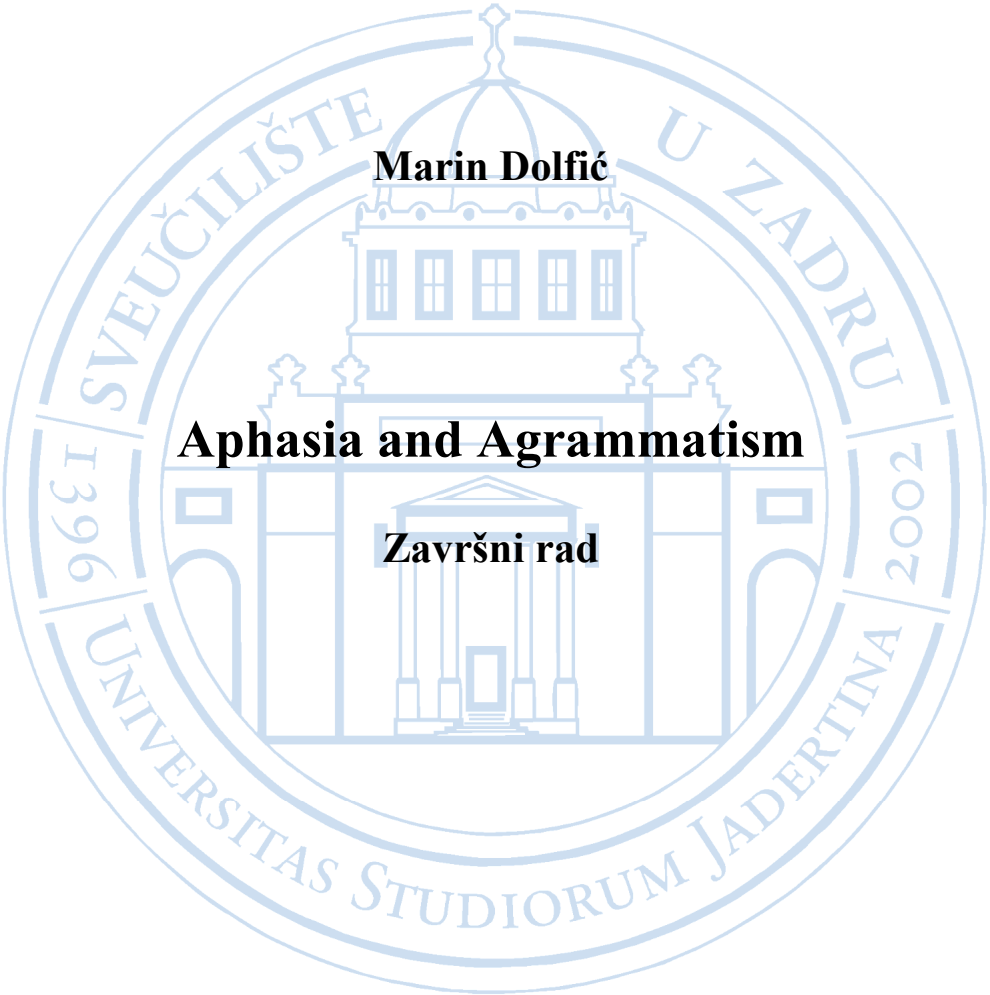
Aphasia and Agrammatism

Dolfić, Marin

Sveučilište u Zadru

Odjel za anglistiku

Preddiplomski sveučilišni studij engleskog jezika i književnosti (dvopredmetni)



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Zadar, 2017.

Izjava o akademskoj čestitosti

Ja, **Marin Dolfić,** ovime izjavljujem da je moj **završni** rad pod naslovom **Aphasia and Agrammatism** rezultat mojega vlastitog rada, da se temelji na mojim istraživanjima te da se oslanja na izvore i radove navedene u bilješkama i popisu literature. Ni jedan dio mojega rada nije napisan na nedopušten način, odnosno nije prepisan iz necitiranih radova i ne krši bilo čija autorska prava.

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Sadržaj mojega rada u potpunosti odgovara sadržaju obranjenoga i nakon obrane uređenoga rada.

Zadar, 25. rujna 2017.

