1976).<sup>4</sup> The double dissociation of the deficits observed in these two aphasic syndromes was hence regarded as evidence for the view that the language faculty consists of two autonomous modules – a semantic and a syntactic component – that can be selectively affected in language disorders.<sup>5</sup>

Moreover, data from language impaired speakers might also serve to evaluate competing linguistic theories. Out of a set of competing theories the one that captures the observed language deficits best, i.e. the theory that allows for representing preserved and impaired structures or functions as natural classes within the theory, is to be preferred as it has the property of *breakdown compatibility* (Grodzinsky 1990: 17). Consider as example two linguistic constructions X and Y which are considered to be qualitatively different by theory A, while theory B assumes that the same mental representations and/or operations underlie constructions X and Y. In this case, the observation that construction X is impaired in a

specific deficit syndrome while construction Y is unaffected can be considered as providing evidence for theory A which assumes that X and Y belong to different natural classes within the theory. In contrast, the observed selective deficit is incompatible with theory B which argues that constructions X and Y belong to the same natural class and should hence be equally affected by the language disorder.

With respect to exploring the human language faculty, a further goal of investigating language disorders is to find out about the localisation of specific grammatical capacities in the human brain. By exploring the correlation between specific language deficits and the localisation of the brain lesions causing these deficits, the hope is to identify those brain areas that critically subserve a specific grammatical structure or operation. Whereas modern imaging techniques such as PET and fMRT indicate all brain areas active during a given cognitive operation, only lesion studies allow for identifying those brain areas whose functioning is critical for performing this cognitive operation. A lesion of such a brain area should necessarily result in a deficit of the cognitive operation that critically depends on the normal functioning of this brain area (Rugg 1999). In practice, however, factors such as the variability of lesion sites across individuals diagnosed with a specific disorder syndrome (e.g. Basso et al. 1985; De Bleser 1988; Willmes and Poeck 1993), the neuroanatomical variation between individuals (e.g. Uylings et al. 1999) and the variability of language deficits observed in individuals with similar brain lesions (cf. section 3) pose a challenge to this goal.

Summarising, the linguistic investigation of language deficits offers a fascinating route to find out about the nature, the localisation and the functioning of the human language faculty. Also the study of language deficits has been considered to serve as “a natural laboratory in which linguistic theories may be tested” (Levy and Kavé 1999: 138). Nevertheless, despite this potential, researchers have been very careful to advance theoretical claims on the basis of data from language impairments. Rather than making use of impairment data to advance syntactic theory, syntactic theories have been used to explain impairment data.

## **1.2. What are typical symptoms of syntactic deficits?**

A deficit affecting syntactic competence or the processing of syntactic structures in language performance has to be observable in language production and/or language comprehension. Whether or not a language impaired individual displays a syntactic deficit in language comprehension can only be revealed by explicitly testing an individual's language comprehension capacities. In contrast, symptoms suggestive of a syntactic deficit can in general quite readily be observed in the spontaneous-speech production of affected individuals and often require no explicit testing. Consequently, much research on syntactic deficits has concentrated on the signs of syntactic deficits that are observable in spontaneous-speech production.

### **1.2.1. Typical signs indicative of syntactic deficits in spontaneous-speech production**

While deficit symptoms in spontaneous speech may vary between different syndromes, research has revealed a surprising correspondence of such symptoms across different language disorders. Among the core symptoms indicative of syntactic deficits in spontaneous-speech production are the following:

- Problems with bound inflectional morphology are a core symptom in many language disorders. Depending on the morphological system of the respective language, such problems might lead to the omission or substitution of inflectional markers. Omissions of inflectional markers only occur if the remaining word stem is a possible word in the

respective language (such as *book* in \**two book* or *kiss* in \**she kiss*) (Grodzinsky 1984). Where the omission does not result in a possible word (e.g. Italian \**libr-* instead of *libri*<sub>[PL]</sub>, Russian \**knig-* instead of *knigi*<sub>[PL]</sub>), inflectional markers are substituted – often by unmarked forms such as the infinitive or the past participle form in verbs and the nominative form in nouns. Omissions and/or substitutions of bound inflectional morphemes are a characteristic symptom of agrammatic Broca's aphasia (Menn and Obler 1990a) and Specific Language Impairment (Levy and Kavé 1999) across languages. Similar problems have, however, also been observed in Autism (Tager-Flusberg 2002), in Down's syndrome (Laws and Bishop 2003), in children who have undergone hemispherectomy (Curtiss and Schaeffer 2005) and in Wernicke's aphasia (Kolk and Heeschen 1992) (cf. Penke 2008 for an overview on inflectional deficits).

- Free function words such as determiners, auxiliaries or conjunctions are often omitted. Such omissions characterise agrammatic Broca's aphasia (Menn and Obler 1990a) but similar omissions are also found in Specific Language Impairment (Leonard 1995), Down's syndrome (Eadie et al. 2002), Autism (Bartolucci, Pierce, and Streiner 1980) and in children who have undergone hemispherectomy (Curtiss and Schaeffer 2005).
- Sentence length is reduced in language disorders such as agrammatic Broca's aphasia (Menn and Obler 1990a), Specific Language Impairment (Leonard 1998), Autism and Down's syndrome (Tager-Flusberg et al. 1990). Especially in Broca's aphasia, spontaneous speech is often reduced to one- or two-word utterances which only contain the central open class words of the intended utterance. Wernicke's aphasia is, in contrast, characterised by an increase in sentence length due to the wild-running concatenation of sentence constituents and formulas that often results in long utterances devoid of meaning (cf. Edwards 2005).
- The produced utterances are of reduced syntactic complexity. In languages which allow for different word orderings, canonical word ordering is preferred to other word-order patterns. Thus, in German the canonical SVO order is preferred to the likewise grammatical OVS or XVS orderings in main clauses. Subordinate clauses are rarely produced in spontaneous speech, as are *wh*-questions or passives. This has been observed for agrammatic Broca's aphasia (Bates et al. 1988; Menn and Obler 1990a), Wernicke's aphasia (Bates et al. 1988; Niemi and Laine 1997), for Specific Language Impairment (Hamann, Penner, and Lindner 1998), Down's syndrome (Rondal and Comblain 1996) and for children who have undergone hemispherectomy (Curtiss and Schaeffer 2005).
- In languages with overt obligatory verb movement for finite verbs, verbs might appear in a non-finite form in main clauses and do not undergo verb movement. These structures are called root-clause infinitives. They regularly occur in agrammatic Broca's aphasia (Kolk and Heeschen 1992) and in Specific Language Impairment (Rice and Wexler 1996).

Whereas there is general agreement on the deficit symptoms that occur in spontaneous-speech production of a given language disorder, the relevance of deficit symptoms that have been identified in especially designed language experiments tailored by individual researchers to test a specific syntactic-deficit account regarding a specific language disorder are often discussed controversially among researchers. Controversies concern issues such as whether the observed deficits are characteristic symptoms of a specific syntactic language disorder that can be observed in every affected individual, or whether the experimental set-

up contains flaws in procedure, experimental material or subject choice that render the data invalid (cf. e.g. the discussions following the article of Grodzinsky 2000).

Nevertheless, carefully designed experiments that allow for a systematic collection of relevant data with respect to a critical syntactic construction are necessary to further our understanding of language disorders. A well-known limitation of spontaneous-speech data is that more complex syntactic structures are only rarely produced. However, absence of evidence is not necessarily evidence of absence. That is, we simply cannot know whether a specific form or construction is missing because the individual can no longer produce it due to her/his language deficit, or whether the form or construction is not showing up by coincidence, for example, because we just did not look at a large enough data set. A systematic collection of relevant data in an experiment allows for disentangling this issue. Comparisons between elicited and spontaneous-speech data have indeed illustrated that constructions or forms whose production poses a problem for a language-impaired speaker are often avoided in spontaneous-speech production and do only show up when the context provided in the experimental task requires the production of this specific form or construction (Kolk and Heeschen 1992; Penke 2006: §2.1.1).

A further advantage of experimental studies is that the researcher can control for all sorts of confounding factors that might influence the individual's language behaviour such as, for instance, word-form frequency and the length of sentences. Thus, language impaired speakers might experience problems with agreement or case inflection but correctly use inflected suppletive forms that have a very high frequency of occurrence (Penke 1998: 200–202, 211–212). Also, a syntactic construction might pose problems for a subject simply because it contains more words to produce, parse or remember than another less affected construction. In a carefully designed experiment such potentially influencing factors are controlled for. On the other hand, the control over the experimental material and the reaction of the tested individual is obtained at a price, namely the (sometimes highly) artificial situation which might also influence the individual's language behaviour (e.g. Heeschen and Schegloff 2003). Thus, experimental data has to deal with the critique that observed deficits might be due to the unnaturalness of the situation and might just constitute an artefact caused by the chosen experimental design. Hence, it is important to adopt different methodologies in experimental testing.

### **1.2.2. Typical signs indicative of syntactic deficits in language comprehension**

While symptoms indicative of syntactic deficits are readily observable in spontaneous-speech production, syntactic deficits in language comprehension are hidden from simple observation and can only be revealed by explicit testing. As mentioned above, experimental data are, however, susceptible to criticism targeting the experimental set-up. Thus, it can be argued that the number of items was too small to obtain reliable results or that confounding factors were not sufficiently controlled for. Moreover, whereas spontaneous-speech symptoms are generally seen as facts which are rarely disputed, the matter of syntactic comprehension deficits targets a theoretical issue that is related to the nature of the language faculty. The presence or absence of comprehension deficits is, therefore, hotly disputed. The controversy is on the following issue: Chomsky's generative approach to the human language faculty predicts that the language organ underlying this faculty should be affected by brain lesions leading to language disorders. A deficit impairing the syntactic competence of an individual should affect language production and language comprehension in parallel (Weigl and Bierwisch 1970) since the human grammatical competence is put to use in both, language

production and comprehension (i.e. in performance) (Chomsky 1980). Consequently, researchers who assume that syntactic disorders are due to a deficit in syntactic competence try to provide evidence for a deficit that affects all facets of language performance (language production, comprehension and judgement) in parallel. In contrast, researchers who assume that there is no such thing as syntactic competence, that syntactic disorders are due to modality specific processing disorders or that syntactic competence is basically intact in language disorders aim to show that language comprehension is not affected or, at least, not affected in parallel to language production.

A classic design in testing language comprehension capacities is the sentence-picture matching task where the subject has to match a given sentence such as *The boy is kissed by the girl* to one of two pictures: one depicting the action described in the sentence (e.g. girl kissing boy), the other depicting the reverse action (e.g. boy kissing girl). Studies on language comprehension capabilities adopting this or related designs have revealed that in languages with a canonical SVO word order, most language-impaired individuals display a better understanding of sentences with this canonical word order compared to sentences with a non-canonical word order such as object-clefts (*it is the boy who the girl kissed*), object relatives (*the boy who the girl kissed...*), passives (*the boy is kissed by the girl*) or object topicalisations where the object has moved out of its base-generated position and precedes the subject. The typical error in the comprehension of these constructions is that the first noun phrase occurring in the sentence is interpreted as the AGENT of the action. Whereas this does not lead to comprehension problems when the first noun phrase is indeed the AGENT, such as in active SVO sentences (*the girl kissed the boy*), subject cleft constructions (*it is the girl that kissed the boy*) or subject relative clauses (*the girl who kissed the boy ...*), problems arise when the first noun phrase is the THEME of the action. Then, for example, a passive clause such as *the boy is kissed by the girl* might be interpreted as *boy kissed girl*.

The English passive construction is certainly the best investigated construction in language comprehension studies. A good understanding of active sentences compared to an impaired understanding of passive sentences (e.g. *The boy is kissed by the girl* interpreted as *boy kissed girl*) has been observed in Broca's aphasia (Caramazza and Zurif 1976; Grodzinsky 2000) and Wernicke's aphasia (Bastiaanse and Edwards 2004), in dementia of the Alzheimer type (Grober and Bang 1995), in Autism (Tager-Flusberg 1981), Specific Language Impairment (Van der Lely 1996), Down's syndrome (Ring and Clahsen 2005), in children with early unilateral focal brain lesions (Dick et al. 2004) and in children who underwent a resection of the left brain hemisphere (Dennis and Whitaker 1976).<sup>6</sup>

These findings have, however, not gone unchallenged and heated discussions have especially ignited on the comprehension problems in agrammatic Broca's aphasia (cf. Martin 2006 for overview). For one, a subject-object asymmetry resulting from the strategy to regard the first noun phrase as AGENT of the action is also regularly observed in control subjects who do not suffer from any language impairment (cf. e.g. MacWhinney and Bates 1989; Ferreira 2003). Thus, this asymmetry and the underlying comprehension strategy do not seem to be specific for syntactic deficits in language impaired speakers. Also, there has been a fierce controversy on the issue of how to deal with individual language-impaired speakers who do not display the described comprehension problem. A comparison of language-comprehension abilities in a group of unimpaired control subjects and a group of language-impaired speakers will typically yield that the group of language-impaired speakers performs significantly worse with respect to the comprehension of non-canonical structures (such as

passives) compared to the group of unimpaired control subjects. Whereas this finding will hold for the group, there might be some language-impaired individuals who will perform within the range of the unimpaired control subjects. Indeed, a number of case studies of agrammatic Broca's aphasics who do not display problems in understanding non-canonical sentences have been published (cf. Berndt, Mitchum, and Haendiges 1996; Martin 2006 for overview). One group of researchers regards these studies as providing evidence for the claim that language comprehension problems are no characteristic sign of Broca's aphasia and, therefore, do not have to be captured in deficit theories accounting for this disorder. The other group, however, argues that these cases are to be expected in a normally-distributed group of language-impaired subjects and constitute no counter-cases for the claim that the described language comprehension deficit is a characteristic symptom in Broca's aphasia (cf. Grodzinsky 2000; Drai and Grodzinsky 2006 and the subsequent discussions on methodological considerations how to compare and evaluate group differences).

