A discussion of “linguistic” approaches to agrammatic disorder studies

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<thead>
<tr>
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<tbody>
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A discussion of “linguistic” approaches to agrammatic disorder studies

I. Introduction
This paper is intended to be an overview of a trend in research into agrammatism. However, it is not going to be a totally indiscriminate or unbiased overview - mostly because the size of the literature does not allow this. My primary goal is not so much to provide a database of research into agrammatism, but to give a snapshot of a direction in research of brain damaged ‘agrammatic’ subjects that I consider influential. I will argue that there are inherent problems with this avenue of research. However, this paper is not an attempt at arguing that research into syntactic performance of agrammatics is meaningless. I believe that one can use patient syntactic performance to forward certain models of linguistic functional architecture, however, it has to be done in a way scientific inquiry is usually done: one constructs a hypothesis which is empirically verifiable, then it is tested and either confirmed or refuted. Unfortunately, this simple practice used often in hard sciences seems to have been abandoned for a different approach where researchers arbitrarily designate a set of evidence and then provide an account for it without considering potential counter-evidence and more importantly potentially simpler accounts. Giving a novel account for a small subset of data that revises well established theories is not something unusual, relativity is a good example. However, I will argue that this is not the case with theories of agrammatism. First of all there is no well established theory, in fact I will argue it is not clear of what this would be a theory of since agrammatism is not a well defined phenomenon. However, even assuming that agrammatism as a syndrome is a real phenomenon, the approach I will discuss will be argued to be internally incoherent and thus problematic independently of the fact that this family of proposals has failed to undergo empirical verification.

This paper has three major sections. First I will discuss problems connected with attempts at establishing what agrammatism is and what it isn’t, and whether one can classify it as a syndrome. Afterwards I will explore a family of ‘linguistic’ theories of agrammatic comprehension put forward by Grodzinsky (1990, 1995). I will concentrate on the internal inconsistencies of the proposal and will attempt to see if one can extract any predictions that can

1 I would like to thank Alfonso Caramazza and Noam Chomsky for their comments. All errors are obviously mine.
be made on the basis of these proposals. Finally, I will examine these empirical predictions discussed in the previous section and will argue that they are false.

II. What is Agrammatism

The division of subjects into those who exhibit fluent and grammatical speech but with numerous substitutions and into those who not have syntax but are capable of retrieving correctly content words has become a standard textbook distinction. The former are considered to be paragrammatics (a disorder connected with Wernicke’s syndrome) and the latter agrammatics (connected with Broca’s aphasia). Initially this division was more or less clear cut, however, as more and more research was carried out on both groups of patients, it became evident that the clinical description involves a very diverse group of symptoms.

There is no single clear cut definition of what agrammatism is. Distinctive properties involve laboured speech and a lack of use of grammar in production and/or comprehension. Production is typically characterised by so called ‘telegraphic speech’ that involves the lack of function words and inflection. Sentences are usually simple two three word constructions.

The speech of agrammatic patients clearly indicates the lack of markers indicating the organisation of sentences and the number of closed class items in the speech of agrammatics is relatively small. Garrett (1992) notes that there is a reason to classify lexical items into two distinct classes and this division into two classes of lexical items is visible in the contrast between two kinds of disorders: agrammatism and paragrammatism.\(^2\)

Patients exhibiting agrammatism exhibit also comprehension deficits (Caramazza & Zurif 1976, Linebarger, Schwarz & Saffran 1983, Zurif 1990). Comprehension is good for single words but breaks down with sentences involving complex thematic relation mapping. Thus, reversible sentences where thematic roles of the two arguments can be switched around pose most problems.\(^3\)

Caramazza & Zurif (1976) found that non-canonical word orders of these reversible sentences pose most problems in comprehension tasks of agrammatics, thus sentence (2) will be more difficult to comprehend than sentence (1):

1. The horse kicked the giraffe
2. The giraffe was kicked by the horse

\(^2\)Garrett (1992) classifies agrammatism as a disorder of production.

\(^3\)Examples of such sentences are: ‘The horse kicked the giraffe’ vs. ‘The giraffe kicked the horse’. Irreversible ones are: ‘The boy opened an envelope’.
It has to be underlined, however, that these characteristic properties of agrammatism are not present in every patient that is classified as agrammatic. Thus it is hard to establish a core set of properties that can be clearly defined as characterising agrammatism.

Additionally, the discussed comprehension difficulties are not typical just of patients that are classified agrammatic. Non-agrammatic patients also seem to have problems in comprehending reversible sentences with non-canonical word orders (Caplan & Hildebrandt 1988).