Contents

1. Introduction

The following paper has aphasia as its main subject. Most of the attention will be on Broca's aphasia and agrammatism, while Wernicke's aphasia, paragrammatism, jargonaphasia as a syndrome characteristic of paragrammatism, and other syndromes of aphasia will be just shortly presented.

The word aphasia covers an area so broad that it is really difficult to write a review paper that would discuss, even just superficially, all of the aspects, and the areas that this term encompasses. Besides being such a broad term, aphasia can be studied from a linguistic point of view, and from a medical point of view. This means that one can analyze aphasic disorders, putting emphasis on the location of the lesions in brain, and try to learn what parts of brain are affected in language, in order to understand the human brain better. On the other hand, this also means that one can approach aphasia from a linguistic point of view, putting emphasis on studying the impaired language and its deficits in order to understand how a non-impaired language functions, and subsequently understand human language better. This paper provides medical data in a minimalistic amount and focuses on the linguistic aspects of aphasia.

The goal of this paper is to provide an insight into the syndromes, symptoms, causes and effects of aphasia, and some linguistic theories accounting for it.

In chapter 2, a brief historical overview is given, introducing the most famous aphasiologists and their contribution. This is followed by a description of the connectionist model that explains the cause and effect of the damage location and the type of aphasia in chapter 3. Chapter 4 continues with presenting a brief classification of fluent and non-fluent aphasias, while the next chapter presents findings on agrammatism. Chapters 6 and 7 deal with Wernicke's aphasia and its most severe syndrome: jargonaphasia, followed by a short overview of other syndromes of aphasia and a conclusion.

2. Historical outline

The most widely known aphasia types are definitely Broca's and Wernicke's aphasia. However, these two terms are rather vague, because they do not neatly organize the subtypes of aphasias and their symptoms into easily distinguishable categories. They are rather just a demarcation for the tip of an iceberg that goes very, very deep, and hides a complex network of causes and effects of language disorders (Code, 1989).

Both Broca's and Wernicke's aphasia are named after the two doctors that extensively studied these disorders and the area of brain affected by them, their contribution to aphasiology is so big that not only the two of the most extensively researched aphasias are named after them, but also, the two different areas of brain in charge of different cognitive functions (Eling, 1994; Code, 1989).

2.1. Paul Broca

Paul Broca was a surgeon with particular interest in human brain and its functions. A patient named Leborgne, presented with severe speech disorders; the word *tan* was the only word he could utter; was the patient that brought Broca his fame. It is important to mention that, besides the severe speech disorders, Leborgne was a perfectly healthy man (Eling, 1994). Broca defined aphasia with the following words:

> "The general language faculty remains unaltered, where hearing is intact, where all the muscles, even those of the voice and articulation, obey the voluntary will and where a cerebral lesion abolishes 'articulated language' completely. What those patients lack is solely the faculty to articulate words." (Eling, 1994; pp. 35)

The reason why aphasic patients cannot articulate words, according to Broca, has to do with memorizing the muscle movements required to articulate words. He, furthermore, argues that this motoric part of the speech might not be the sole reason behind aphasia, but just a part

of a larger mechanism that constitutes language production. He, however, did not manage to put a firm grasp on this second part or to describe it. Wernicke did it later (Eling, 1994).

2.2. Carl Wernicke

The knowledge about the brain in time of Wernicke and Broca was still very unclear. There were some fierce arguments about what and how causes speech disorders. Such a commotion around a topic as important as this hindered full progress, and it is namely this situation that Wernicke worked in (Keyser, 1994).

Wernicke, influenced by Meynert, seemed to be right on track with researching this topic. He proposed that the part of the brain in charge of language production could be divided into a sensory and motor part (Keyser, 1994). His most important contribution was the proposition that the motor activity is always accompanied by a sensory stimulation, "therefore, the memory image of motor activity at the same time was to be fixed both in the motor and in the sensory cortex"(Keyser, 1994; pp. 66-65). This means that the sensory and the motor area of the brain are interconnected.

The key to explaining the connection between Wernicke's sensory language center and Broca's motor language center rests in the recognition of sounds as elements of language. The sound arrives at the auditory cortex and is stored into the association cortex right next to it. Through connection between acoustic and motor cortex the speech patterns are stored in the motor cortex, which produces oral language. Since this process starts in the first years of life, the human brain is equipped with a huge storage of memory images by means of which it produces language based on the above described principle (Keyser, 1994).