Besides the above described problems in understanding non-canonical sentence structures, comprehension problems have been found in the area of binding. Interestingly, it seems as if problems in this area might be more specific to different language impairments and individuals with different disorder syndromes seem to display distinct problems in interpreting pronouns. Thus, children with Specific Language Impairment have been found to give an anaphorical reading to pronouns and might choose a picture showing that 'Mowgli tickles himself' as adequate depiction of the sentence *Mowgli tickles him* (Van der Lely and Stollwerck 1997). A similar behaviour has been observed in individuals with Broca's aphasia (Grodzinsky et al. 1993). Wernicke's aphasics, in contrast, seem to display a general disruption in understanding pronouns and, thus, might consider that a sentence like *Mowgli tickles him* does not depict a picture where Mowgli tickles Baloo (Grodzinsky et al. 1993). A different picture has again been observed in individuals with Down's syndrome who experience problems in understanding reflexive pronouns and might hence accept a picture showing that 'Mowgli tickles him' as illustration of the sentence *Mowgli tickles himself* (Ring and Clahsen 2005). In contrast to all these groups, individuals with Williams syndrome have been found to be unimpaired with respect to the comprehension of pronouns and anaphors (Clahsen and Almazan 1998; Ring and Clahsen 2005). The syndrome-specificity of the deficits observed in this area of grammar has inspired researchers during the last years to pinpoint the precise nature of the deficits related to pronoun comprehension in the different syndromes (cf. section 2.5).

## **2. Accounting for syntactic deficits**

Over the last 30 years a multitude of deficit approaches have been advocated to capture the above described symptoms as resulting from syntactic deficits. Such attempts range from deficit theories which assume rather global deficits encompassing all of syntax to approaches which presuppose rather subtle deficits affecting only particular syntactic structures, processes or features. Most of these attempts have been formulated within the framework of Generative Grammar and here within the theories of Government and Binding (Chomsky 1981) and the Minimalist Program (Chomsky 1995, 2000).

### **2.1. Global syntactic deficit accounts**

The first syntactic deficit theories proposed to account for the language impairments found in agrammatic Broca's aphasia date from the late 1970s and the early 1980s. The framework of Generative Grammar was still in its youth by then and research on language disorders

focused on finding evidence for the assumption that the human language faculty consists of autonomous modules such as a syntax module (see section 1.1.). Given this research agenda, researchers focussed on the issue whether Broca's aphasia could be described as a language disorder that selectively affected the syntactic component, sparing the semantic component (e.g. Caramazza and Berndt 1978), and whether this disorder was due to an impairment in syntactic competence that affected language production and comprehension in parallel (e.g. Heilman and Scholes 1976; Caramazza and Zurif 1976) (see section 1.2.2.).

Syntactic-deficit approaches to agrammatic Broca's aphasia assumed rather global deficits encompassing all syntactic capacities. An example for these approaches is the *Lexical-Node Hypothesis* suggested by Caplan (1985). According to this approach, agrammatic Broca's aphasics are not able to construct syntactic phrase structures. They only have access to lexical categories such as verbs and nouns and to the information that is stored with these lexical elements in the mental lexicon. Besides semantic information this includes syntactic information on word class, subcategorisation frames and thematic roles. Since the capacity to build up hierarchically ordered syntactic structures is lost in agrammatic Broca's aphasia, the main lexical elements are linearly ordered. Thematic roles such as AGENT or THEME are assigned to linear positions in these word strings: AGENT is assigned to the noun in front of the verb, THEME is assigned to the noun coming after the verb (cf. fig.1). The incapacity to build-up phrase-structure representations also entails that syntactic operations or relations which build on phrase structures, such as movement operations or the establishment of syntactic relations between hierarchically ordered constituents, are no longer possible. With this account, Caplan tried to provide a unitary account for the omission of function words and inflectional morphemes, for the preponderance of canonical sentence structures and for the comprehension problems that result when sentences with non-canonical ordering are presented.

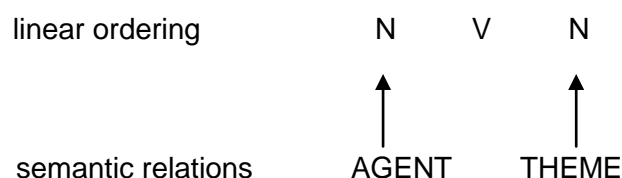


Figure 1: Sentence representations according to a global syntactic deficit account

## 2.2. Deficits in structure building

With time and concurring with the development of generative syntax, more fine-grained accounts of syntactic deficits were developed that no longer assumed a global deficit affecting all syntactic operations and relations. Instead, approaches were proposed that suggested more specific deficits in the build-up of syntactic phrase structure.

### 2.2.1. Surface-structure-deficit accounts

With the elaboration of the theory of Government and Binding (Chomsky 1981), the distinction between a thematic D(eep)-structure and a S(urface)-structure presented itself as an obvious candidate in accounting for the syntactic deficits observed in Broca's aphasia. D(eep)-structure is the level that encodes the lexical properties of the sentence elements and the thematic relations that hold between these elements. Surface properties of the sentence such as the ordering of sentence elements and the inflection of these elements are reflected by S(urface)-structure which is derived from D(eep)-structure by movement transformations.

Functional heads and their projections are central in deriving S(urface)-structure. Functional heads host inflectional morphology such as agreement, tense and case markings that have to be associated with lexical elements such as verbs and nouns. Functional elements like complementisers and auxiliaries are base-generated in functional heads such as C, T and AGR, and functional heads and their specifiers offer landing sites for constituents that move out of their base-generated D-structure positions (Haegeman 1991).

As presented above (see section 1.2.1.), the omission of functional elements (e.g. articles, complementisers and auxiliaries) and substitutions respectively omissions of inflectional morphemes are the core symptoms of agrammatic language production. In contrast, lexical information (e.g. word class and subcategorisation information) and the establishment of semantic relations between lexical elements seem relatively unimpaired in Broca's aphasia. This dissociation led researchers to hypothesise that agrammatic Broca's aphasia might be due to an impairment in deriving S(urface)-structure representations – an impairment that ultimately results from a deficit in projecting functional heads (e.g. De Bleser and Bayer 1991; Ouhalla 1993). According to Ouhalla (1993) for instance, agrammatic individuals no longer have access to the inventory of functional categories that is part of Universal Grammar and, hence, can no longer project these functional heads into syntax. While the derivation of S-structure is impaired according to these approaches, all information that comes from the lexicon is unaffected and can be projected into syntax. Thus, the build-up of lexical projections such as VPs, NPs, and PPs according to the X'-scheme is still possible and the argument structure as well as the thematic structure of lexical heads can be expressed. In consequence then, the build-up of syntactic structure is reduced to the D-structural level in these approaches (cf. figure 2). All syntactic operations that require functional heads and their projections at the S-structural level are, however, assumed to be impaired.

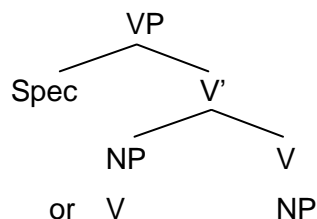


Figure 2: Sentence representations according to a S(urface)-structure-deficit account

While S(urface)-structure-deficit approaches differ with respect to whether or not inflected forms can be extracted from the lexicon, the deficit in building-up S(urface)-structure representations should in any case affect movement transformations. Movement transformations require a landing site for the moved constituent. These landing sites are provided by functional heads and their specifier positions. If functional heads are no longer projected, systematic movement transformations should not be observable in agrammatic speech production. Obligatory verb movement has provided an excellent test case for this prediction. Consider German, a V2 language where the finite verb has to move to the C position, the second structural position, in main clauses. Non-finite verbs, in contrast, remain clause-finally in the V position of the head-final German VP (cf. figure 3).

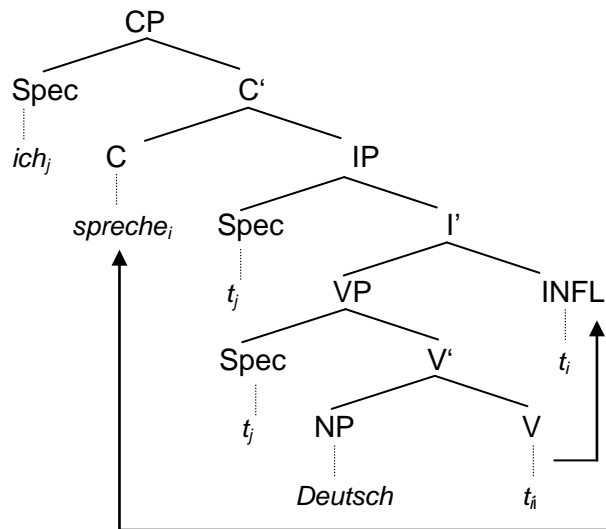


Figure 3: V2 movement in German<sup>7</sup>

An inability to build-up S(urface)-structure and to project the functional head C should necessarily affect the obligatory V2 movement of finite verbs in German main clauses. In consequence, a systematic V2 placement of finite verbs in main clauses is not expected to occur in the language production of German agrammatic Broca's aphasics. However, an investigation of spontaneous and elicited speech produced by four German agrammatic aphasics revealed that out of 615 finite verbs for which verb placement could be unambiguously determined, 607 (98.7%) were correctly placed in V2 position (Penke 2001). Only 2% were incorrectly placed clause-finally. In contrast to the placement of finite verbs, 97.8% of the 182 non-finite verbs produced by these subjects were left clause-finally in the VP. These data indicate that the V2 placement of finite verbs and the resulting asymmetry in the positioning of finite and non-finite verbs in German main clauses are retained in German individuals with Broca's aphasia. Since V2 movement requires a functional projection outside VP, the data from German, as well as similar data from Dutch, French and Italian Broca's aphasics (Kolk and Heeschen 1992; Lonzi and Luzzatti 1993), provide strong evidence against deficit approaches which assume that functional categories can no longer be projected in agrammatic aphasia.

### 2.2.2. Deficits affecting the build-up of specific functional projections:

As indicated above, deficit approaches assuming that all functional heads and their projections are impaired in a language disorder such as agrammatic Broca's aphasia seem to posit too much a deficit. As the data on verb movement in Broca's aphasia indicate, not all functional heads are missing from agrammatic S(urface)-structure representations. Research consequently focussed on the syntactic functions specific functional categories have, exploring whether or not these particular functions are retained or impaired in language disordered subjects. These endeavours were furthered by developments in syntactic theory such as the *Split-INFL Hypothesis* (Pollock 1989) which proposed that the functional category INFL should be divided up into two independent functional categories: T for tense marking and AGR for establishing subject-verb agreement. This proposal and subsequent related work increased the number of potential functional heads to investigate in language disorders. As a result of this research agenda, a number of dissociations have been reported

in the literature on syntactic deficits, indicating that syntactic functions related to specific functional categories are impaired whereas syntactic functions related to other functional categories are retained in particular language disorders.

Consider for example the *Tree-Pruning Hypothesis* advocated by Friedmann and Grodzinsky (1997) which claims that syntactic structures in agrammatic aphasics are "pruned" at the tense node. The account was proposed on the basis of data from a Hebrew-speaking agrammatic aphasic subject who displayed a strong dissociation within verbal morphology. While this subject had a good command of agreement inflection, she markedly failed in producing verb forms correctly inflected for tense. Moreover, this subject could not produce embedded sentences and *wh*-questions, omitting the sentence-initial complementiser or *wh*-phrase most of the time. In accounting for this data, Friedmann and Grodzinsky made use of the Split-INFL Hypothesis and assumed an ordering of functional heads according to which C is higher than T which is higher than AGR. Given this ordering, the observed deficit can be captured by the assumption that all functional heads from T upwards can no longer be projected in agrammatic representations, thus accounting for retained agreement inflection in this subject. The Tree-Pruning Hypothesis implies that all syntactic operations that necessarily rely on the functional projection TP and on functional projections higher up the syntactic tree, such as CP, can no longer be performed in agrammatic subjects.

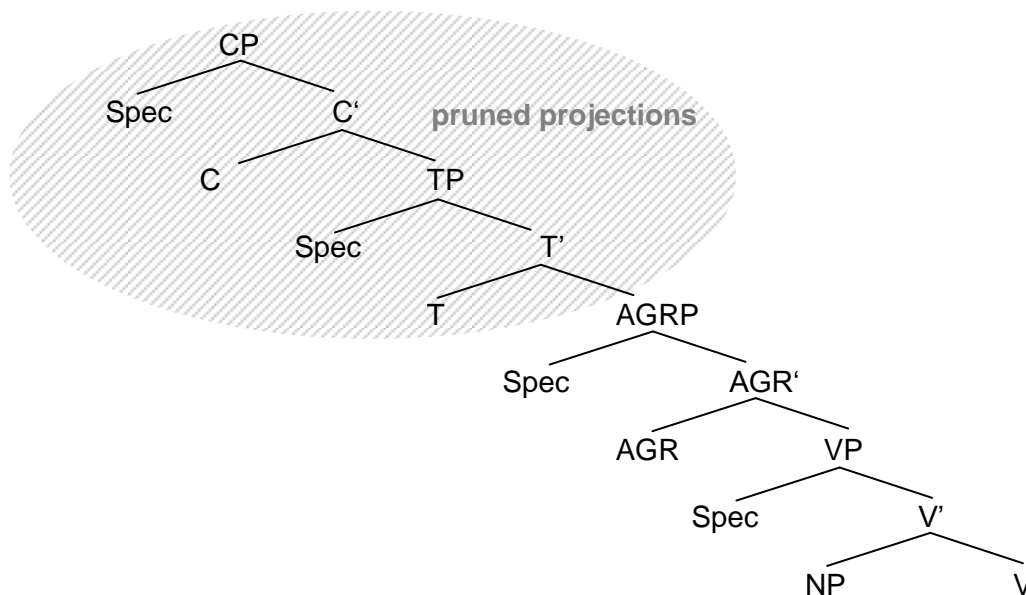


Figure 4: A pruned syntactic representation according to the Tree-Pruning Hypothesis

Accounts such as the Tree-Pruning Hypothesis also allow for relating the severity of the syntactic deficit to the type and number of functional heads affected. The more severe the agrammatic deficit, the fewer functional projections can be built up (Friedmann and Grodzinsky 1997) or generated by the syntactic operation *merge* (Hagiwara 1995). Whereas a mild deficit might only affect the CP layer in phrase-structure representations, a severe deficit might affect the build-up of all functional projections down to the VP layer. The integration of gradability into accounts such as the Tree-Pruning Hypothesis is certainly interesting since language deficits come in different levels of severity in different individuals. However at present, the suggestion to relate the severity of the disorder to the amount of

functional heads that can be projected by language impaired individuals suffers from a lack of independent criteria to indisputably establish the severity of the disorder in a given subject. Without independent criteria to establish the severity of a language deficit circular arguments are likely to arise: A deficit in producing a language structure X (e.g. V2 movement) is taken as evidence for a severe disorder. A severe disorder is claimed to lead to a pruning of functional nodes high-up in the syntactic tree (e.g. CP). Evidence that the respective functional layer (CP) is indeed pruned is then claimed to come from the observation that structure X (e.g. V2 movement) is impaired. As long as independent criteria are missing, potential counter-evidence to these deficit approaches – such as the data on German verb movement which indicate that the functional head C can still be projected in agrammatic aphasic subjects – is discarded by claiming that these data come from individuals who are not impaired severely enough to display the suggested deficit. In practise, this renders the falsification of gradable deficit theories quite impossible.