Moreover, it has been shown that agrammatic patients exhibit intuitions concerning the well-formedness constraints that govern the distribution of closed class elements (Linebarger et. al. 1983, Saddy 1990, Schwartz et. al. 1987, Shankweiter et. al. 1989). Such observations imply that there is some access to information concerning closed class elements even when there is a comprehension and/or production deficit.

<table>
<thead>
<tr>
<th>Description</th>
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<tr>
<td>Problems in production of function words, but verbal inflection intact,</td>
<td>Miceli et. al. (1983), Saffran et. al. (1980)</td>
</tr>
<tr>
<td>although the verbal form might be semantically inappropriate.</td>
<td></td>
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<tr>
<td>Problems with verbal inflections.</td>
<td>Goodglass (1976)</td>
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<tr>
<td>Trouble with production of auxiliary verbs and weak pronouns, but less</td>
<td>Nespoulous et. al. (1988).</td>
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<tr>
<td>trouble with other function words.</td>
<td></td>
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<tr>
<td>Patients have more problems in processing English possessive and third</td>
<td>Goodglass &amp; Berko (1960)</td>
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<tr>
<td>person [s] morpheme than the plural [s] morpheme.</td>
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<tr>
<td>Inflectional endings more prone to stranding errors than derivational</td>
<td>Kean (1977)</td>
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<td>endings.</td>
<td></td>
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<tr>
<td>Function words that follow phonologically stressed words have a better</td>
<td>Goodglass (1976)</td>
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<tr>
<td>chance of being correctly produced than words following unstressed words.</td>
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Table 1. A selection of agrammatic disorder symptoms

<table>
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<th>Symptom</th>
<th>Source</th>
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<td>Problems with processing function words only when produced within a sentence vs correct production of these words in isolation.</td>
<td>Caramazza &amp; Hillis (1989)</td>
</tr>
<tr>
<td>Problems with production of complex NP’s and embedded verbs.</td>
<td>Menn et. al (1990)</td>
</tr>
<tr>
<td>Problems with the production of certain prepositions</td>
<td>Rizzi (1985)</td>
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Table 1. Provides only a sample of the disorders that have been reported to be connected with agrammatism. It is by no means a comprehensive list and it serves only to indicate the diversity of clinical data that is subsumed under this deficit.

Badecker & Caramazza (1985) point out the classification of subjects as agrammatics is based solely on clinical intuitive judgements. In other words, there is no theory of processing that would be the basis of classifying whether a given subject is agrammatic or not. This calls into question whether one can talk about agrammatism as being a psychological disorder.

It is clear that attempts to account for the ever growing facts connected with this disorder have not really considered the possibility that agrammatism as such cannot be accounted for by a single deficit of processing, syntax or phonology. For example, it remains unclear whether production and comprehension deficits can be assumed to have a similar underlying deficit.

There is no precise theory of production or comprehension and the interaction between the two. Moreover, it seems that the set of comprehension deficits of agrammatics is not a mirror image of the set of production deficits. I know of no reports claiming that agrammatics have problems in comprehending tense or aspect, or having problems in comprehending pronouns or prepositional phrases although such deficits have been reported in production, see for example Grodzinsky 1990). Furthermore, certain comprehension deficits have not been mirrored in production. Thus a production problem mirroring comprehension problems involving psych verbs (Grodzinsky 1990) has to the best of my knowledge never been reported. This of course does not imply that maybe certain deficits will turn out to be visible in subject’s comprehension and production. It is also not obvious whether there is need for these two sets of deficits to be mirror images of each other. Such phenomena remain unclear for the reason that there is no theory behind the deficit of agrammatism. This has been clearly argued for in Badecker & Caramazza (1985). All we have is a set of facts that has been selected on the basis of certain intuitions of various researchers.
There have been attempts to make sense out of the various approaches taken to study agrammatic phenomena. Badecker & Caramazza (1985) classify current research on agrammatism as basically stemming one of two approaches. The first kind of approach, called by them the empirical approach, assumes that a given symptom constitutes the crucial element in defining the deficit and remains at the same time, the central of all possible deficits. Such an approach would entail that any patient exhibiting the central symptom would be considered as agrammatic. This, in turn, implies that the variety of non-central symptoms exhibited by different agrammatic patients will have to be sieved through and classified as being related to the central symptom or not. Then the determined subset of symptoms as well as the central symptom would have to be accounted for. This is a very atheoretical approach since it is not clear what criteria will be used for determining the subset of correlated symptoms.

Another approach would be to a priori define agrammatism as involving a given symptom, for example, the omission of function words, regardless of whatever other symptoms a given patient might exhibit. Badecker & Caramazza (1985) argue that such a definitional approach runs into certain inherent problems. Primarily, it makes it impossible to empirically verify what constitutes agrammatism since “we could not provide evidence for a psychologically objective category - i.e., one whose existence is owed to the structure of the underlying cognitive mechanisms - because any instance of this system would, by definition, be an indication of the syndrome” (Badecker & Caramazza 1985:104).