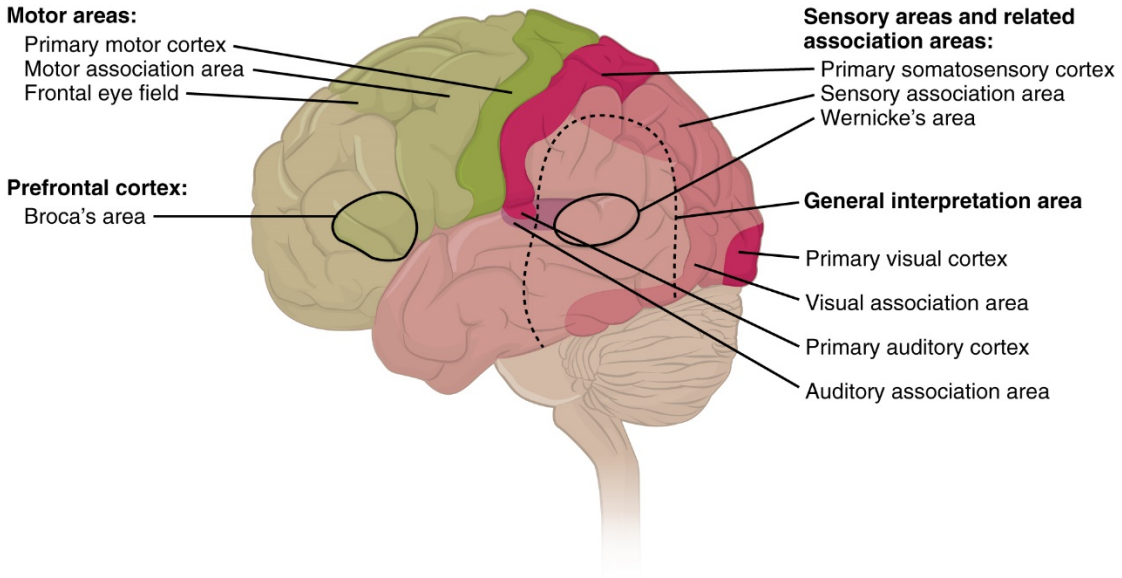

Figure 1. Wernicke's and Broca's area

3. Aphasia Symptoms Models

3.1. Wernicke-Lichtheim's model

Wernicke's elaboration of the connection between the sensory and motor part of the brain led to clearer understanding of causes and effects in aphasiology and set the direction for further researching. The patients Wernicke studied presented with a speech disorder pattern completely different from those in Leborgne; the Broca's patient; unlike him, whose speech production was completely obstructed, Wernicke's patients' speech production was fluent, characterized by sound production errors, which were sometimes so severe that they resembled a jargon (Code, 1989).

The model that Wernicke proposed after studying his patients not only accounted for fluent and non-fluent aphasias, but it also predicted some, then undiscovered, types of

aphasias, and, most importantly, it set a solid foundation for further researchers by serving as a base for much of the neurolinguistics research, even today (Code, 1989).

Wernicke's model, the connectionist model; labeled so because of Wernicke's discovery of the connections between motor and sensory areas of the brain; was further developed by Lichtheim, who applied localizationist tradition on it, and thus created a model which is still most frequently used (Reinvang, 1985; Code, 1989).

The Wernicke-Lichtheim model identifies Broca's and Wernicke's area[1], and strives to provide a detailed description of speech disorder symptoms caused by damage to different areas of the language related part of the brain.