Despite empirical controversies on the issue whether or not the Tree-Pruning Hypothesis adequately captures the deficits observed in agrammatic Broca's aphasia across languages (cf. e.g. Lee 2003; Duman et al. 2007; Lee, Milman, and Thompson 2008; Neuhaus and Penke 2008), the Tree-Pruning Hypothesis has also been challenged on theoretical grounds. For one, it has been argued that the suggested order of functional categories that places the T node above the AGR node does not hold across languages. In German, for instance, the AGR node is placed above the T node reflecting affix-order in German verbs (Baker's 1985 Mirror Principle). A pruning of the syntactic tree above the T node would, hence, lead to an inability to project any functional categories in German – a prediction which is not borne out since agreement inflection (involving the AGR node) and V2 movement (involving the C node) are largely intact in German agrammatic aphasics (Penke 2000; Nanousi et al. 2006 for a similar argument with respect to Agrammatism in Greek). Also, in more recent developments of the Minimalist Program tense and agreement are no longer treated as two independent functional categories (Chomsky 2000), but agreement is an operation checking uninterpretable features of T. Without an independent functional category AGR, however, the dissociation between retained agreement inflection and impaired tense inflection observed by Friedmann and Grodzinsky (1997) can no longer be captured in the Tree-Pruning account, since agreement, like tense inflection, would involve the pruned T node (cf. Wenzlaff and Clahsen 2004).

Other approaches have considered the CP layer as the locus where language deficits strike in agrammatic Broca's aphasia or Specific Language Impairment. Proposals such as by Platzack (2001) and Hamann, Penner, and Lindner (1998) capture the observation that in agrammatic aphasics and Specific Language Impaired children syntactic structures targeting the CP-layer such as *wh*-questions or subordinate clauses are often missing in speech production or are incorrectly produced lacking *wh*-pronouns or complementizers (cf. section 1.2.1). However, while such constructions might be at times omitted or incorrectly produced, studies have also found that agrammatic aphasics and Specific Language Impaired children do not lack the ability to produce correct *wh*-questions and subordinate clauses targeting the CP layer (cf. e.g. Penke 2001; Lee, Milman, and Thompson 2008; Neuhaus and Penke 2008; Rothweiler, Chilla, and Clahsen to appear). These findings are incompatible with the assumption that the CP layer can no longer be projected in disorder syndromes such as Agrammatism or Specific Language Impairment (cf. section 3).

### 2.3. Deficits with the feature specification of functional heads

A different type of deficit theories assumes that while the build-up of syntactic structure via the projection of functional heads is intact, the syntactic features that are hosted by these functional categories are underspecified. This type of approach goes back to an account on agrammatic language production proposed by Grodzinsky (1984).

Due to the Anglo-American dominance in modern generative linguistic research, most research on language impairments up to the early 1980s was concerned with the deficits that were observed in English-speaking individuals. Since the omission of inflectional affixes is a core symptom of English speakers with Broca's aphasia, omissions of inflectional markers were seen as characteristic sign of this disorder across languages. The assumption that across languages bound and free functional elements (inflectional markers and function words) are omitted in Broca's aphasia seemed to suggest that the build-up of syntactic structure, specifically the build-up of functional projections, was impaired in agrammatic individuals (see sections 2.2.1.–2.2.2.). In comparing deficits associated with agrammatic Broca's aphasia across languages, Grodzinsky (1984), however, observed that omissions of inflectional markers only occur in agrammatic speech production if the remaining word stem is a possible word in the respective language, as is the case in English. In languages such as Hebrew, Russian, or Italian, where the omission of inflectional markers results in stems which cannot surface as possible words in these languages (Italian *\*libr-* instead of *libri*, Russian *\*knig-* instead of *knigi*), omission errors do not occur. Rather, in these languages inflectional markers are often substituted by other markers in the speech of agrammatic aphasic individuals, resulting in inflectional errors.

Such inflectional errors are problematic for deficit accounts which assume that all functional projections are missing in syntactic representations of agrammatic individuals. According to Government and Binding theory, the standard syntactic framework at this time, inflectional morphology is hosted by functional heads. Hence, the presence of inflectional markers, even incorrect ones, indicated that syntactic representations contain functional heads that dominate these inflectional markers. Therefore, Grodzinsky suggested that the build-up of S(urface)-structure representations itself is not impaired. However, functional heads are underspecified, since the specific feature values of the syntactic features dominated by functional heads are deleted in agrammatic syntactic representations (cf. fig. 5). As a consequence, free standing functional elements such as articles or auxiliaries are omitted in agrammatic language production. In addition, the deletion of specific feature values results in omissions of bound inflectional morphology if the omission of an inflectional marker leads to a possible surface word in the respective language. In languages where omissions are not an option, any feature value, matching or not, is selected for an underspecified syntactic feature and, hence, any possible inflectional form can be produced – the correct one or an incorrect one. Given these considerations, a sentence such as *the boy is kissing the girl* would consequently be produced as *boy kiss girl* by English-speaking agrammatic subjects.

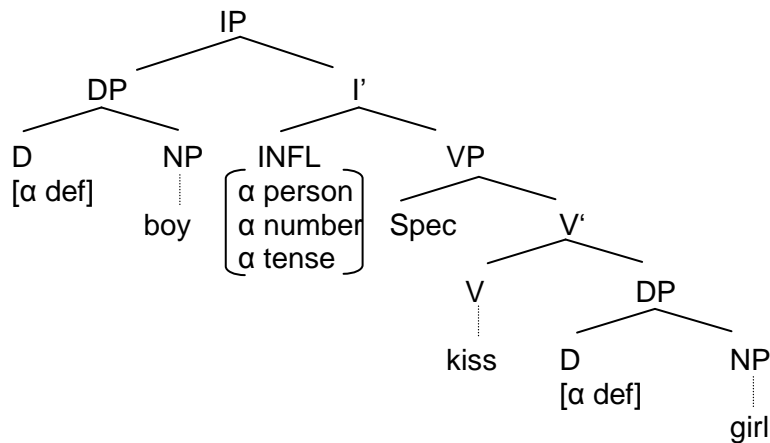


Figure 5: Syntactic S(urface)-structure representation with underspecified functional heads, resulting in the 'agrammatic' sentence *boy kiss girl*

Grodzinsky's finding that omissions of inflectional elements will only occur where licensed by the grammar of a given language constitutes an important generalisation on inflectional deficits in language disorders. However, his proposal that all functional heads are underspecified turned out to be too strong. Consider for instance subject-verb agreement inflection in German Broca's aphasics. According to Grodzinsky's account the agreement features dominated by the functional head INFL are underspecified. Consequently, agreement affixes should be omitted since this is a morphologically legitimate option in German. In contrast to this prediction, subject-verb inflection is basically intact in German Broca's aphasics. An analysis of subject verb agreement inflection in spontaneous speech production (Penke 1998: 191) and several elicitation experiments (Penke 1998: 192–195; Janssen and Penke 2002) conducted with a total of 14 German agrammatic aphasics revealed that all tested individuals were able to systematically mark agreement in German (individual correctness scores ranging from 71% to 100%, mean 92%).

Nevertheless, Grodzinsky's suggestion that syntactic deficits might be due to underspecified feature values of functional heads inspired researchers in the field, a reason certainly being the advent of the Minimalist Program (Chomsky 1995, 2000) in which syntactic features play a central role in the derivation of syntactic structure. In the Minimalist Program, functional heads dominate bundles of syntactic features that have to be checked against the inflectional endings of a form generated in the lexicon. Feature checking will eliminate the abstract features dominated by functional heads. To enable feature checking the inflected form has to move into a checking domain of the functional head hosting the respective syntactic feature. If the syntactic features realized by an inflected form are compatible with the syntactic features dominated by a functional head, the syntactic features headed by this functional category can be checked off and will be eliminated from the syntactic representation. The Minimalist Program draws a distinction between different types of features. Strong features force overt movement, i.e. feature checking has to occur prior to spell-out. Weak features are checked after spell-out. Their movement into the checking domain therefore is covert, i.e. not visible on the surface. Another distinction is drawn between interpretable and uninterpretable features. Interpretable features can be interpreted at the logical form level, i.e. they are relevant to the semantic interpretation of an expression.

Uninterpretable features, in contrast, cannot be interpreted at this level and need to be checked off before the logical form level, lest the derivation crashes at this level.

The issue whether syntactic impairments can be related to deficits causing the underspecification of specific features while sparing others has inspired research during the last 15 years. Rice and Wexler (1996) adopted this line of explanation to account for the observation that children with Specific Language Impairment display a protracted period where they optionally produce root infinitives instead of main clauses with finite verbs (cf. section 1.2.1.). According to the *Tense-Omission Model* (Wexler 1994) an optional infinitive results when the child leaves the tense feature of the functional category INFL underspecified. In this case, the verb displays non-finite inflection and does not undergo verb movement. In children with Specific Language Impairment the time period where the tense feature may be left underspecified is said to be extended (*Extended Optional Infinitive Hypothesis*). Whereas the tense feature may be unspecified in syntactic representations of children with Specific Language Impairment, all other functional categories, features or syntactic operations are said to be intact. This claim is, however, disputed on the basis of data indicating that subject-verb agreement inflection is impaired in Specific Language Impairment although the agreement features should be unaffected according to the Extended Optional Infinitive Hypothesis (cf. Clahsen, Bartke, and Göllner 1997).<sup>8</sup>

Based on the observation that verbal agreement inflection is impaired in German children with Specific Language Impairment whereas tense marking is significantly better retained, Clahsen, Bartke, and Göllner (1997) have proposed that Specific Language Impairment is due to a deficit particularly affecting the uninterpretable phi-features of verbs, i.e. the verb's agreement features.<sup>9</sup> A reverse deficit affecting interpretable tense features has been claimed to account for the observation that tense inflection is more impaired than verbal agreement inflection in German agrammatic Broca's aphasics (Wenzlaff and Clahsen 2004). Note, however, that the language deficits in Specific Language Impairment and agrammatic aphasia are not as selective as suggested above. Whereas Clahsen, Bartke, and Göllner (1997) state that only agreement but not tense inflection is impaired in Specific Language Impairment, a number of studies have found tense inflection to be severely affected in English children with Specific Language Impairment (e.g. Rice and Wexler 1996; van der Lely and Ullman 2001). Conversely, deficits with agreement inflection are typically observed in English-speaking Broca's aphasics (e.g. Goodglass, Christiansen, and Gallagher 1993; Faroqi-Shah and Thompson 2004), contrary to the claim of Wenzlaff and Clahsen (2004).

Rather, the findings suggest that deficits with respect to inflectional morphology vary across languages. In English, an analytic language with a largely reduced inflectional component, inflectional markers – be it tense or agreement markers – tend to be omitted, resulting in inflectional errors. In languages where inflectional systems are more elaborate and express more syntactic information (such as in Finnish, German, Italian, Polish or Spanish) omission rates of inflectional markers are markedly lower compared to English (e.g. Bates, Friederici, and Wulfeck 1987; Dromi et al. 1999). Thus, whereas for instance the English 3<sup>rd</sup> person singular marker -s was found to be omitted in about half of the obligatory contexts for this marker in data from children with Specific Language Impairment (Clahsen, Bartke, and Göllner 1997) and from adult Broca's aphasics (Goodglass, Christiansen, and Gallagher 1993), omission rates for subject-verb agreement inflection in German-speaking subjects with these disorders are considerably lower. Clahsen, Bartke, and Göllner (1997) report an omission rate of 20% for German children with Specific Language Impairment. The

five agrammatic aphasic subjects investigated by Penke (1998: 191) omit agreement affixes in only 15 out of 914 cases (1.6%) in spontaneous-speech production. Such variations across languages suggest that language-specific factors related to the complexity and importance of morphological inflectional systems critically affect which inflectional errors will occur in language-impaired speakers (see Penke 2008 for discussion). Note that language-specific variations in inflectional errors are problematic for deficit theories supposing selective deficits with interpretable or uninterpretable features. The categorization as uninterpretable or interpretable feature holds for all languages. Hence similar deficits with tense or agreement inflection should be observed across languages. At present, the evidence for deficit accounts which suggest that the impairments observed in language disorders such as Specific Language Impairment and Agrammatism are selective to specific features while sparing others is inconclusive.

#### **2.4. Deficits with movement**

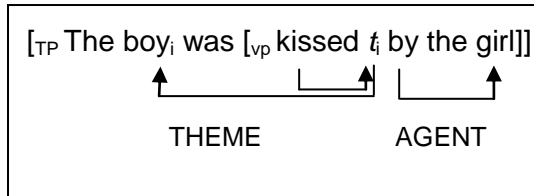
As presented in section 1.2.2., language impaired individuals often show a better understanding of sentences with a canonical SVO order of sentence constituents (*the girl kissed the boy*) compared to sentences with a non-canonical word order such as object-clefts (*it is the boy who the girl kissed*), object relatives (*the boy who the girl kissed...*), passives (*the boy was kissed by the girl*) or object topicalisations. What is common to these sentence structures is that in all these cases the object has moved out of its base-generated position and now precedes the subject. Grodzinsky has, therefore, suggested that it is the movement of the object that causes comprehension deficits in Broca's aphasia (Grodzinsky 1984, 2000). Specifically, he proposed that the deficits in language comprehension are due to the fact that movement traces in syntactically derived sentences, such as passives or object extracted questions, are deleted from syntactic representations.