The empirical approach has one central problem, namely that we have to have a theoretical basis for establishing the central deficit. It is possible to assume that the empirical approach involves the constant narrowing down of the crucial elements of the deficit. Thus initial attempts at establishing what agrammatism is will be inherently too broad. Badecker & Caramazza (1985) point out that in order for such an approach to be valid one has to be able to establish the degree of variation within a given syndrome as opposed to variation across syndromes. Also one has to establish an objective set of criteria according to which we would be able to select what constitutes a syndrome.

As it has been pointed out in previous sections, the variation of symptoms attributed to agrammatism is fairly wide. In fact, we cannot even be certain that the central assumption concerning the omission of function words and inflections can be upheld, since there are reports where inflection and function words are relatively preserved.
Zurif and Piñango (1999) argue for something that seems to resemble this narrowing down strategy. For them the location of the neuroanatomical lesion plays the central role in selecting patients. Thus Zurif and Piñango (1999:137) in response to Berndt and Caramazza (1999) and Berndt et. al (1996) argue that: “if following Berndt et. al. (1996), one seeks a comprehension pattern for patients labelled only agrammatic aphasics the search is unrewarding [...] we see no reason to pursue the Berndt and Caramazza approach - at least not if the aim is to relate aspects of language processing to neuroanatomical organisation.”

Zurif and Piñango (1999) further argue that “agrammatism on its own does not implicate left anterior cortex in the manner Broca’s aphasia does”.

In other words criticisms of patient selection failed to take into account that potential counter-example patients are not Broca’s aphasics, whereas the proposed theory only applies to those agrammatics who are Broca’s aphasics.

Zurif and Piñango also point to neuro-imagining studies seem to indicate that Broca’s region is especially involved in syntactic processing. It exhibits activation as a function of syntactic complexity (Caplan et. al 1998) and in contexts involving canonical non-canonical word order contrasts (Cooke et. al. 1999).

Even assuming that these preliminary neuro-imaging studies turn out to be correct in predicting the claimed correlation, we are still faced with the same problem of patient selection. We have narrowed down the group and maybe eliminated a few counter-examples. However, the problem concerning whether agrammatism is a syndrome remains.

II.1 What is a syndrome?

Badecker & Caramazza (1985) discuss three different possible understandings of what constitutes a syndrome. The weak sense of a syndrome involves an assumption that a statistical correlation of certain symptoms can constitute the basis for establishing a syndrome. A strong sense of a syndrome is established in terms of the possible deficits to given components of the processing system. “Unlike a syndrome-in-the-weak-sense, a syndrome-in-the-strong-sense is not identified directly with the various patterns of performance of individual members of this group or category, but with those aspects of performance which result from the particular deficits to the normal system in terms of which the syndrome is defined” (Badecker & Caramazza 1985:113).

Badecker & Caramazza advocate a weaker reading of the strong definition of a syndrome since they allow two patients to be classified as having the same syndrome even if the damaged and undamaged systems of both patients are not absolutely identical. Badecker & Caramazza (1985:113) argue that “so long as all and only the patients categorised by syndrome X are
impaired by means of damage to a specific fixed set of processing components, we can still speak of X as a syndrome which corresponds to those aspects of performance that can be attributed to the deficits in those particular components”.

If we assume such a definition, the status of agrammatism as a syndrome becomes questionable.

III. “Linguistic” accounts of the “agrammatic deficit”.  

I hope to have shown that attempting to establish the nature of the agrammatic disorder if far from easy. Let me, however, assume that there is some way to clarify these issues since this will allow me to discuss an approach which is based on such an assumption.

The family of theories proposed in Grodzinsky (1990, 1995) has become influential in studies of agrammatic comprehension. Unfortunately, these proposals seem to expose the problems involving studies of agrammatic patients and where there is a very real threat of a mismatch between complex proposals and empirical/conceptual grounding. The theory concerns the underlying deficit of agrammatic comprehension, although one must note that production was considered to be the characteristic trait of agrammatics.

Grodzinsky (1986, 1990, 1995) proposed a series of accounts of agrammatic deficits making use of linguistic models of the human knowledge of grammar dubbed generally as a Government and Binding (Chomsky 1981, 1982). I will refer to Grodzinsky’s (1990) version of this account, but the problems raised here will pertain to all versions. Grodzinsky proposes that specific damage to the syntactic representation adopted of an agrammatic is supposed to give predicted comprehension asymmetries. Crucially, damage to this module forces comprehension to bypass the damaged syntactic module and use a simple heuristic model of comprehension where the linear order of heard NP’s becomes mapped to a linear order of thematic roles: Agent and then Patient.