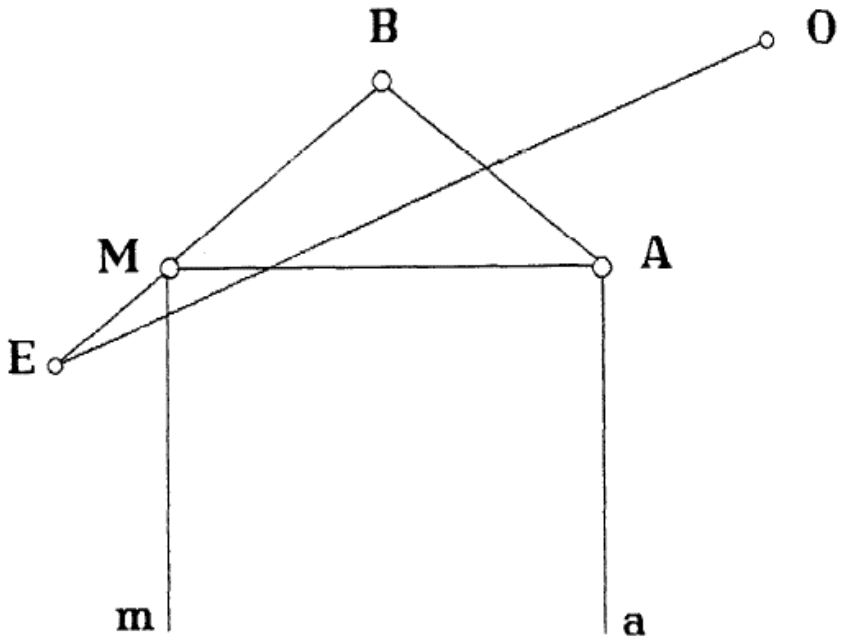

Figure 2. Wernicke-Lichtheim's house (Code, 1989)

[1] See Figure 1. to see where the two areas are located in the brain.

The letters in the Wernicke-Lichtheim's model represent the following: the capital letter *A* represents the center for auditory images; the Wernicke's area; the capital *M* represents the center for motor images, also known as the Broca's area. The capital *B* represents the part of the brain in which every concept is individually analyzed. The capital *O* represents the visual representation center,[2] and the final capital letter *E* represents the motor writing center. The remaining two are marked with lowercase letters *a*, and, *m,* where *a* represents the primary auditory area, while *m* represents peripheral speech organs (Code, 1989). Each of these letters represents crucial components and areas in the production of language, and it is in these areas that lesions form, and, subsequently, cause different aphasias.

With Wernicke-Lichtheim model, explaining the effect of lesions on different areas seems less abstract. Therefore, a direct damage to *M;* the Broca's area; produces Broca's aphasia (Code, 1989). This aphasia is considered a non-fluent one, meaning that a patient with Broca's aphasia will probably present with an impaired speech consisting of poorly intelligible short phrases. Auditory comprehension is here just slightly impaired, preserved in most of the cases. In general, Broca's aphasia understands the loss of volitional speech, writing, repetition of names and words, reading aloud, and writing to dictation (Reinvang, 1985; Code, 1989).

Furthermore, a lesion in the *A* area; the Wernicke's area; produces Wernicke's aphasia. This aphasia is, on the other hand, considered fluent; the speech is characterized by sound production errors, which are sometimes so severe that the speech of these patients resembles a jargon. Grammatical structure remains complex, but the auditory comprehension is obstructed. Wernicke's aphasia, therefore, often understands jargonaphasia, which will be analyzed in more detail later on in the text. It also usually includes problems with repetition, writing to dictation and reading aloud (Reinvang, 1985; Code, 1989). It is important to

[2] Reading is one such activity, processed by this part of the brain (Code, 1989).

mention that all of these symptoms of aphasias are not strictly attached to damage in the Wernicke's area only. This complex network of cause and effect is so delicate and intertwined that it is almost impossible to strictly categorize symptoms, aphasias, and the lesion location (Code, 1989).

Furthermore, an interruption in the pathway between *A* and *M* produces Conduction aphasia, which is, despite the fact that it is, after Wernicke's aphasia, the most common fluent aphasia type, still a rare type of aphasia. Conduction aphasia results in an inability to repeat words or names, reading aloud, and writing to dictation are also impaired, while auditory and written comprehension are mostly preserved (Code, 1989).

The connection between the Broca's area and the organs of speech, that is between *M* and *m,* if damaged results in apraxia of speech. This is a type of aphasia very similar to Broca's aphasia. It includes loss of volitional speech, inability to repeat words or names and reading aloud, while understanding of speech and writing remains intact. The crucial difference between apraxia of speech and Broca's aphasia is the fact that in Broca's aphasia volitional writing and writing to dictation is impaired, while in apraxia it is not (Code, 1989).

The next two types of aphasia are transcortical motor and transcortical sensory aphasia. Transcortical motor aphasia is a result of a damaged pathway between *M* and *B,* the motor center, and the center for elaborating concepts. Here, the repetition of words and names is excellent. However, the comprehension of speech and text is severely impaired.