In the framework of Government and Binding (Chomsky 1981), S(urface)-structure is derived from D(eep)-structure by movement transformations. Every moved constituent leaves behind a trace. The moved constituent and its trace are connected via a syntactic chain. In case of moved noun phrases, the most important syntactic function of this chain is to transmit the theta role assigned by the verb to its arguments within VP to the moved constituent. The theta role is assigned within VP to the trace of the moved constituent. Via the syntactic chain this theta role is then indirectly assigned to the moved constituent co-indexed with this trace (cf. Haegeman 1991).

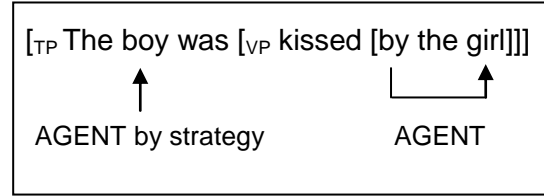
According to Grodzinsky's *Trace-Deletion Hypothesis* such traces are deleted from the syntactic representations of Broca's aphasics. Consider for instance a passive clause as in (1). In a passive clause, the object NP has to move out of VP to the subject position in SpecTP to receive case. It leaves behind a co-indexed trace in its base-position as internal argument of the verb *kiss*. The theta role THEME is assigned by the verb to this trace. Via the syntactic chain connecting the moved NP with its trace, this theta role is indirectly assigned to the moved NP, the derived subject of the sentence (cf. Haegeman 1991). The theta role AGENT is assigned by the preposition *by*. In an agrammatic representation, however, movement traces are deleted. Consequently, the theta role THEME cannot be assigned to the moved object NP via the disrupted chain. Since for interpretation every NP must have a theta role, the NP is now assigned a role by a non-linguistic linear default strategy. This default strategy assigns the AGENT role to the clause-initial NP. Since the preposition *by* also directly assigns the role AGENT to its complement NP, the thematic representation now contains two AGENT roles to choose from in interpretation: one assigned

via the default strategy, the other directly assigned by the preposition. As a consequence, in a sentence-picture-matching task (cf. section 1.2.2.) the comprehension of such sentences will drop to chance level.

(1) unimpaired representation of passives

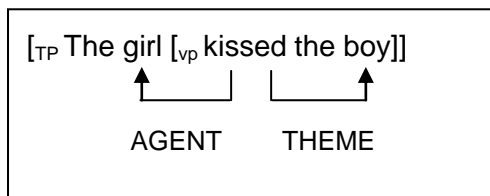


agrammatic representation



For active sentences such as in (2), in contrast, no problem arises in agrammatic comprehension, since both theta roles can be directly assigned: the THEME role is assigned by the verb to its internal argument, the AGENT role is directly assigned to the subject base-generated in SpecTP.<sup>10</sup>

(2) unimpaired/agrammatic representation for active sentences



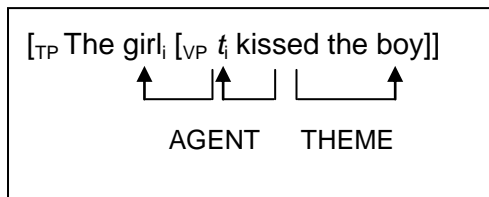
The Trace-Deletion Hypothesis has probably been the most influential deficit theory in the field to date. It has inspired a wealth of research aimed at testing and improving this account. A strength of this theory certainly lies in the fact, that it has been flexible enough to adapt to both, new findings on agrammatic comprehension and new developments in syntactic theory.

Originally, the Trace-Deletion account stated that all types of traces, including traces of moved heads, were deleted in agrammatic representations. Based on evidence that head chains are intact in agrammatic comprehension, the account was modified in the way that only traces in theta positions are said to be deleted in agrammatic representations (Grodzinsky 1995). A further modification was initiated by Hickok and Avrutin (1995) who tested the comprehension of *wh*-questions in Broca's aphasics. The Trace Deletion Hypothesis predicts that the comprehension of *wh*-object questions (*which boy/who did the girl kiss?*) should be at chance level in agrammatic aphasic subjects. The moved object-*wh*-phrase cannot receive a theta role via the disrupted chain and receives the AGENT role via the default strategy. Since the subject NP is also assigned the AGENT role (by the verb), the conflict between two AGENT roles should lead to chance performance in interpretation. Hickok and Avrutin, however, observed that their agrammatic subjects showed this behavioural pattern only for *which N* object questions, but not for *who* object questions. This led to a revision of the Trace-Deletion Hypothesis according to which a default theta role is only assigned to a referential NP, i.e. a NP which is D(iscourse)-linked, as is the cases for *which N* phrases but not for *who* phrases which are said to be quantifiers and thus not D-linked (Grodzinsky 1995).

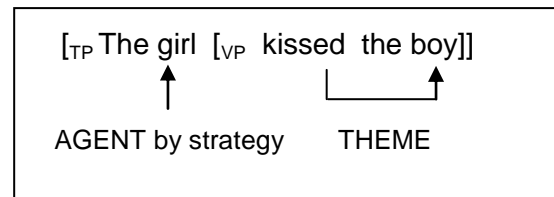
The Trace-Deletion Hypothesis has not only had to adapt to new types of data, but it has also been affected by developments in syntactic theory, most notably by the *VP-internal*

*Subject Hypothesis* (Koopman and Sportiche 1988) according to which the subject NP is not base-generated in SpecTP but in SpecVP. From SpecVP the subject moves to SpecTP to check its features, leaving a trace inside SpecVP. Integrating this assumption into the Trace-Deletion Hypothesis has turned out to be quite challenging.<sup>11</sup> Consider first a simple active sentence such as (3). In the agrammatical representation of this sentence the AGENT role can no longer be assigned to the subject NP moved out of VP since the trace is deleted. Nevertheless, comprehension of such sentences is not affected in Broca's aphasics because the subject NP receives the AGENT role via the default strategy (cf. 3).

(3) unimpaired representation of actives

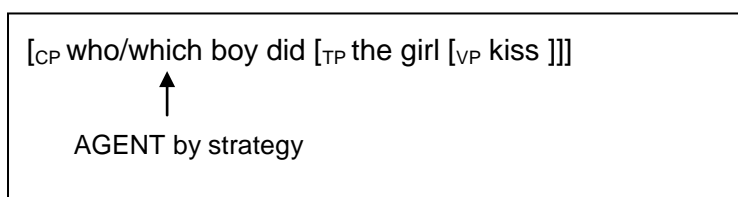


agrammatic representation



For other sentence constructions, however, the integration of the VP-Internal Subject Hypothesis into the Trace-Deletion Hypothesis has led to predictions that are not born out by the agrammatical comprehension data. Consider as an example *wh*-object questions such as (4). In a *wh*-object question neither the object NP nor the subject NP receives a theta role directly by the verb because both phrases are moved out of VP and their traces are deleted. The default strategy would then assign the AGENT role to the clause-initial NP, the object *wh*-pronoun. In consequence, *wh*-object questions should be consistently interpreted as *wh*-subject questions by agrammatical individuals and their performance in understanding such sentences should be worse than chance, a prediction which is, however, not born out by the data (cf. Hickok and Avrutin 1995; see Grodzinsky 2000: 59 for suggestions how to solve this issue within the Trace-Deletion Hypothesis).<sup>12</sup>

(4) agrammatical representation



Deficit accounts related to the Trace-Deletion Hypothesis – which is explicitly restricted to capture only agrammatical language comprehension (cf. Grodzinsky 2000) – have meanwhile been adopted to capture similar problems in individuals with Wernicke's aphasia (Grodzinsky and Finkel 1998) and in children with Specific Language Impairment (Friedmann and Novogrodsky 2004) or hearing impairments (Friedmann and Szterman 2006).

## 2.5. Deficits involving structural dependencies

Whereas Grodzinsky explicitly regards deficits in movement operations as causing the impaired comprehension of sentences with moved object NPs, other researchers have suggested that these comprehension problems are due to a deficit in establishing a structural relationship between the moved constituent and its trace (e.g. Mauner, Fromkin, and Cornell

1993). A deficit in establishing syntactic structural relationships should also affect other syntactic phenomena where a similar structural dependency holds. A case in point is the structural relation that holds between referentially dependent NPs and their antecedents. Van der Lely and colleagues were the first to investigate the comprehension of passives and referentially dependent NPs in children with Specific Language Impairment (van der Lely 1996; van der Lely and Stollwerck 1997). They found that children with Specific Language Impairment were impaired in interpreting passives and referentially dependent NPs and suggested that children with Specific Language Impairment suffer from a deficit with non-local structure-dependent relations that, for instance, impairs the computation of the binding domain. Van der Lely's *Computational Grammatical Complexity Hypothesis* (van der Lely 2005) posits a rather broad grammatical deficit that is not precisely couched in theoretical terms and might encompass all sorts of hierarchically organized structures in syntax, morphology and phonology. It has therefore been criticized for being too broad to capture more fine-grained deficits in Specific Language Impairment affecting, for instance, agreement but not tense inflection (cf. Clahsen 2008).

Interestingly, the deficits obtained for binding relationships might turn out to be specific for certain language disorders. Agrammatic Broca's aphasics and children with Specific Language Impairment display particular problems in interpreting pronouns and often accept a reading in which the pronoun is anaphorically linked to an antecedent. Thus, they might accept a sentence such as *Mowgli is tickling him* as a description of a picture depicting Mowgli tickling himself (Van der Lely and Stollwerck 1997; Grodzinsky et al. 1993). In doing so, these language impaired individuals mirror unimpaired children who also take considerable longer to correctly interpret non-reflexive pronouns compared to reflexive pronouns (cf. Guasti 2002: chapter 8 for overview).

A different deficit has been reported in two recent investigations on the comprehension of passives and anaphoric elements in individuals with Down's syndrome (Ring and Clahsen 2005; Perovic 2006). These studies found a marked impairment in interpreting reflexive pronouns and passive sentences. In contrast, the interpretation of pronouns and active sentences was unimpaired (Ring and Clahsen 2005; Perovic 2006). In an attempt to provide a unitary account for the deficits in interpreting passives and reflexive pronouns, Ring and Clahsen (2005) have suggested that individuals with Down's syndrome suffer from a specific syntactic deficit leading to an inability to construct A(argument)-chains. An A-chain requires that the two elements connected by the chain (the moved object NP and its trace, respectively the reflexive and its antecedent) are in the same local structural configuration, that they share the same syntactic features via coindexation and that the reflexive pronoun respectively the trace are c-commanded by the antecedent respectively the moved object NP (Chomsky 1995; Reuland 2001). A deficit in forming A-chains brings about that a reflexive pronoun cannot be syntactically bound by its local antecedent. Likewise a moved object NP cannot be related to its trace resulting in defective theta-role assignment to the moved NP. Both will lead to comprehension problems. Ring and Clahsen also investigated a group of children with Williams syndrome who were matched for age and IQ with the Down's syndrome subjects. Despite a similar impairment in intellectual development, the subjects with Williams syndrome did not show a deficit in the comprehension of passives or referentially dependent NPs such as reflexive pronouns. Ring and Clahsen therefore suggest that the comprehension problems observed in individuals with Down's syndrome are specific for this syndrome and cannot be explained by their impaired intellectual development. The claim that individuals with Down's syndrome suffer from a specific syntactic deficit in

constructing A-chains is also interpreted as being informative for theoretical accounts of binding. Thus, Perovic (2006) argues that the data support a fractionation of binding into a syntactic component which governs the interpretation of bound anaphors and which is impaired in Down's syndrome and an extra-syntactic component which regulates the coreferential interpretation of pronouns and is intact in individuals with Down's syndrome.

The data and interpretations presented by Ring and Clahsen (2005) and Perovic (2006) are certainly fascinating. Note, however, that much hinges on whether or not the adapted syntactic interpretation of binding relations proves valid. Also, Ring and Clahsen admit that their interpretation does not capture every error pattern observed in their data. Thus, younger children with Down's syndrome also committed significantly more reversal errors than unimpaired control children in interpreting active sentences. As active sentences are said not to involve A-chains, this error pattern is unexplained by Ring and Clahsen's account. Finally, Ring and Clahsen (2005) and Perovic (2006) link the deficit in interpreting reflexive pronouns to the deficit in interpreting passive sentences and provide a syntactic explanation that relates both comprehension problems to an inability to construct A-chains. Note however, that while deficits in interpreting referentially dependent NPs seem to vary between different deficit syndromes, such variation is not observed with respect to the comprehension of passives. Independent of the particular disorder syndrome, language-impaired subjects display similar problems in understanding passive clauses (cf. section 1.2.2.). This observation asks for an explanation. Are the similar problems in passive comprehension caused by different language deficits in different deficit syndromes? And if so what are these deficits that lead to similar performance in passive comprehension but to different performance in interpreting referentially dependent NPs? Further research is needed to evaluate these issues.

### **3. Problems in accounting for syntactic deficits**

As indicated by these short sketches on syntactic deficit theories advocated in the field, to date the controversies about how to capture and explain language impairments that seem to affect syntactic representations have been going on. In these controversies, syntactic deficit theories have been challenged both by conflicting empirical data and by developments in syntactic theory that force an adaptation of deficit approaches to encompass both the new evidence and the new developments in syntactic theory – a venture that often turns out to be quite intricate. Besides the challenges posed by new data and new theoretical developments, syntactic deficit accounts are also subject to more general discussions concerning the issue whether the observed deficits are indeed due to impaired syntactic representations.