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4 I will use the term agrammatic deficit just as reference to whatever set of performance characteristics researches assume as being the deficit. I hope to have shown that there does not seem any coherent set of properties which could be argued to constitute a syndrome, thus my use of the term agrammatic will just be descriptive and will refer to whatever patient is considered by a given researcher to belong to this category. The fact that there does not seem to be a independent set of criteria and patients are not administered identical comprehensive tasks makes it impossible to use the term in any other way.
I will concentrate on the proposed account of comprehension in agrammatics, although Grodzinsky (1990) also gives a GB account of production. However, since then the research effort and controversies seem to have concentrated on comprehension.

Before I discuss Grodzinsky’s proposals in detail, let me quickly outline the linguistic theory behind the proposal. This is crucial since much of the problems in Grodzinsky’s proposals seem to stem from a misunderstanding what this theory is. Government and Binding frameworks assume that phrase structure is generated by a simple algorithm called X-bar structure. You take a head $X^0$ which can be a noun, verb or other category, you project X-bar levels until you create $X^{\text{max}}$ (equivalent of an XP, where $X$ could equal N, A, V, etc.) which is the appropriate phrase headed by the chosen $X^0$. X-bar theory accounts for how phrase structure is created. Crucially, the given head determines to a large degree the type and amount of its complements and is detrimental in generating the appropriate phrase structure. Thus for example, verbs like ‘give’ require two objects and subject and thus an appropriate phrase structure is generated from the verb head (Grimshaw 1990):\(^5\)

\[ V^{\text{max}} = \text{VP} \]

\[
\begin{array}{c}
\text{NP} \quad \text{John} \\
\text{V'} \\
\text{NP} \quad \text{a book} \\
\text{V'} \\
\text{NP} \quad \text{Mary} \\
\text{V}^0 \quad \text{gave}
\end{array}
\]

The above structure is generated from the head and is determined by the sub-categorisation frame

\(^5\) This is a simplified view of things, but sufficient for our purposes.
of the verb. The verb then assigns è-roles to its arguments so they can be interpreted as Patient, Recipient, etc.

However, linguistic structure also involves displacement of phrases and heads. Thus, for example, in question formation in English the wh-word is usually clause initial. However, it is assumed that this is not where the phrase originated from.

3. Who did John kiss?
* 4. Who did John kiss Mary
* 5. John kissed

The triple contrast between (3), (4) and (5) illustrates that the verb ‘kiss’ requires an NP complement (5). Sentence (3) seems to violate that, however, it is grammatical. Furthermore, (4) is bad although ‘kiss’ has a complement. The dilemma is solved if we assume that ‘who’ was at some level of representation (called Deep Structure) a complement of kiss and then underwent a transformation which caused it to be spelled out at the beginning of the clause (so called Surface Structure). Surface structure is postulated to contain information about such transformations, thus we assume that there is a ‘trace’ in the original complement position:

6. Who₁ did John kiss t₁

There are certain conditions involving the relationship between the trace and its antecedent that constrain the types of movement. Details of this are, however, irrelevant here. What is important is that Grodzinsky (1995) proposes that agrammatic patients cannot recover the è-marked trace at surface structure.⁶ This implies that thematic structure is lost in the representation and comprehension cannot make use of generated linguistic representation. Comprehension can take place since there is an independent extralinguistic heuristic algorithm that can assign thematic interpretations to sequences of NP’s on the basis of their linear order. Thus Agent is assigned to the first NP and Patient to the second. This reflects the fact that English is SVO. This heuristic procedure is activated in agrammatics since normal procedures are damaged, however, it is not clear what is the locus of these procedures. Grodzinsky does not place the damaged module in the functional architecture of language processing. Thus we are not clear whether the processor

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⁶ One aspect that differentiates Grodzinsky (1995) from Grodzinsky (1990) is that the newer version assumes that only è-marked traces are deleted/unaccessible.
or the grammar (understood as a set of representations of linguistic knowledge) is damaged. It would seem natural to assume that what he is talking about is not the processor but the representation of linguistic knowledge, this is because the model he adopts is identical to the model of linguistic knowledge adopted in Chomsky (1982, 1986). It has to be stressed that Government and Binding is not a theory of language sentence processing, be it production or comprehension, and the link between knowledge of language and language processing is undefined.

Keeping this in mind, let us examine in more detail Grodzinsky’s claims concerning the lesion responsible for agrammatic comprehension.