Transcortical sensory aphasia is similar to Wernicke's aphasia since both include damage to the Wernicke's area, thus it results in paragrammatic speech and damaged auditory comprehension. The main difference between Wernicke's aphasia and transcortical sensory aphasia is the preserved ability of repetition in transcortical sensory aphasia (Reinvang, 1985; Code, 1989).

It is easy to notice that the symptoms overlap more often than not, which supports the theory that cases of pure aphasia are rare; if not non-existent; and that certain symptoms can occur in more than just one type of aphasia.

3.2. Other Models

The other models are mostly based on the Wernicke-Lichtheim's model, and are not as important as it is, therefore, they will be just shortly presented and discussed in this chapter.

The Wernicke-Lichtheim's model analyses aphasias and their symptoms based on the lost and preserved abilities. Norman Geschwind revised this model, and created a new model, referred to as the Wernicke-Geschwind model. This model is very similar to the classical one. It also argues that the most important areas for aphasiology are the motor and sensory parts of the brain. This model introduced some changes that shed more light on aphasia. He discovered a peculiar type of aphasia, which he named anomia (Code, 1989). This type of aphasia includes word-finding difficulty. An anomic patient will only have word-finding problems, unlike in other aphasias where word-finding difficulty is just one of the symptoms (Code, 1989).

The following two new terms introduced by Geschwind are the global aphasia and isolated speech syndrome. Global aphasia is a type of aphasia that affects broth Broca's and Wernicke's area, a severe case of aphasia. Isolated speech syndrome is a combination of transcortical motor and sensory aphasia (Code, 1989).

There were several models and attempts to produce new, different models, like Luria's functional system or Brown's and Jackson's neurological model, however, none of them is of big importance for the topic of this paper, and they will, therefore, not be given any special attention in this paper.

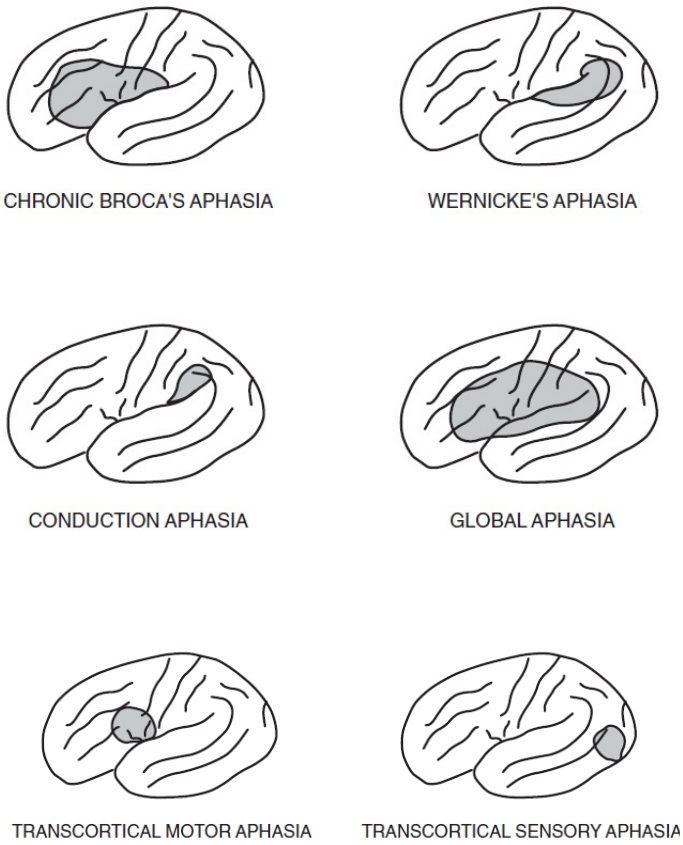

Figure 3. Lesion location for major aphasia types (Saffran, 2000)