The accounts presented in section 2 above propose a deficit in syntactic representations and thus a deficit in grammatical competence that underlies impaired language behaviour. Such a deficit in grammatical competence should affect all aspects of language performance in parallel (Weigl and Bierwisch 1970). A defective syntactic element, structure or operation should no longer be produced, comprehended or judged correctly by an affected individual.<sup>13</sup>

A crucial issue for such accounts is their all-or-none flavour. A specific syntactic structure or function is either intact or impaired and thus the language-impaired speaker is or is not able to produce grammatical structures that involve the respective syntactic representation. Note, however, that such all-or-none behaviour is rarely observed in individuals. Grammatical competence is only *one* factor determining performance, with other factors, such as e.g. processing limitations, social, pragmatic or discourse factors affecting it, too. Indeed, it has been shown that syntactic deficits observed for instance in agrammatic Broca's aphasics

deteriorate or ameliorate depending on task demands or time constraints that lead to a lowering or raising of the processing load associated with the task (Kolk and Heeschen 1992). Tasks which minimize processing load, such as cloze tasks where the subject has to produce only the structure or form of interest whereas the sentential context is given, often lead to better performance than more unrestrained tasks (e.g. Kolk and Heeschen 1992). For an illustration of this point, consider data that come from an investigation on the production of *wh*-questions in German agrammatic Broca's aphasics (Neuhaus and Penke 2008). Production of *wh*-questions was tested by an elicitation task, where subjects had to transform a given main clause into a *wh*-question, and a sentence repetition task, where subjects were asked to repeat *wh*-questions as accurately as possible. The repetition of a given syntactic structure is less demanding than the construction of this structure by the individual her/himself. A comparison of the correctness scores obtained in these two experiments yielded that the production of *wh*-questions resulted in better performance in the less demanding repetition task (mean correctness score 78.6%) than in the more demanding elicitation task (mean correctness score 66.1%), a finding that illustrates the influence of task demands on language behaviour in language impaired individuals. Other syntax-external factors that influence the performance of language-impaired subjects relate to the testing situation, i.e. to factors such as the familiarity with the investigator or the formality of the testing situation (e.g. Heeschen and Schegloff 2003). All these factors contribute to the observation that language-impaired individuals will only rarely achieve a correctness score of 100% for a tested construction in an experimental investigation.

What however is to be concluded from correctness scores of 80%, 60%, 40% or 20%? That is, when is the correctness score low enough to conclude that a syntactic representation is defective and when is it high enough to conclude that syntactic representations are basically unimpaired? Consider again the data on *wh*-question production in German agrammatic Broca's aphasics (Neuhaus and Penke 2008). According to the Tree-Pruning Hypothesis (Friedmann and Grodzinsky 1997) individuals suffering from agrammatic Broca's aphasia should no longer succeed in producing grammatical *wh*-questions since the CP layer is pruned from syntactic representations (see section 2.2.2.). In an experiment eliciting *wh*-questions, the seven tested aphasic subjects succeeded in 66.1% of the contexts in producing a correct *wh*-question that involved the projection of the CP layer, a score that was significantly lower than the 97.5% obtained by unimpaired control subjects (Neuhaus and Penke 2008). The significant difference between aphasic subjects and unimpaired control subjects indicates that the aphasic subjects suffer from an impairment in producing *wh*-questions. But is this impairment due to an inability to project CP as claimed by the Tree-Pruning Hypothesis? If this were the case, how then can we account for the observation that the aphasic subjects succeeded in 66.1% of the contexts in producing *wh*-questions that required the projection of the CP layer?

A related problem comes from the variability in language behaviour a group of individuals suffering from the same language disorder will typically display. In the above-mentioned investigation of *wh*-question production abilities in German Broca's aphasics (Neuhaus and Penke 2008), individual correctness scores of the seven tested aphasic subjects ranged from 27.8% to 96.3%. The scores for the unimpaired control subjects in this task ranged between 88.3% and 100%. Thus, rather than a clear boundary between impaired and spared performance, there is a continuum between the performance of language unimpaired and impaired individuals. This continuum – often observed for impaired and unimpaired performance – and the variability in performance that typically holds in a group of language

impaired individuals render any strong claims on a representational deficit (such as the claim: 'agrammatic syntactic representations are pruned') problematic.<sup>14</sup> As sketched in section 2.2.2., one solution to this problem of variability is to integrate the idea that language disorders are gradable and come in different levels of severity. This move, however, requires independent criteria to establish the severity of language disorder lest to avoid circularity in argumentation.

As the data on *wh*-question production in German agrammatic aphasia illustrate, data of language-impaired subjects are complex and difficult to interpret in practice. Their interpretation as well as the conclusions drawn on the basis of such data crucially depend on issues related to statistical methodology, subject sampling and data analysis that have been at the heart of many controversies in the field (cf. e.g. Draai and Grodzinsky 2006; Grodzinsky 2000 and the discussions following these two articles). The problems related to the identification of clear-cut behavioural patterns, the gradability of language deficits and the variability of language behaviour between and within language impaired individuals have led to a second group of approaches accounting for language deficits. In contrast to representational syntactic-deficit accounts, processing accounts state that syntactic representations are not impaired in language disorders and can still be put to use. According to these accounts, the observed language problems result from a deficit in language processing caused by limitations of the processing capacity. Such processing limitations may be domain-specific, i.e. restricted to the processing of language, or domain-general, affecting non-verbal processing alike. Processing accounts have suggested, for instance, that the language behaviour observed in language impaired subjects is caused by reductions in memory components such as working memory (e.g. Just and Carpenter 1992) or verbal short-term memory (e.g. Gathercole and Baddeley 1990). A consequence of such a deficit might be that language input can not be kept in working memory long enough to extract or check morpho-syntactic information, especially in complex sentences where, for instance, deviations of canonical ordering occur. Other researchers have suggested that processing limitations result from a restricted amount of energy available to the subject for a specific task (e.g. Lapointe 1985; Bates and Wulfeck 1989; Avrutin 2000) or from desynchronisation in the temporal course of language processing (e.g. Kolk 1995). In the former case, it might be too costly for a language impaired individual to compute specific syntactic constructions or relations. In the later case he/she might not be able to compute syntactic structure or structural relationships in a given temporal window, either because the computation is too slow or its results decay too quickly.

Limitations of processing capacity have been held responsible for the language impairments found in a wide variety of language deficits such as agrammatic Broca's aphasia (e.g. Bates and Wulfeck 1989; Kolk 1995; Caplan and Waters 1995), Wernicke's aphasia (e.g. Lavorel 1982), Specific Language Impairment (cf. Leonard 1998; Leonard et al. 2007 for overview), Down's syndrome (e.g. Chapman, Hesketh, and Kistler 2002) and early unilateral focal brain lesions in children (Bates and Roe 2001).

A critical issue with respect to processing accounts is whether the proposed limitations in domain-general processing capacities cause the observed language impairments or whether they are a co-symptom that accompanies syntactic deficits in specific language disorders without causing them. A deficit in relating moved NPs to their traces, for example, might be more pronounced if more words have to be kept in memory between the two constituents. In this case, the limitation in working memory aggravates the syntactic deficit without triggering it, however. Similarly, a limitation in establishing agreement relationships might be

aggravated by defective auditory processing capacities. While such a processing impairment might particularly affect the perception of inflectional affixes that are often realised in unstressed syllables and might hence influence an individual's behaviour, it nevertheless might not be the cause for the underlying syntactic deficit.

Despite this issue, processing accounts are well suited to capture the continuum in performance that can be observed between impaired and unimpaired subjects and within groups of individuals suffering from the same language impairment. The concept that language performance, and thus also language deficits, are gradable comes naturally to processing accounts. Processing limitations can vary depending on the severity of the underlying disorder that affects the processing of syntactic structures or structural relationships. With growing processing costs or decreasing processing capacities the ability to implement the intact syntactic knowledge in language performance ameliorates or deteriorates. Processing-deficit accounts are, hence, also able to account for language behaviour that is neither completely impaired nor completely intact. And moreover, they provide an explanation for the observation that the grammatical deficits observed in language impaired speakers deteriorate or ameliorate depending on task demands or time constraints that lead to a lowering or raising of the processing load (Kolk and Heeschen 1992) – a finding that is difficult to account for in representational-deficit theories which claim that syntactic representations are either intact or impaired.

The long-standing debate between representational-deficit accounts and processing-deficit accounts is one of the central debates in the field of language disorders and far from being settled. However, an integration of these two types of accounts might well turn out to be a fruitful third path in research on language disorders. Analyses of the affected behaviour with respect to the syntactic structures, elements or operations involved inform us *where* to expect problems in language impaired speakers. For example, syntactic structures that involve the projection of the CP layer, the interpretation of particular features, the processing of specific binding relations or the interpretation of moved noun phrases which reverse canonical ordering of arguments are likely to cause problems for language impaired individuals. Processing-deficit accounts help us in predicting *when* such a deficit is likely to appear, namely when processing load increases, for example because more syntactic operations have to be computed or the task is more taxing and exceeds the processing capacities of the individual subject. During the last years a number of approaches have been postulated that aim to pinpoint which syntactic operations are particularly prone to be affected by limitations in processing capacities, thus integrating the *where* and *when* perspectives (e.g. Garaffa and Grillo 2008 on the interpretation of non-canonical sentences, Pinango and Burkhardt 2001; Vasic, Avrutin, and Ruigendijk 2006 and Stavrakaki and Van der Lely 2010 on the interpretation of pronouns, Jakubowicz 2011 on *wh*-questions).

An interesting finding suggestive of the proposed integration between representational-deficit accounts (*where*-accounts) and processing-deficit accounts (*when*-accounts) is the observation that syntax-related deficits are largely unspecific to a particular deficit syndrome. Rather than finding deficits that are characteristic for only one particular deficit syndrome, deficit symptoms such as omissions of free functional elements, problems with bound inflectional morphology, reduction of syntactic complexity and problems in interpreting sentences with non-canonical argument order or pronouns are found in a wide variety of acquired and developmental deficit syndromes (cf. section 1.2.). These symptoms seem to mark critical areas in the language system that are typically affected when an individual suffers from a limitation of her/his language ability. Syntactic deficit theories serve to inform

us about the factors that make these critical structures, elements or operations vulnerable to language impairments. The observation that the same critical entities are affected and similar deficit symptoms occur throughout syndromes suggests that processing deficits target these critical areas in the language system and result in similar deficit symptoms across syndromes.

#### **4. Summary and outlook**

The investigation of syntactic disorders is a fascinating topic where broad theoretical issues on the nature of the human language capacity and specific theoretical issues related to syntactic representations interact with empirical research conducted with a challenging subject group. A focus in the research on syntactic disorders is to find out if and how syntactic representations are defective in language impaired speakers. As indicated by the overview in section 2, syntactic theory and syntactic deficit approaches have co-evolved during the last 40 years. New developments in syntactic theory, such as the distinction between S(urface)-structure and D(eep)-structure, the Split-INFL Hypothesis or the role ascribed to features, have prompted new developments in accounting for language disorders. To date researchers active in the field have not agreed on how to capture and explain syntax-related deficits for a single deficit syndrome. However, the controversies that have arisen in the field have undeniably advanced our knowledge on the language impairments observed in particular deficit syndromes. With new developments in syntactic theory and subsequent new suggestions how to capture syntax-related language impairments new facets of syntactic knowledge have come under scrutiny in language impaired individuals. Consequently, a growing body of data has been collected over the years on which aspects of syntax are impaired or retained in particular deficit syndromes. As is the case in science, each new piece of evidence has given rise to new and more specific questions to investigate and answer, in this way accumulating our knowledge on language deficits.

Whatever approach to syntactic deficits is taken (a representational approach, a processing approach or an integrative approach) progress in the research on syntactic deficits will critically depend on syntactic theories. For one, the characterisation of affected and unaffected constructions as natural classes within a theory allows for testable predictions regarding which constructions or operations should be affected in a particular deficit syndrome. Moreover, syntactic theories provide a means to determine the complexity of a construction or operation. Following the intuition of most researchers in the field that more complex constructions or operations are more likely to be affected in language disorders than less complex ones, a theoretically motivated measure of complexity is helpful in guiding research where to look for syntactic deficits in language impairments.

While syntactic theories and the advancements within theoretical approaches have been used to capture and explain language impairments, data of language impaired speakers have on the other hand only rarely been taken as evidence in advancing syntactic theories. However, this aspect of the investigation of language deficits certainly merits some research efforts. Insights in our language faculty can only be gained by the analysis of performance data reflecting the application of this knowledge. In our aim to elucidate the nature of the human language faculty, we should consider all sorts of evidence – including data that come from the investigation of language disorders. Data presented by theoretical linguists in advancing syntactic theories is no privileged type of data in this respect, but it constitutes just another type of performance data that can be used to investigate our language faculty (cf.

Penke and Rosenbach 2007). The value of data from language impaired individuals has, for instance, been demonstrated in research on inflectional morphology. Here the selective vulnerability of regular respectively irregular inflectional morphology in specific language syndromes has been taken as evidence for a qualitative distinct representation of regular and irregular inflection in the human mind/brain (cf. Pinker 1999; Penke 2006 for overview). Also, the finding that regular and irregular inflection often dissociate in language disorders can be used as diagnostic for which inflectional markers are regular or irregular in a particular inflectional system (cf. Penke and Krause 2002; Penke 2008). Research that does not only take syntactic theory as the basis for explaining language deficits, but which aims to advance syntactic theory by taking into account error data or breakdown patterns observed in language impaired individuals might prove to be valuable in investigating the nature of syntactic representations.

### **Acknowledgement**

I am grateful to Liliane Haegeman, Monika Rothweiler, Eva Wimmer and an anonymous reviewer for helpful comments and suggestions.

### **5. References**

Avrutin, Sergey 2000 Comprehension of discourse-linked and non-discourse-linked questions by children and Broca's aphasics. In Yosef Grodzinsky, Lewis Shapiro and David Swinney (eds.), *Language and the Brain: Representation and Processing*, 295–313. San Diego: Academic Press.

Avrutin, Sergey 2001 Linguistics and Agrammatism. *GLOT International* 5(3).

Baker, Mark 1985 The mirror principle and morphosyntactic explanation. *Linguistic Inquiry* 16, 373–416.

Bartke Susanne and Julia Siegmüller (eds.) 2004 *Williams Syndrome across Languages*. Amsterdam: Benjamins.