He notes that agrammatics comprehend above chance in active sentences and below chance in passive sentences. This he argues is a result of a lesion in being able to maintain traces of ê-positions. A moved element, if it is an argument (subject and object) will have its first trace in an ê-position. The elimination of these traces will make it impossible to establish what is the Agent and what is the Patient of a simple active or passive clause:

7. John hit Mary
8. Mary was hit by John

However, the heuristic algorithm that I already mentioned that establishes the first NP to be a subject/Agent will correctly establish the meaning of (7) but not (8). Thus agrammatics’ above chance performance is predicted in actives. This proposal implies that any type of information involving movement (for example case and agreement between arguments and verbs) should be non-existent.

7 I will not discuss here whether it is productive to have such a distinction, what is crucial here is that the Government and Binding model cannot and is not assumed in verbatim to be a description of the language processor.

8 Note that there is an implicit assumption that one single comprehension deficit is a core property of agrammatic performance. This implies the empirical approach, as defined by Badecker & Caramazza (1985) with all the negative consequences implied in their paper.

9 This is problematic for the account of agrammatic production in Grodzinsky (1990). He proposes that the lack of agreement and tense morphology is a result that these features are deleted in INFL - an abstract syntactic head that licences agreement and tense. Crucially, this licensing take place via movement, however, we cannot have movement if trace deletion takes place and nothing else but a truncated phrase structure is present. Obviously this is assuming that what we are talking about is the GB model where comprehension and production share the same representation.
Thus, agrammatics should not be capable of recovering any information except phrase structure. Again, it is hard to establish what that means unless we know what the damaged representation is. It cannot be what linguists call Surface Structure since Surface Structure is identical for comprehension and production and thus everything else being equal there should be no dissociation between comprehension and production deficits.\(^\text{10}\)

It is also crucial to note that the elimination of traces in è-role positions predicts that the lexical representation of verbs is not met. This is because sub-categorisation is considered to be closely connected with è-role assignment. If we have deletion of traces at Surface Structure then sub-categorisation requirements of verbs are no longer being met. Obviously, we could assume that sub-categorisation needs only to be satisfied at Deep Structure. However, then would have nothing blocking movement of an NP into the position of the just deleted trace. We could imagine the subject of a subordinate clause raising to the matrix clause object position - something like an ECM construction but with an overt object already raised higher.\(^\text{11}\)

\*9. John was informed by the Police Roger\(_1\) that t\(_1\) was a criminal
\*10. John was hit by the Police Mary

Sentences like (9) are unlikely to be produced or tested on patients - they are too complex. However, violations of sub-categorisation should give errors like (10) in production or grammaticality judgements. To the best of my knowledge there have been no reports of production deficits violating sub-categorisation. More importantly, there are reports of correct grammaticality judgements by agrammatics involving sub-categorisation. This means that something is constraining sub-categorisation at Surface Structure. This means that in order for any constituent structure to be present, we need to assume that the deficit in comprehension does not only involve the elimination of theta role traces but also selective damage to the verb’s sub-categorisation frame. It has to be selective since in cases of

\(^{10}\) Obviously the catch word is ‘everything else being equal’, in brain damaged patients that is highly unlikely. However, before we are presented with a model that accounts for the lack of exact performance in both modalities, we must assume that the theory is lacking some component. Otherwise we would have to give the benefit of the doubt and assume that the details can be worked out. However, this implies that no predictions can be made since there is always a chance that some yet unknown factor is responsible for the existence of potential counterexamples.

\(^{11}\) E(xceptional) C(ase) M(arking): cases where the subject of a subordinate clause receives accusative case from the verb of the matrix clause: “He wants her to go home”
subjects in canonical word orders there is no deficit.

Again, it has to be stressed that the proposals in Grodzinsky (1990, 95) are vague on what is the level of representation that is damaged. It looks like Surface Structure, but its properties are completely different. It is not the language processor, or if it is, it is a very unusual one since it is basically a carry over of a theory of linguistic representation that says nothing about real time processing.