Syndrome	Speech	Naming	Sentence Production	Word Comprehension	Sentence Comprehension	Repetition
Global Aphasia	Impaired	Impaired	Absent	Impaired	Impaired	Impaired
Broca's Aphasia	Non-fluent: articulation impaired	Impaired to relatively good; nouns > verbs	Impaired, agrammatic	Impaired to relatively good	Impaired for semantically reversible sentences	Impaired
Conduction Aphasia	Fluent paraphasic; conduite d' approche	Variable but periphrastic	Well structured, periphrastic	Good	Variable; may be impaired for complex sentences	Impaired
Wernicke's Aphasia	Fluent but paraphasic; may be excessive	Impaired	Structured but empty	Impaired	Impaired	Impaired
Transcortical Sensory	Fluent, paraphasic	Impaired	Structured but empty	Impaired	Impaired	Preserved
Transcortical Motor	Preserved but sparse	Variable	Variable	Good	Variable	Preserved
Anomia	Fluent but hesitant due to poor word retrieval	Impaired	Structured but impaired by word finding difficulty	Variable	Relatively good	Preserved

Table 1. Table of aphasia syndromes (Saffran, 2000)

4. Fluent and Non-Fluent Aphasia Distinction

Fluent aphasic speech, compared to non-fluent aphasic speech is at normal rate, without hesitations. However, a specific characteristic of fluent aphasic speech is that it is often meaningless (Edwards, 2005). This is quite obvious in the following passage taken from Saffran (2000), where a patient answers to the question about what he liked to cook. The words in bold italics are non-words.

"I don't know how there is any single way, there's so many thing, you know, that I like. I like meats, I have liked beef, the Germans, you know, and what, well the French you ***koot*** the whole, I can't recall the word that I can't ***thay***. It was the ——— where you make all the food, you make it all up today and keep it till the next day. With the French, you know, uh, what is the name of the word, God, public ***serpinz*** they talk about, uh but I have had that, it was ***ryediss***, just before the storage you know, seven weeks, I had personal friends that, that, I would cook an' ***food*** the food and serve ***fer*** four or six ***mean*** for an evening" (Saffran, 2000; pp. 412).

The types of aphasias that are considered fluent are: Wernicke's aphasia, transcortical sensory aphasia, conduction aphasia, and anomia. Wernicke's, and conduction aphasia are the most common fluent types of aphasias (Edwards, 2005).

When it comes to non-fluent aphasia, the most commonly mentioned, discussed, and researched aphasia is the Broca's aphasia. Here, characteristics of non-fluent aphasic speech are that it is often lacking in some grammatical features like determiners, auxiliary verbs, or verb inflections, and it consists of short utterances (Edwards, 2005). An example of non-fluent speech, where a patient is asked to retell the story of Cinderella, is presented below.

"Long ago Cinderella. One time many years ago two sisters and one stepmother. Cinderella is washing clothes and mop floor. One day big party in the castle. Two girls dresses is beautiful. Cinderella is poor. Two sisters left. In the castle Cinderella is. . .

Godmother. Oh, what's wrong? No money. A little mouse. Cinderella hurry. Queen. Magic wand. Mouses. Oh big men now. Magic wand pumpkin then chariot. Cinderella dresses no good. Cinderella. On my god beautiful now. Next time, twelve o'clock, hex. Then Cinderella party. Many men at the party. Prince is . . . no good. Oh, prince is . . . Cinderella (Saffran, 412).

5. Broca's Aphasia and Agrammatism

The number of researches of Broca's aphasia is by far greater than the number of researches of Wernicke's aphasia (Edwards, 2005). The viewpoints on agrammatism are so abundant, and the theories about its cause and nature so different that it is easy, especially for a reader without any previous knowledge about the important linguistic theories, and aphasic disorders themselves, to get so confused as to not to be able to conceive what agrammatism actually is, nor grasp the relevance of writing about such a vast subject matter (Gjerlow and Obler, 1999).

Below is the so called "cookie theft picture" from Boston Diagnostic Aphasia Examination, the most commonly mentioned picture for aphasic testing. The question that aphasic patients are asked when presented this picture is: "Tell me everything you see going on in this picture."