Bartolucci, Giampiero, Sandra J. Pierce, and David Streiner 1980 Cross-sectional studies of grammatical morphemes in autistic and mentally retarded children. *Journal of Autism and Developmental Disorders* 10(1): 39–50.

Basso, Anna, André Roch Lecours, Silvia Moraschini, and Marie Vanier, M. 1985 Anatomoclinical correlations of the aphasias as defined through computerized tomography: Exceptions. *Brain and Language* 26: 201–29.

Bastiaanse, Roelien and Susan Edwards 2004 Word order and finiteness in Dutch and English Broca's and Wernicke's aphasia. *Brain and Language* 89: 91–107.

Bates, Elisabeth, Angela Friederici, and Beverly Wulfeck 1987 Grammatical morphology in aphasia. *Cortex* 23: 545–574.

Bates, Elisabeth, Angela Friederici, Beverly Wulfeck, and Larry A. Juarez 1988 On the preservation of word order in aphasia: Cross-linguistic evidence. *Brain and Language* 33: 323–363.

Bates, Elisabeth and Beverly Wulfeck 1989 Crosslinguistic study of aphasia. In: Brian MacWhinney and Elisabeth Bates (eds.), *The Crosslinguistic Study of Sentence Processing*, 328–374. Cambridge: Cambridge University Press.

Bates, Elisabeth and Katherine Roe 2001 Language development in children with unilateral brain injury. In: Charles A. Nelson and Monica Luciana (eds.), *Handbook of Developmental Cognitive Neuroscience*, 281–307. Cambridge, MA: MIT Press.

Bellugi, Ursula and Marie St. George (eds.) 2000 Linking cognitive neuroscience and molecular genetics: New perspectives from Williams syndrome. Special issue of *Journal of Cognitive Neuroscience* 12: Supplement.

Bellugi, Ursula, Liz Lichtenberger, Wendy Jones, and Zona Lai 2000 The neurocognitive profile of Williams syndrome. A complex pattern of strengths and weaknesses. *Journal of Cognitive Neuroscience* 12 (Supplement): 7–29.

Berndt, Rita Sloan, Charlotte C. Mitchum, and Anne N. Haendiges 1996 Comprehension of reversible sentences in “agrammatism”: A meta-analysis. *Cognition* 58: 289–308.

Bishop, Dorothy 2009 Genes, cognition, and communication: Insights from neurodevelopmental disorders. *Annals of the New York Academy of Sciences* 1156(1): 1–18.

Butterworth, Brian and David Howard 1987 Paragrammatisms. *Cognition* 26(1): 1–37.

Caplan, David 1985 Syntactic and semantic structures in agrammatism. In: Marie Louise Kean (ed.), *Agrammatism*, 125–152. Orlando: Academic Press.

Caplan, David and Gloria S. Waters 1995 Aphasic disorders of syntactic comprehension and working memory capacity. *Cognitive Neuropsychology* 12: 637–651.

Caramazza, Alfonso 1984 The logic of neuropsychological research and the problem of patient classification in aphasia. *Brain and Language* 21: 9–20.

Caramazza, Alfonso 1992 Is cognitive neuropsychology possible? *Journal of Cognitive Neuroscience* 4(1): 80–95.

Caramazza, Alfonso and Edgar Zurif 1976 Dissociation of algorithmic and heuristic processes in language comprehension: evidence from aphasia. *Brain and Language* 3: 572–582.

Caramazza, Alfonso and Rita Sloan Berndt 1978 Semantic and syntactic processes in aphasia: A review of the literature. *Psychological Bulletin* 85: 898–918.

Chapman, Robin S., Hye-Kyeong Seung, Scott Schwartz, Elizabeth Kay-Raining Bird 1998  
Language skills of children and adolescents with Down syndrome: II. Production deficits.  
*Journal of Speech, Language, and Hearing Research* 41: 861–873.

Chapman, Robin S., Linda J. Hesketh, and Doris J. Kistler 2002 Predicting longitudinal change in language production and comprehension in individuals with Down's syndrome: hierarchical linear modelling. *Journal of Speech, Language, and Hearing Research* 45: 902–915.

Chomsky, Noam 1957 *Syntactic Structures*. The Hague: Mouton.

Chomsky, N. 1965 *Aspects of the Theory of Syntax*. Cambridge, MA: MIT Press.

Chomsky, Noam 1980 Rules and representations. *Behavioral and Brain Sciences* 3: 1–14.

Chomsky, Noam 1981 *Lectures on Government and Binding*. Dordrecht: Foris.

Chomsky, Noam 1995 *The Minimalist Program*. Cambridge, MA: MIT Press.

Chomsky, Noam 2000 Minimalist inquiries: The framework. In: Roger Martin, David Michaels, and Juan Uriagereka (eds.), *Step by step*, 89–155. Cambridge MA: MIT Press.

Chomsky, Noam 2002 *On Nature and Language*. Cambridge: Cambridge University Press.

Clahsen, Harald 2008 Chomskyan syntactic theory and language disorders. In: Martin Ball, Michael Perkins, Nicole Mueller, and Sara Howard (eds.), *The Handbook of Clinical Linguistics*, 165–183. Oxford: Blackwell.

Clahsen, Harald, Susanne Bartke, and Sandra Göllner 1997 Formal features in impaired grammars: A comparison of English and German SLI. *Journal of Neurolinguistics* 10(2/3): 151–171.

Clahsen, Harald and Mayella Almazan 1998 Syntax and morphology in Williams syndrome. *Cognition* 68: 167–198.

Curtiss, Susan and Jeannette Schaeffer 2005 Syntactic development in children with hemispherectomy: The I-, D-, and C-systems. *Brain and Language* 94: 147–166.

De Bleser, Ria 1988 Localisation of aphasia: Science or fiction. In: Gianfranco Denes, Carlo Semenza, and P. Bisiacchi (eds), *Perspectives of cognitive neuropsychology*, 161–185. Hove: Lawrence Erlbaum.

De Bleser, Ria and Josef Bayer 1991 On the role of inflectional morphology in agrammatism. In: Michael Hammond (ed.), *Theoretical Morphology: Approaches in Modern Linguistics*, 45–69. San Diego: Academic Press.

Dennis, Maureen and Harry A. Whitaker 1976 Language acquisition following hemidecortication. *Brain and Language* 3: 404–433.

Dick, Frederic, Elizabeth Bates, Beverly Wulfeck, Jennifer Utman, Nina Dronkers, and Morton Ann Gernsbacher 2001 Language deficits, localization, and grammar: Evidence for a distributive model of language breakdown in aphasic patients and neurologically intact individuals. *Psychological Review* 108(4): 759–788.

Dick, Frederic, Beverly Wulfeck, Magda Krupa-Kwiatkowski, and Elizabeth Bates 2004 The development of complex sentence interpretation in typically developing children compared with children with specific language impairments or early unilateral focal lesions. *Developmental Science* 7(3): 360–377.

Drai Dan and Yosef Grodzinsky 2006 A new empirical angle on the variability debate: Quantitative neurosyntactic analyses of a large data set from Broca's Aphasia. *Brain and Language* 96: 117–128.

Dromi, Esther, Laurence B. Leonard, Galit Adam, and Sara Zadunaisky-Ehrlich 1999 Verb agreement morphology in Hebrew speaking children with specific language impairment. *Journal of Speech and Hearing Research* 42: 1414–1431.

Duman, Tuba Y., Gülşat Aygen, Neşe Özgirgin, and Roelien Bastiaanse 2007 Object scrambling and finiteness in Turkish agrammatic production. *Journal of Neurolinguistics* 20(4): 306–331.

Eadie, P.A., M.E. Fey, J.M. Douglas and C.L. Parsons 2002 Profiles of grammatical morphology and sentence imitation in children with Specific Language Impairment and Down's syndrome. *Journal of Speech, Language, and Hearing Research* 45: 720–732.

Edwards, Susan 2005 *Fluent Aphasia*. Cambridge: Cambridge University Press.

Edwards, Susan and David Lightfoot 2000 Intact grammars but intermittent access. *Behavioral and Brain Sciences* 23(1): 31–32.

Fanselow, Gisbert and Sascha Felix 1987 *Sprachtheorie: Eine Einführung in die generative Grammatik. [Linguistic Theory: An Introduction into Generative Grammar, Volume II]*. Tübingen: Francke.

Faroqi-Shah, Yasmeen and Cynthia K. Thompson 2004 Semantic, lexical, and phonological influences on the production of verb inflections in agrammatic aphasia. *Brain and Language* 89: 484–498.

Ferreira, Fernanda 2003 The misinterpretation of noncanonical sentences. *Cognitive Psychology* 47(2): 164–203.

Fodor, Jerry A. 1983 *The Modularity of Mind*. Cambridge, MA: MIT Press.

Friedmann, Naama and Yosef Grodzinsky 1997 Tense and agreement in agrammatic production: Pruning the syntactic tree. *Brain and Language* 56: 397–425.

Friedmann, Naama and Rama Novogrodsky 2004 The acquisition of relative clause comprehension in Hebrew: A study of SLI and normal development. *Journal of Child Language* 31, 661–681.

Friedmann, Naama and Ronit Szterman 2006 Syntactic movement in orally trained children with hearing impairment. *Journal of Deaf Studies and Deaf Education* 11(1): 56–75.

Fromkin, Victoria A. 1971 The non-anomalous nature of anomalous utterances. *Language* 47(1): 27–52.

Fromkin, Victoria A. 1997 Some thoughts about the brain/mind/language interface. *Lingua* 100: 3–27.

Garraffa, Maria and Nino Grillo 2008 Canonicity effects as grammatical phenomena. *Journal of Neurolinguistics* 21: 177–197.

Gathercole, Susan and Alan D. Baddeley 1990 Phonological memory deficits in language impaired children: Is there a causal connection? *Journal of Memory and Language* 29: 336–360.

Goodglass, Harold, Julie Ann Christiansen, and Roberta Gallagher 1993 Comparison of morphology and syntax in free narrative and structured tests: Fluent vs. nonfluent aphasics. *Cortex* 29: 377–407.

Gopnik, Myrna and Martha B. Crago 1991 Familial aggregation of a developmental language disorder. *Cognition* 39: 1–50.

Grober, Ellen and Shereen Bang 1995 Sentence comprehension in Alzheimer's disease. *Developmental Neuropsychology* 11: 95–107.

Grodzinsky, Yosef 1984 The syntactic characterization of agrammatism. *Cognition* 16: 99–120.

Grodzinsky, Yosef 1990 *Theoretical Perspectives on Language Deficits*. London: MIT Press.

Grodzinsky, Yosef 1995 A restrictive theory of agrammatic comprehension. *Brain and Language* 50: 27–51.

Grodzinsky, Yosef 2000 The neurology of syntax. *Behavioral and Brain Sciences* 23(1): 1–71.

Grodzinsky, Yosef, Amy Pierce, and Susan Marakovitz 1991 Neuropsychological reasons for a transformational derivation of syntactic passive. *Natural Language and Linguistic Theory* 9(3): 431–453.

Grodzinsky, Yosef, Ken Wexler, Yu-Chin Chien, Susan Marakovitz, and Julie Solomon 1993 The breakdown of binding relations. *Brain and Language* 45: 396–422.

Grodzinsky, Yosef and Lisa Finkel 1998 The neurology of empty categories: Aphasics' failure to detect ungrammaticality. *Journal of Cognitive Neuroscience* 10(2): 281–292.

Guasti, Maria Teresa 2002 *Language Acquisition*. Cambridge MA: MIT Press.

Haegeman, Liliane 1991 *Introduction to Government and Binding Theory*. Oxford: Blackwell.

Hagiwara, H. 1995 The breakdown of functional categories and the economy of derivation. *Brain and Language* 50: 92–116.

Hamann, Cornelia, Zvi Penner, and Katrin Lindner 1998 German impaired grammar: The clause structure revisited. *Language Acquisition* 7(2-4): 193–245.

Hauser, Marc D., Noam Chomsky, and W. Tecumseh Fitch 2002 The faculty of language: What is it, who has it, and how did it evolve? *Science* 298 (5598): 1569–1579.

Heeschen, Claus and Herman Kolk 1988 Agrammatism and paragrammatism. *Aphasiology* 2, 299–302.

Heeschen, Claus and Emanuel Schegloff 2003 Aphasic agrammatism as interactional artefact and achievement. In: Charles Goodwin (ed.), *Conversation and Brain Damage*, 231–282. Oxford: Oxford University Press.

Heilman, Kenneth M. and Robert J. Scholes 1976 The nature of comprehension errors in Broca's, conduction and Wernicke's aphasics. *Cortex* 12: 258–265.

Hickok, Gregory and Sergey Avrutin 1995 Representation, referentiality, and processing in agrammatic comprehension: Two case studies. *Brain and Language* 50: 10–26.

Jakubowicz, Celia 2011 Measuring derivational complexity: New evidence from typically developing and SLI learners of L1 French. *Lingua* 121: 339–351.

Janssen, Ulrike and Martina Penke 2002 How are inflectional affixes organized in the mental lexicon? Evidence from the investigation of agreement errors in agrammatic aphasics. *Brain and Language* 81: 180–191.

Johnston, J.R. 1994 Cognitive abilities of language-impaired children. In: Ruth Watkins and Mabel Rice (eds.), *Specific Language Impairments in Children: Current Directions in Research and Intervention*, 107–121. Baltimore: Brookes.

Just, Marcel A. and Patricia A. Carpenter 1992 A capacity theory of comprehension: Individual differences in working memory. *Psychological Review* 98: 122–149.

Karmiloff-Smith, Annette 1998 Development itself is the key to understanding developmental disorders. *Trends in Cognitive Sciences* 2: 389–398.

Kernan, Keith and Sharon Sabsay 1996 Linguistic and cognitive ability of adults with Down's syndrome and mental retardation of unknown etiology. *Journal of Communication Disorders* 29: 401–421.

Kolk, Herman 1995 A time-based approach to agrammatic production. *Brain and Language* 50: 282–303.