Current linguistic research has come closer to a model which could be considered to be compatible with an assumption that it closely interacts with the real-time processor or is in fact a model of syntactic processing. Chomsky (1995) proposed to discard X-bar theory of phrase structure and replace it with an approach where phrase structure is created with the help of two processes Merge which takes two elements and creates a new set and Move which also involves Merge but differs in that both elements are already part of the created phrase structure. The crucial difference for us is that there is no independent module that creates phrase structure frames to which lexical items are attached. Phrase structure is created during the derivation from lexical items (which include abstract phrase heads like Tense or Complementiser). The model is a more dynamic approach to syntactic derivation. It is not a model of real-time processing, but is considered to interact very closely with the language processor through an operation called cyclic Spell-Out where parts of the representation are passed on for further processing. It is not a clearly spelled out proposal as far as the interaction between syntactic structure building operations and on-line processing, although unlike GB, it at least attempts to address these issues. What is important for the discussion here is that a deficit that deletes traces, which are no longer a separate entity from lexical items but actually copies of these items, will cause the whole derivation to irreparably crash and even phrase structure will no be salvaged. This is because movement builds phrase structure and both require valid traces. Thus, if one carries over Grodzinsky’s proposal to recent development in linguistic theory, one will have to assume that comprehension and production is totally asyntactic and any syntactic regularity that one sees in production or comprehension is totally accidental or a result of some other mechanism. Note that the simple heuristic procedure of identifying what is an Agent and what is Patient of a clause is not sufficient since it cannot account for simple things like word order, for example. Of course one might say that research into agrammatism provides crucial evidence that the GB approach is superior to the current Minimalist approach. This might be true, however, the shaky empirical grounding of Grodzinsky’s proposals casts some doubt whether it would be wise
to disregard years of productive research into syntactic theory.

IV. Empirical problems

The proposals in Grodzinsky (1990) do not make many predictions that could be clearly verified. The major prediction: passive sentences have chance probability of comprehension and active ones an above chance probability comprehension is also the major criterion of selection of patients (Berndt & Caramazza 1999). Thus the prediction bears out automatically. All other predictions require an interpretation of the proposed theory. Berndt & Caramazza (1999) and Berndt et. al (1996), have carried out a detailed survey of the patients classified as agrammatics in the literature and compared to those considered by Grodzinsky and his collaborators. They show that there is a strong selective bias towards only considering patients that exhibit passive/active contrasts in comprehension. This makes impossible to consider this contrast as a prediction of the theory. One might say that this is not a problem, those patients that do not exhibit the passive/active contrast have another deficit. One can argue what to call agrammatism and what not. Crucially, there is a theory that describes a deficit involving passive/active contrasts in comprehension.

In a sense this is what is argued in Zurif and Piñango (1999), patient selection is important, however, we are proposing an account of a deficit which has a correlation with Broca’s region and not a loose set of properties that seem to intuitively select patients as agrammatics. This seems at first hand to be refutation of Berndt & Caramazza’s (1999) criticism. However, there is one snag. As I have already mentioned there are Wernicke’s aphasics who also have a comprehension deficit identical to that in Broca’s agrammatics. Zurif and Piñango argue that this is irrelevant since studies by Zurif et. al. (1993) claim to have shown that Wernicke’s aphasics processing of long distance gap-filler relations is different from that of Broca’s aphasics. They utilise a paradigm of lexical decision and tested words that were related or unrelated semantically to the filler of the object gap. There were two positions tested: at the location of the gap and at a pre-gap position. Their findings showed that Broca’s aphasics’ responses to semantically related or unrelated words did not differ both at the gap and pre-gap positions. In contrast, Wernicke’s aphasics were observed to be faster with semantically related than unrelated ones in the gap position, but not in the pre-gap one.

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12 This is in fact a result of a more general prediction: non-canonical word orders (subject precedes object) in English will be comprehended at chance level.
These findings are the basis of the argument that Wernicke’s aphasics’ cannot share with Broca’s aphasics a common underlying cause of deficits in comprehension since, unlike Broca’s aphasics, Wernicke’s can compute gap-filler relations in real-time.

There is one conceptual problem, namely Zurif and Piñango (1999) now assume an interpretation that the model of the deficit proposed in Grodzinsky (1995) is a model of a real time processor. As I have pointed out, GB is not a theory of processing and cannot be simply named as one since it does not address any of the issues that are relevant for processing. In fact GB does not even assume that there is a real-time algorithm assigning gap-filler relations. Many linguistics assume that surface structure is a static representation of native speaker intuitions, and even those who advocate movement as an operation and not a relation (so called derivational syntacticians) do not claim that it happens in real time.

However, for the sake of argument, let me assume that this issue can be somehow resolved. There are still some empirical questions that question this line of approach. Mauner (1995) notes that peculiarities in the way data was normalised in Swinney et. al. (1996). Responses longer than 2750ms were excluded. The problem is that Broca’s aphasics are slower in lexical decision tasks (Bradley et. al 1980) thus there is a bias in the cut off of patients. A procedure to analyse outlier responses ($t > 2750$ms) was used where responses exceeding $\pm 2$SD of the mean were removed. Crucially, however, it was not reported whether the cutoff was calculated for each of the subject’s responses independently or was it obtained from a collapsed set responses for each group of subjects (slow and fast). The first possibility would not be biased against slower Broca’s aphasics, but the second approach would discriminate Broca’s aphasics since it would disproportionally exclude the slowest responses from the slowest group.