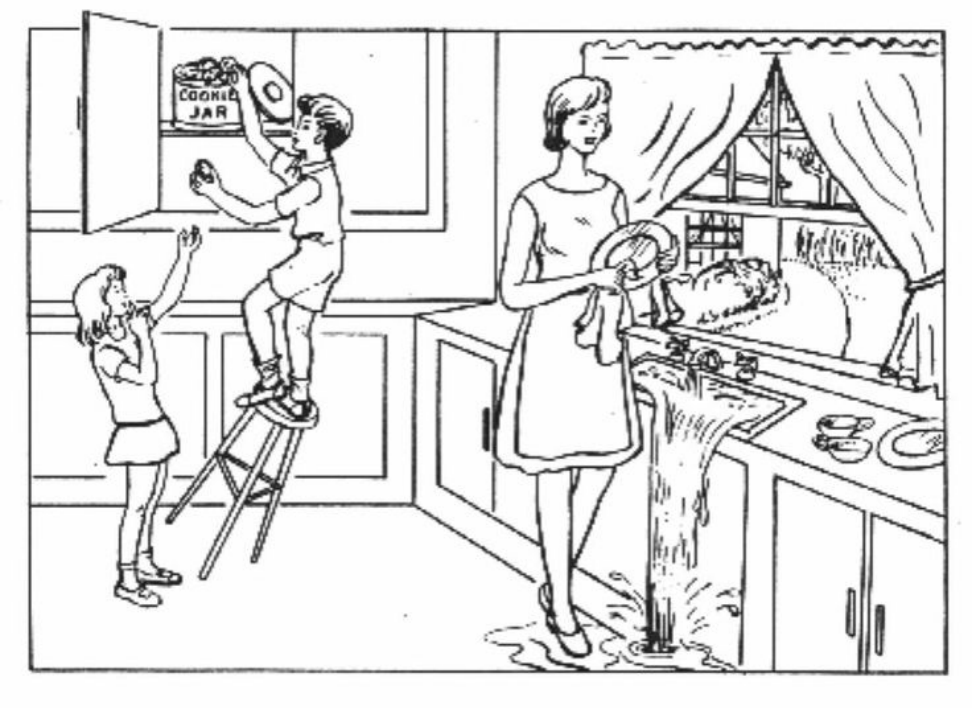

Figure 4. The cookie theft picture

A patient named L.M., who presented with Broca's aphasia, responded with the following description:

"kid…kk…can…candy…cookie…candy…well I don't know but it's writ…easy does it…slam…early…fall…men…many…no…girl… dishes…soap…soap…water…water…falling pah that's all…dish…that's all. cookies… can…candy…cookies cookies…he…down…That's all. Girl… slipping water…water…and it hurts… much to do… Her… clean up…Dishes… up there… I think that's doing it. [The examiner asks: What is she doing with the dishes?] Discharge no… I forgot…dirtying clothes [?] dish [?] water… [The examiner probes: What about it?] slippery water…[?] scolded…slipped" (Gjerlow and Obler , 1999; pp. 41).

Besides the obvious "Why?" there is a number of questions that such a speech pattern poses. Here, there is this obvious pattern where the patient keeps pausing before pronouncing the desired word, as if trying to recall it. The question that one might ask is whether it is really

the case that the brain has forgotten which muscles to involve in order for them to produce the desired sting of words, that is, translating the neural input, as Broca initially proposed (Gjerlow and Obler, 1999).

Furthermore, the word that L.M. produces here is not the target word *cookie,* but *candy*, *cookie* comes afterwards. This could be explained as a word substitution. The fact that he uses *discharge* and *dirtying the laundry* for washing the dishes might imply that L.M. cannot remember the desired words, which subsequently implies a disruption of the lexicon, in that it does not process, or completely lack the information about subcategorization, thus yielding inappropriate verbs and nouns (Gjerlow and Obler , 1999).

The vast number of questions that can be asked about what constitutes the mechanism of this disorder, and the various approaches, led to a number of different researches all striving to provide an accurate theory to account for agrammatism.

The views on agrammatism therefore go from considering it a group of unrelated symptoms, all of which should be studied individually, to attempts at theories that should account for all the symptoms (Gjerlow and Obler, 1999). The question of interest for the linguists that studied this disorder is whether linguistic construct really exists as a part of human psychological reality, and an attempt at getting closer to understanding how language functions inside the brain (Friedmannn, 2006).

Broca's agrammatic patients are usually unable to produce a well-formed sentence. They lose the ability to correctly mark tense on verbs, they fail to use relative sentences, subject pronouns, use subordination, and form wh-questions. On the other hand, subject agreement inflection is mostly intact, object pronouns, and coordination remains (Friedmannn, 2006).