Kolk, Herman and Claus Heeschen 1992 Agrammatism, paragrammatism and the management of language. *Language and Cognitive Processes* 7: 89–129.

Koopman, Hilda and Dominique Sportiche 1988 The position of subjects. *Lingua* 85: 211–58.

Kosslyn, Stephen M. and Michael Van Kleeck 1990 Broken brains and normal minds: Why Humpty Dumpty needs a skeleton. In: Eric L. Schwartz (ed.), *Computational Neuroscience*, 390–402. Cambridge, MA: MIT Press.

Lai, Cecilia S., Simon E. Fisher, Jane A. Hurst, Faraneh Vargha-Khadem, and Anthony P. Monaco 2001 A fork-head-domain gene is mutated in a severe speech and language disorder. *Nature* 413: 519–523.

Lapointe, Steven G. 1985 A theory of verb form use in the speech of agrammatic aphasics. *Brain and Language* 24: 100–155.

Lavorel, P.M. 1982 Production strategies: a systems approach to Wernicke's aphasia. In: Michael Arbib, David Caplan, and John C. Marshall (eds.), *Neural Models of Language Processing*, 135–164. New York: Academic Press.

Laws, Glynis and Dorothy Bishop 2003 A comparison of language abilities in adolescents with Down's syndrome and children with Specific Language Impairment. *Journal of Speech, Language, and Hearing Research* 46: 1324–1339.

Lee, Miseon 2003 Dissociations among functional categories in Korean agrammatism. *Brain and Language* 84(2):170–88.

Lee, Jiyeon, Lisa Milman, and Cynthia K. Thompson 2008 Functional category production in English agrammatism. *Aphasiology* 22(7/8): 893–905.

Leonard, Laurence 1995 Functional categories in the grammars of children with specific language impairment. *Journal of Speech and Hearing Research* 38: 1270–1283.

Leonard, Laurence 1998 *Children with Specific Language Impairment*. Cambridge, MA: MIT Press.

Leonard, Laurence, Susan Weismer, Carol Miller, David Francis, Bruce Tomblin, and Robert Kail 2007 Speed of processing, working memory, and language impairment in children. *Journal of Speech, Language, and Hearing Research* 50: 408–428.

Levy, Yonata 1996 Modularity of language reconsidered. *Brain and Language* 55: 240–263.

Levy, Yonata and Gitit Kavé 1999 Language breakdown and linguistic theory: A tutorial overview. *Lingua* 107: 95–143.

Levy Yonata and Jeannette Schaeffer (eds.) 2002 *Language Competence across Populations - Towards a Definition of Specific Language Impairment in Children*. Mahwah, NJ: Lawrence Erlbaum.

Liégeois, Frédérique, Torsten Baldeweg, Alan Connelly, David G. Gadian, Mortimer Mishkin, and Faraneh Vargha-Khadem 2003 Language fMRI abnormalities associated with *FOXP2* gene mutation. *Nature Neuroscience* 6(11): 1230–1237.

Lonzi, Lidia and Claudio Luzzatti 1993 Relevance of adverb distribution for the analysis of sentence representation in agrammatic patients. *Brain and Language* 45: 306–317.

MacWhinney, Brian and Elisabeth Bates 1989 *The Crosslinguistic Study of Sentence Processing*. Cambridge: Cambridge University Press.

Marin, Oscar S.M., Eleanor M. Saffran, and Myrna F. Schwartz 1976 Dissociations of language in aphasia: Implications for normal function. In: Steven R. Harnad, Horst D. Steklis, and Jane Lancaster (eds.), *Origins and Evolution of Language and Speech*, 868–884. New York: Academy of Sciences.

Martin, Randi C. 2006 The neuropsychology of sentence processing: Where do we stand? *Cognitive Neuropsychology* 23(1): 75–95.

Maurer, Gail, Victoria Fromkin, and Thomas L. Cornell 1993 Comprehension and acceptability judgements in agrammatism: disruptions in the syntax of referential dependency. *Brain and Language* 45: 340–370.

Meaburn Emma, P.A. Dale, Ian W. Craig, and Robert Plomin 2002 Language-impaired children: No sign of the *FOXP2* mutation. *Neuroreport* 13(8): 1075–1077.

Menn, Lise and Loraine K. Obler 1990a Cross-language data and theories of agrammatism. In: Lise Menn and Loraine K. Obler (eds.), *Agrammatic Aphasia: A Cross-Language Narrative Sourcebook, Vol. III*, 1369–1389. Amsterdam: Benjamins.

Menn, Lise and Loraine K. Obler (eds.) 1990b *Agrammatic Aphasia: A Cross-Language Narrative Sourcebook, Vol. I-III*. Amsterdam: Benjamins.

Müller, Ralph-Axel 1992 Modularism, holism, connectionism: Old conflicts and new perspectives in aphasiology and neuropsychology. *Aphasiology* 6: 443–475.

Nanousi, Vicky, Jackie Masterson, Judith Druks, and Martin Atkinson 2006 Interpretable vs. uninterpretable features: Evidence from six Greek-speaking agrammatic patients. *Journal of Neurolinguistics* 19(3): 209–238.

Neuhaus, Eva and Martina Penke 2008 Production and comprehension of *wh*-questions in German Broca's aphasia. *Journal of Neurolinguistics* 21: 150–176.

Niemi, Jussi and Matti Laine 1997 Syntax and inflectional morphology in aphasia: Quantitative aspects of Wernicke speakers narratives. *Journal of Quantitative Linguistics* 4(1-3): 181–189.

Ouhalla, Jamal 1993 Functional categories, agrammatism and language acquisition. *Linguistische Berichte* 143: 3–36.

Penke, Martina 1998 *Die Grammatik des Agrammatismus: Eine linguistische Analyse zu Wortstellung und Flexion bei Broca-Aphasie [The Grammar of Agrammatism: A Linguistic Analysis of Word Order and Inflection in Broca's Aphasia]*. Tübingen: Niemeyer.

Penke, Martina 2000 Unpruned trees in German Broca's aphasia. *Behavioral and Brain Sciences* 23(1): 46–47.

Penke, Martina 2001 Controversies about CP: a comparison of language acquisition and language impairments in Broca's aphasia. *Brain and Language* 77: 351–363.

Penke, Martina 2006 *Flexion im mentalen Lexikon [Inflectional Morphology in the Mental Lexicon]*. Tübingen: Niemeyer.

Penke, Martina 2008 Morphology and language disorder. In: Martin Ball, Michael Perkins, Nicole Mueller, and Sara Howard (eds.), *The Handbook of Clinical Linguistics*, 212–227. Oxford: Blackwell.

Penke, Martina and Marion Krause 2002 German noun plurals – a challenge to the Dual-Mechanism Model. *Brain and Language* 81: 303–311.

Penke, Martina and Anette Rosenbach 2007 What counts as evidence in linguistics? In: Martina Penke and Anette Rosenbach (eds.), *What Counts as Evidence in Linguistics – the Case of Innateness*, 1–50. Amsterdam: John Benjamins.

Perovic, Alexandra 2006 Syntactic deficit in Down's syndrome: more evidence for the modular organisation of language. *Lingua* 116: 1616–1630.

Pinango, Maria and Petra Burkhardt 2001 Pronominals in Broca's aphasia comprehension: The consequences of syntactic delay. *Brain and Language* 79(1), 167–168.

Pinker, Steven 1999 *Words and Rules*. New York: Basic Books.

Pinker, Steven 2002 *The Blank Slate: The Modern Denial of Human Nature*. New York: Viking.

Platzack, Christer 2001 The vulnerable C-domain. *Brain and Language* 77: 364–377.

Pollock, Jean-Yves 1989 Verb movement, Universal Grammar, and the structure of IP. *Linguistic Inquiry* 20: 365–424.

Reuland, Eric 2001 Primitives of binding. *Linguistic Inquiry* 32: 439–492.

Rice, Mabel L. and Ken Wexler 1996 Towards tense as a clinical marker of specific language impairment in English-speaking children. *Journal of Speech and Hearing Research* 39: 1239–1257.

Ring, Melanie and Harald Clahsen 2005 Distinct patterns of language impairment in Down's syndrome and Williams syndrome: The case of syntactic chains. *Journal of Neurolinguistics* 18: 479–501.

Rondal, Jean and Annick Comblain 1996 Language in adults with Down's syndrome. *Down's syndrome Research and Practice* 4(1): 3–14.

Rothweiler, Monika, Solveig Chilla, and Harald Clahsen accepted Subject verb agreement in Specific Language Impairment: A study of monolingual and bilingual German-speaking children. *Bilingualism: Language and Cognition*.

Rugg, Michael D. 1999 Functional neuroimaging in cognitive neuroscience. In: Colin Brown and Peter Hagoort (eds.), *The Neurocognition of Language*, 15–36. Oxford: Oxford University Press.

Schaeffer, Jeannette 2000 Aphasia research and theoretical linguistics guiding each other. *Behavioral and Brain Sciences* 23(1): 50–51.

Shallice, Tim 1988 *From Neuropsychology to Mental Structure*. Cambridge: Cambridge University Press.

Shattuck-Hufnagel, Stefanie 1979 Speech errors as evidence for a serial order mechanism in sentence production. In: William E. Cooper and Edward C.T. Walker (eds.), *Sentence*

*Processing: Psycholinguistic Studies Presented to Merrill Garrett*, 295–342. Hillsdale, NJ: Lawrence Erlbaum.

Schulz, Petra and Naama Friedmann (eds.) 2011 Specific Language Impairment (SLI) across languages: Properties and possible loci. Special issue of *Lingua* 121.

SLI Consortium 2002 A genomewide scan identifies two novel loci involved in specific language impairment. *American Journal of Human Genetics* 70: 384–398.

Stavarakaki, Stavroula and Heather Van der Lely 2010 Production and comprehension of pronouns by Greek children with specific language impairment. *British Journal of Developmental Psychology* 28: 189–216.

Stromswold, Karen 2001 The heritability of language: A review and metaanalysis of twin, adoption, and linkage studies. *Language* 77(4): 647–723.

Tager-Flusberg, Helen 1981 Sentence comprehension in autistic children. *Applied Psycholinguistics* 2: 5–24.

Tager-Flusberg, Helen 2002 Language impairment in children with complex neurodevelopmental disorders: The case of Autism. In: Yonata Levy and Jeannette Schaeffer (eds.), *Language Competence across Populations - Towards a Definition of Specific Language Impairment in Children*, 297–321. Mahwah, NJ: Lawrence Erlbaum.

Tager-Flusberg Helen, Susan Calkins, Tina Nolin, Therese Baumberger, Marcia Anderson, and Ann Chudwick-Dias 1990 A longitudinal study of language acquisition in autistic and Down's syndrome children. *Journal of Autism and Developmental Disorders* 20(1): 1–21.

Tallal Paula 1990 Fine-grained discrimination deficits in language-learning impaired children are specific neither to the auditory modality nor to speech perception. *Journal of Speech and Hearing Research* 33: 616–621.

Teuber, Hans-Lukas 1955 Physiological psychology. *Annual Review of Psychology* 9: 267–296.

Thal, Donna, Stacy Tobias, and Deborah Morrison 1991 Language and gesture in late talkers: A one-year follow-up. *Journal of Speech and Hearing Research* 34(3): 604–612.

Tomasello, Michael 2003 *Constructing a Language. A Usage-Based Theory of Language Acquisition*. Cambridge, MA: Harvard University Press.

Townsend, J., Beverly Wulfeck, S. Nichols and L. Koch 1995 *Attentional Deficits in Children with Developmental Language Disorder*. Technical Report CND-9503. Center for Rerearch in Language, University of California at San Diego.

Tsimpili, Ianthi Maria 2001 LF-interpretability and language development: A study of verbal and nominal features in Greek normally developing and SLI children. *Brain and Language*, 77: 432–448.

Uylings, Harry, Lidia Malofeeva, Irina Bogolepova, Katrin Amunts, and Karl Zilles 1999 Broca's language area from a neuroanatomical and developmental perspective. In: Colin Brown and Peter Hagoort (eds), *The Neurocognition of Language*, 319–336. Oxford: Oxford University Press.

van der Lely, Heather 1996 Specifically language impaired and normally developing children: Verbal passive vs. adjectival passive sentence interpretation. *Lingua* 98: 243–272.

Van der Lely, Heather 1997 Language and cognitive development in a grammatical SLI boy: Modularity and innateness. *Journal of Neurolinguistics* 10 (2/3): 75–107.

van der Lely, Heather 2005 Domain-specific cognitive systems: Insight from Grammatical-SLI. *TRENDS in Cognitive Sciences* 9(2): 53–59.

van der Lely, Heather and Linda Stollwerck 1997 Binding theory and specifically language impaired children. *Cognition* 62: 245–290.

van der Lely, Heather and Michael T. Ullman 2001 Past tense morphology in specifically language impaired and normally developing children. *Language and Cognitive Processes* 16(2/3): 177–217.

Vasic, Nada, Sergey Avrutin, and Esther Ruigendijk 2006 Interpretation of pronouns in vpellipsis constructions in Dutch Broca's and Wernicke's aphasia. *Brain and Language* 96, 191–206.

Wang, Paul and Ursula Bellugi 1993 Williams syndrome, Down's syndrome, and cognitive neuroscience. *American Journal of Diseases of Children* 147: 1246–1251.

Weigl, Egon and Manfred Bierwisch 1970 Neuropsychology and linguistics: Topics of common research. *Foundations of Language* 6: 1–18.

Wenzlaff, Michaela and Harald Clahsen 2004 Tense and agreement in German agrammatism. *Brain and Language* 89: 57–68.

Wexler, Ken 1994 Optional infinitives, head movement and economy of derivation. In: Norbert Hornstein and David Lightfoot (eds.), *Verb Movement*, 305–350. Cambridge: Cambridge University Press.

Wexler, Ken, Carson T. Schütze, and Mabel Rice 1998 Subject case in children with SLI and unaffected controls: evidence for the Agr/Tns omission model. *Language Acquisition* 7: 317–344.

Willmes, Klaus and Klaus Poeck 1993 To what extent can aphasic syndromes be localized? *Brain* 116(6): 1527–1540.