Mauner also points to other statistical problems with the study in Swinney et. al. (1996). More importantly, however, the paradigm itself is questionable. Mckoon et. al. (1994a, b) has reported obtaining similar results in lexical decision tasks with sentences that do not have any gaps and thus there are no gap-filler relations that are being tapped. This would indicate that whatever this paradigm is measuring, it does not have to be syntactic in nature. If so, the whole argument for distinguishing Broca’s and Wernicke’s aphasics’ long distance gap-filler assignment abilities in real time collapses.

Thus we are back to the initial problem, whose deficits are we to account for? Should we just arbitrarily select the group of patients that fit, or apply some independent procedure. Obviously, the latter is the desirable solution.
However, let me again put aside these problems and explore some more fundamental problems involving the way patient data is statistically processed in collapsed patient studies that are supposed to support the theories in Grodzinsky (1995)

IV.2 A ideal native speaker

In linguistic theory there is a tacit assumption that what we are describing in linguistic models is an ideal native speaker of a given language. Thus we assume that idiosyncratic differences will be filtered out with the right amount of sampling. This is possible since we assume that underlyingly there are regularities shared by all native speakers of a given language. However, this reasoning cannot be so easily carried over to patient studies. For one, it is not clear that agrammatics have one common underlying functional architecture. Stroke damage is dictated not by the functional architecture of the brain but by the positioning of blood vessels. Also we know that non-agrammatics also have deficits that involve contrasts in passive/active comprehension. This is not analogous to a case of finding a generalisation that cuts across languages since we are not sure what factors influence each of the deficits. In other words, theories of the language faculty assume that there is a core Universal Grammar which undergoes maturation into given languages. Cross-linguistic generalisation are possible since they are attributed to the effects of UG. There is no ‘UG’ of Aphasia. Generalisation within given disorders are very tricky and across disorders impossible at this state of our knowledge.

Why am I raising this issue? It seems that Grodzinsky’s proposals assume such an ‘ideal native speaker of agrammatism’. Berndt & Caramazza (1999) point out that even if we forget about biassed patient selection practices and just look at the pool of selected patients we find serious problems. In a nutshell, in order to establish chance performance on has to have a sufficient amount of trials <N>. However, it only make sense to have a large <N> for each given patient, crucially we cannot collapse small samples <n> from different patients into one big sample <N> since is nothing else but construction an ‘ideal native speaker of agrammatism’. Apart from the already mentioned conceptual problems that this entails, there is also a good chance that we are collapsing patients that do not have the given contrast between passive/active comprehension, or as shown by Berndt & Caramazza have exactly the opposite that predicted comprehension pattern. Thus we can add the performance of patient who has above chance passive comprehension (not predicted by Grodzinsky’s theory) with a patient who has below chance performance in passive comprehension (not really predicted either) and come out with chance performance which is predicted. The same can happen with the comprehension of active
sentences. Let us take an extreme case and imagine that an aphasic patient is substituted with a non-brain damaged subject, patient X will have highly above chance performance on actives, but when added to a patient who is very bad on comprehending actives, but just above chance, the ‘ideal’ patient will have the predicted robust pattern of above chance comprehension on active sentences. I will not present the relevant calculations here, they can be found in Berndt & Caramazza (1999). Crucially, if we do not have access to a sufficient amount of trials in each individual patient we cannot add their results.

This is a serious problem that does not seem to be prone to any easy solutions. It has also generated a lot of discussion (Grodzinsky 1999). I will not go into the details of this discussion since it also seems to involve more fundamental issues concerning group patient studies, and for reasons of space that is impossible here.

Keeping all these problems in mind, let us move on just to continue the discussion, and attempt to see if the theory of trace deletion can yield some testable predictions. After all, Karl Popper correctly argued that a good theory has to account for the facts but crucially it has to be capable to undergo negative verification. We have argued that trace deletion theory accounts for the facts by definition because of the way patients are selected, let us see if we can find any other prediction.

Let us assume that the proposed consequences of trace deletion are a correct way of interpreting the proposal. Then we can assume that the syntax component is damaged severely enough that anything but constituent structure is unavailable. This entails that subjects exhibiting the active/passive asymmetry should not be able to have any intuitions concerning grammaticality judgements since constituent structure is insufficient to rule out ungrammatical structures. This is a clear prediction, we should not see patients that are above chance on actives and chance on passive and at the same time be able to have correct grammaticality judgements. Note that this crucially, involves judgements concerning any type of construction since è-positioned traces are deleted in all constructions not just in passives and actives.

This prediction seems incorrect. Studies by Linebarger and colleagues have found that subjects who have attested disassociations in comprehending passives and actives are able to discriminate grammatical from ungrammatical sentences that have exactly the same structure that supposedly is hard for them to comprehend. For example, Linebarger (1989,1990,1995) reports that agrammatic individuals can distinguish ill-formed passives from well formed ones (following Mauner et. al. 1993):
This seems to be an interesting correlation with production deficits that agrammatics exhibit. However, I will not explore it here.

(11) *a. John was finally kissed Louise
    b. The boy was followed by the girl
    *c. The boy was followed the girl
    d. The boy was following the girl

This is not to say that agrammatics have perfect grammaticality judgements. Linebarger (1995) shows that among other things, agreement seems to pose problems, thus sentences involving tag questions and reflexives are less likely to have correct judgements.13 Grammatical judgement evidence contradicts the assumption that there is no valid syntactic representation in the comprehension module of agrammatics. However, this assumption, as I have pointed out, is crucial in understanding Grodzinsky’s proposals. In fact it is one of the few predictions that the whole proposal makes.

Additionally, sentence priming paradigms have been used on agrammatic patients. It turns out that comprehension increases with priming (Friederici et. al 1989, Hartsuiker et. al. 1998). This would further corroborate the conclusions in Linebarger (1995) that agrammatics have a valid syntactic representation.

IV.3 Two types of chains - a case study in revising a theory

The above discussion indicates that not only are we not sure what agrammatism is, but also that the ‘linguistic’ family of theories constructed on this ‘shaky ground’ is full of internal inconsistencies, and, more importantly, does not give the right empirical predictions. This last section is a case study of how this ‘house of cards’ is constructed. In that sense this does not add anything to the discussion on the merits of this theory but exposes the flaws in the way it is constructed.

Thus, let me quickly discuss a small modification in the proposals by Grodzinsky concerning the comprehension deficit. Again, this is not really relevant as far as the problems raised with this approach and is more relevant for showing what is wrong with proposing the type of research that underlines the ‘linguistic’ study of comprehension deficits.

13 This seems to be a interesting correlation with production deficits that agrammatics exhibit. However, I will not explore it here.
In Grodzinsky (1990) the assumption was that all chains that have as their ‘tail’ a è-marked trace are damaged.\footnote{For reasons of clarity I do not discuss the other shift between Grodzinsky (1990) and (1995) concerning whether all traces or just the è-marked one are damaged.} However, Hickock and Avrutin (1995) reported a patient that had a dissociation in comprehension of wh-questions of the which-NP type and the who/what type.

Question constructions within the framework of Grodzinsky (1990) should be comprehended above chance if the wh-word is in subject position and at chance level in cases when the wh-word originated from the object position. However, Hickock and Avrutin found that this applies only to questions with which-NP words. Questions that involve who/what wh-words were not subject to the same pattern and no contrast was visible between subject and object positions. Differences between wh-questions involving these types of wh-words have long been known in linguistic literature. The initial fact was discovered in Pesetsky (1982) and later elaborated in Cinque (1990). The notion of referentiality seems to be crucial here. The two types of words participate in two different types of chains thus their different grammatical properties. ‘Which-NP’ words form binding chains because of their referential properties, whereas ‘who/what’ words form government chains. The facts distinguishing the behaviour of the both types of words are well documented cross-linguistically, however, the account of why they differ is far from clear, especially since the distinction between government and binding chains is becoming problematic within recent frameworks.

Nevertheless, Hickock and Avrutin proposed a modification to the theory of agrammatic comprehension deficit by narrowing it down to damaged binding chains with government ones left intact. That is traces in è-positions are not visible or deleted if the are part of a government chain. This is one of the revisions adopted in Grodzinsky (1995). However, this extremely fine distinction between the types of chains participating in the comprehension deficit is based on just one report. Furthermore, when trying to replicate the results in Hickock and Avrutin (1995), Thompson (1999) found only one patient out of the five studied to have this distinction. It has to be noted that all five patients exhibited the relevant active/passive distinction which would imply that these two deficits do not necessarily go together.

Thus it seems the hypothesis has some empirical problems since it predicts that these two deficits should go together.
Also, Caplan (1995) notes that the original study in Hickok and Avrutin was flawed in that it from the onset was concerned with finding data to support a very elaborate hypothesis and in the process it ignored much a simpler account of the facts. An alternate hypothesis proposed by Caplan (1995:329) is that “the visual processing associated with matching the expression which man to an item may be greater than that associated with matching the expression who to an item in a picture.”

This, I think, underscores the need postulated by Caplan (1995) to have proper controls for such experiments.

The above paper was aimed to show that however appealing is the rush to apply elaborate and sophisticated linguistic theories to aphasic performance, one should proceed very carefully.

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