Wimmer, Eva 2010 *Die syntaktischen Fähigkeiten von Wernicke-Aphasikern. Eine experimentelle Studie. [Syntactic Capacities in Wernicke's Aphasia]* Dissertation University of Duesseldorf. <http://docserv.uni-duesseldorf.de/servlets/DocumentServlet?id=16808>.

## 6. Appendix

### ***Broca's aphasia***

Broca's aphasia is an acquired language disorder that is typically caused by strokes affecting anterior parts of the left hemisphere. The spontaneous speech production of Broca's aphasics displays a symptom called Agrammatism. Agrammatic speech is characterized by the following signs: (i) omission of free function words, (ii) problems with bound inflectional morphology leading to omissions and/or substitutions (see Penke 2008 for overview), (iii) reduction of sentence length, leading to the preponderance of one or two word utterances in spontaneous speech, (iv) occurrence of root-clause infinitives, and (v) reduced syntactic complexity, resulting in a preponderance of canonical sentence structures and a lack of more complex structures such as subordinate clauses, *wh*-questions or utterances with a topicalised constituent. In language comprehension, most Broca's aphasics display a better understanding of sentences with a canonical word order compared to sentences with non-canonical word order where the object has moved out of its base-generated position and precedes the subject.

Major controversies are concerned with the issue whether or not Agrammatism is due to a deficit in syntactic competence, and if so, how to account for this deficit within a theoretical approach (see sections 2. and 3.).

Suggested reading: Menn and Obler (eds.) (1990b); Penke (1998); Grodzinsky (2000); Avrutin (2001)

### ***Wernicke's aphasia***

The language disorder in Wernicke's aphasia is typically acquired as a result of strokes in left temporo-parietal brain regions. One of the core symptoms in this type of aphasia is so-called *Paragrammatism*. This symptom describes a speech which, although fluent, is characterized by repetitions of words and phrases, aborted sentences, word order errors and sentence blends. Moreover, Wernicke's subjects have severely impaired language comprehension abilities and general word retrieval problems which are indicated by semantic and phonological paraphasias and neologisms in naming and spontaneous speech. It has been suggested that this lexical-semantic deficit is the basis for Paragrammatism while syntax per se is intact. Wernicke's aphasia as a purely lexical disorder sparing syntax has thus been traditionally claimed to be the mirror image of agrammatic Broca's aphasia which has been postulated to be a syntactic disorder with an intact lexicon. The double dissociation between these two different aphasic language disorders that are prototypically associated with lesions in different brain areas has been considered as supporting the argument that grammar and lexicon constitute autonomously operating modules of the language faculty that can be independently impaired (cf. section 1.1). More recent studies with Wernicke's aphasics have however challenged the traditional view on the language deficits associated with Wernicke's aphasia. Thus, studies have provided evidence that complex syntactic structures such as *wh*-questions or passives are impaired in Wernicke's aphasics in both language production as well as comprehension (cf. Edwards 2005, Wimmer 2010). Moreover, the observed deficits and performance patterns often resemble the language deficits observed in Broca's aphasia. For instance, subject *wh*-questions are better comprehended and produced than object *wh*-questions in both German Broca's and Wernicke's aphasics (cf. Wimmer 2010). The structurally based problems in Wernicke's aphasia and the observed similarities to

Broca's aphasia cast doubt on the double dissociation postulated for the two aphasic syndromes. To date, the debate whether the grammatical difficulties of Wernicke's aphasics are due to their lexical-semantic deficit, an additional syntactic deficit or limitations in the language-processing capacity (cf. section 3) is controversial.

Suggested reading: Butterworth & Howard (1987); Edwards (2005); Wimmer (2010)

### ***Specific Language Impairment***

Specific Language Impairment is a cover term for delays respectively disorders of the normal acquisition of language that cannot be attributed to obvious neurological, cognitive, psycho-emotional or significant hearing impairments. Consequently, Specific Language Impairment has proven to be a heterogeneous disorder and has been divided into syndrome subgroups. Relevant for the issue of syntactic deficits is the syndrome subgroup called Grammatical Specific Language Impairment that is characterised by problems in the acquisition of morphosyntax. Core symptoms of Grammatical Specific Language Impairment include impairments with inflectional morphology and difficulties with producing or interpreting complex sentences, such as subordinate clauses, relative clauses or passives.

Whether the language impairments in Grammatical Specific Language Impairment are due to a representational deficit affecting syntactic structures or relationships and if so how to account for them (cf. section 2) , or whether the observed impairments are due to a processing deficit that affects language processing capacities (cf. Leonard 1998) is a major issue of controversy in the field. A related debate concerns the question whether the language deficits of children with SLI are language-specific, or whether they can be attributed to domain-unspecific cognitive impairments, such as deficits in rapid auditory discrimination, in symbolic play, in nonverbal attention or in spatial imagery (cf. Tallal 1990; Thal, Tobias, and Morrison 1991; Johnston 1994; Townsend et al. 1995). Note, however, that the crucial issue here is not whether children with SLI have any non-linguistic cognitive deficits, but whether these deficits can explain the observed language impairments.

Suggested reading: Leonard (1998); Levy and Kavé (1999); Levy and Schaeffer (eds.) (2002); Van der Lely (2005) and Schulz and Friedmann (eds.) (2011)

### ***Williams syndrome***

Williams syndrome is a rare neurodevelopmental disorder of genetic origin that is associated with physical (e.g. renal and cardiovascular) anomalies, a global cognitive impairment and selective deficits in non-linguistic cognitive domains such as visuospatial constructive cognition where they display marked problems in constructing or drawing a coherent Gestalt.

One of the central controversies in the investigation of Williams syndrome concerns the issue whether or not there is a dissociation between grammatical knowledge and other cognitive skills in individuals with this syndrome. Children with Williams syndrome show normal language skills with respect to regular inflectional morphology and the production respectively comprehension of complex sentence structures (e.g. subordinate clauses, passive clauses, tag questions, anaphors) despite severe mental retardation (Bellugi et al. 2000; Clahsen and Almazan 1998). The purported dissociation between intact grammar skills and impaired general cognitive capacities observed in this disorder has been taken as evidence that the human language faculty is autonomous from other cognitive domains and that the properties of the language faculty cannot be put down to the operation of domain-

general principles (see Bellugi et al. 2000). This view has, however, been challenged by researchers who dispute the autonomy of language from general cognitive capacities. These researchers have tried to show that grammatical abilities are not spared in affected children and, hence, are not dissociated from general cognitive capacities (Karmiloff-Smith 1998). Moreover, it has been suggested that children with Williams syndrome undergo a different neural and cognitive development, resulting in the construction of a qualitatively different grammatical system in the brain. Note that if the grammatical system of individuals with Williams syndrome were indeed qualitatively different from the 'normal' system, nothing could be concluded on the basis of data from Williams syndrome about the 'normal' language faculty, for instance about its autonomy from general cognition (Karmiloff-Smith 1998).

Suggested reading: Bellugi and St George (eds.) (2000); Bartke and Siegmüller (eds.) (2004)

### ***Down's syndrome***

Down's syndrome is a congenital neurodevelopmental disorder caused by a third copy of chromosome 21 that leads to moderate mental retardation and characteristic physiological traits. The language deficits of individuals with Down's syndrome display characteristics similar to deficits observed in Specific Language Impairment. In speech production, free grammatical morphemes are often omitted (Eadie et al. 2002) and the production of bound grammatical morphemes is impaired (Laws and Bishop 2003). In language comprehension, deficits arise in the comprehension of passive sentences and in establishing grammatical binding relations between anaphors and their referents (Ring and Clahsen 2005; Perovic 2006).

A critical issue regarding the language deficits in Down's syndrome is whether the general limitation of cognitive capacities in individuals with Down's syndrome is also responsible for the observed language deficits. If this were the case, claims regarding the modularity and autonomy of language capacities from general cognitive capacities (cf. Fodor 1983) would be severely threatened. To investigate this issue, language capacities of individuals with Down's syndrome are compared to language capacities of individuals who have a similar level in nonverbal cognitive development without suffering from Down's syndrome. These can be unimpaired younger children or children with mental retardations due to a different neurodevelopmental disorder, such as individuals with Williams syndrome. A language performance that differs quantitatively and/or qualitatively from the performance of mental-age-matched controls is seen as indicative of an indigenous language deficit because the deficit surpasses a level that could be accounted for by the level of cognitive development. Especially the differences in capacities and deficits observed in individuals with Down's syndrome and individuals with Williams syndrome indicate that language capacities are independent from general cognitive capacities since a comparable general cognitive impairment affects language differently in these two syndromes.

Suggested reading: Wang and Bellugi (1993); Kernan and Sabsay (1996); Chapman et al. (1998); Bellugi et al. (2000)

## Notes

---

- <sup>1</sup> Whenever the term Specific Language Impairment is used in the text, this term refers to the subgroup of this deficit syndrome classified as Grammatical Specific Language Impairment (cf. appendix).
- <sup>2</sup> That our species must be in some way biologically prepared to acquire language is today widely accepted among researchers (cf. Pinker 2002: §1; Tomasello 2003: 284). The debate rather focuses on what this innate language capacity precisely consists of, and whether it is specific to language and independent of other cognitive capacities (i.e. domain-specific) or whether it is based on domain-general principles shared with other cognitive domains (cf. e.g. Hauser, Chomsky, and Fitch 2002 and the controversies started by this article; Pinker 2002 and Penke and Rosenbach 2007 for overview).
- <sup>3</sup> The assumptions of modularity and autonomy are, for instance, refuted in functional approaches to language that reject the notion of a domain-specific language capacity that performs language-specific operations on language-specific representations (cf. Penke and Rosenbach 2007). The fractionation assumption has been challenged on the ground that lesions which consistently affect only one particular language structure or operation while sparing another one seem somewhat implausible considering the huge variability of brain lesions observed between individuals suffering from the same disorder syndrome (e.g. Marin, Saffran, and Schwartz 1976; Müller 1992). The transparency assumption is for example threatened by an individual's ability to compensate her/his language deficit, resulting in language data that might mirror the application of adaptation strategies rather than the impairment of the affected component (e.g. Marin, Saffran, and Schwartz 1976; Heeschen and Kolk 1988). For a discussion of these arguments see Caramazza (1992) and Penke (2006: §2.3).
- <sup>4</sup> Meanwhile, a number of studies have provided evidence that syntax is not unaffected in Wernicke's aphasia but that the deficits seen in Wernicke's aphasia largely resemble the impairments observed in Broca's aphasia (see appendix and e.g. Edwards 2005; Wimmer 2010 for overview). These findings challenge the view of a double dissociation between syntactic and lexical-semantic deficits in Broca's and Wernicke's aphasia.
- <sup>5</sup> The term double dissociation (Teuber 1955) describes the following situation: for two entities X and Y, deficit syndrome A impairs X and spares Y, whereas deficit syndrome B spares X and impairs Y (cf. Shallice 1988; Penke 2006: §2.3 for discussion).
- <sup>6</sup> Note, however, that individuals with Williams syndrome do not seem to display such problems (Clahsen and Almazan 1998; Ring and Clahsen 2005).
- <sup>7</sup> The figure depicts the standard double-movement analysis of German V2 movement (cf. Fanselow and Felix 1987; Haegeman 1991). In main clauses, the verb starts out in the VP-final V position and successively moves over the INFL node, where it incorporates its finite inflectional morphology, to the C position. The subject moves to SpecIP to realize its nominative case features and to enter into an agreement relation with the finite verb in INFL. In addition, the subject or any other constituent has to move to SpecCP to ensure that the finite verb ends up in the second structural sentence position (SVX or XVS word order).
- <sup>8</sup> A later version of the Extended Optional Infinitive Hypothesis has been reformulated to encompass underspecification of the tense and/or the agreement features (Wexler, Schütze, and Rice 1998). See Clahsen (2008) for discussion.
- <sup>9</sup> See Tsimpli (2001) for a broader deficit account which assumes a deficit in the accessibility of all uninterpretable features – not just the verb's phi-features – in individuals with Specific Language Impairment.

- 
- <sup>10</sup> Grodzinsky and colleagues have suggested that the dissociation between impaired comprehension of passives and spared comprehension of actives observable in agrammatic aphasia argues for a syntactic approach to capture passives as developed in the framework of Government and Binding and discussed in the text. They argue that syntactic frameworks such as Lexical Functional Grammar or Generalised Phrase Structure Grammar where the derivation of passives does not involve movement but a lexical derivation can not account for this very circumscribed deficit in agrammatic comprehension. Hence, they suggest that these latter approaches lack breakdown compatibility and are, therefore, inferior to the framework of Government and Binding (cf. Grodzinsky, Pierce, and Marakovitz 1991).
- <sup>11</sup> A different theoretically based challenge is posed by the fact that the recent framework of the Minimalist Program discards with movement operations which Grodzinsky explicitly sees at the basis of agrammatic comprehension problems (cf. Edwards and Lightfoot 2000).
- <sup>12</sup> Because of the challenges the integration of the VP-Internal Subject Hypothesis poses to the Trace-Deletion Hypothesis, Schaeffer (2000) has proposed to take the dissociation between impaired comprehension of sentences including object traces and spared comprehension of sentences with subject traces in agrammatic aphasia as evidence that the VP-Internal Subject Hypothesis is not valid.
- <sup>13</sup> Note that Grodzinsky (2000) has advocated the view that language production and language comprehension might be differently affected in Agrammatism. Whereas he assumes syntactic representations to be pruned in production, comprehension is claimed to suffer from the deletion of traces in syntactic representations. Such a move, however, presupposes the existence of two independent syntactic representations that are targeted by different language processes (comprehension vs. production) and that can be selectively affected in language disorders – a picture that deviates from the standard Chomskyan conception of an abstract grammatical competence that is put to use in language production *and* comprehension (Chomsky 1980).
- <sup>14</sup> Likewise, the finding that sentences with non-canonical structure are often misunderstood by unimpaired subjects when put under stressful conditions such as noise is also regarded as evidence for a continuum connecting performance in language impaired and unimpaired speakers and, hence, for the validity of accounts that assume impaired processing capacities while syntactic representations are unaffected (cf. Dick et al. 2001).