The general view on agrammatism changed rapidly. It was first argued that agrammatic patients lose the syntactic ability completely. Some of the researches attributed these errors to a phonological factor. Others claimed that agrammatism understands a disorder in all the grammatical elements. Luckily, the subject matter of agrammatism has not been abandoned, or considered solved enough to be dropped, therefore, research data accumulated successively (Gjerlow and Obler, 1999; Friedmann, 2006).

What contributed the most is the empirical evidence that agrammatism is of a more delicate nature, and that its volume is not as vast as it had been considered. There is solid proof of the relationship between the hierarchical structures of sentences, and how it reflects on agrammatism (Friedmann, 2006). The following chapter provides an insight into the hierarchical structure of the severity of agrammatism, and the nature of its consequences.

5.1. Speech Production in Agrammatism

Syntactic trees or *phrase markers* are the terms through which speech production will be explained in this chapter. According to Pollock and Chomsky (Pollock, 1989; Chomsky, 1995), sentences are represented through these terms, and they imply that function and content words are represented in different nodes on the *syntactic trees* or *phrase markers*. In the following picture Pollock's syntactic tree is represented (Friedmann, 2006).

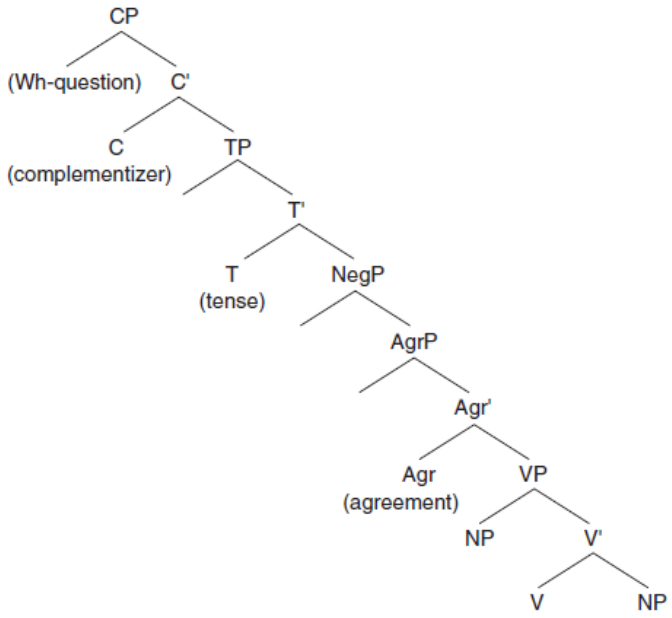

Figure 5. Split inflection syntactic tree (Pollock, 1989)

The markings on top of the phrase marker functional nodes denote the following: AgrP stands for agreement phrase, that is, the agreement between subject and verb in gender, number, and person. TP represents tense phrase, or the tense inflection part of the verb (Friedmann, 2006).

The movement of the verbs, therefore, understands a movement from the lowermost node V, within the VP node, to AgrP, and subsequently, to TP, where it obtains the tense inflection. This structure shows that content and function words are differently represented, through different nodes. The uppermost node CP, complementizer phrase, hosts complementizers. These are the elements that move to CP node, like auxiliary verbs in English yes/no questions, wh-elements, like *where* and *what,* and embedding elements, such as *that* in English language. Therefore, an inability to correctly inflect verbs, or produce, for example, wh-questions, is due to an impairment in one of the nodes, that is, due to "the inability to project syntactic trees up to their highest nodes" (Friedmann, 2006; pp. 65). This

is also known as the Tree Pruning Hypothesis (Friedmann and Grodzinsky, 1997, 2000; Friedmann, 2006). The Tree Pruning Hypothesis accounts for errors in agrammatic production (Edwards, 2005).

The lower down the hierarchical structure the node, the more severe the disorder (Friedmann, 2006). The basic presumption of all the studies of the hierarchical organization of sentences is that if a certain node is damaged, like TP node for example, a patient should not be able to access any of the higher nodes, including the TP node itself. Also, if a CP node is damaged, there should be an obvious pattern of difference between sentences that demand and those that do not demand access to CP node. This is supported by several studies, which will be presented in the continuation of the text (Friedmann, 2006).

Firstly, the studies dealt with the inflection of verbs, and the agreement between the verb and its subject. What they found is that the agreement relationship remains almost intact, or insignificantly small, compared to the error pattern in verb inflection. In a study on English language, conducted by Benedet, Christiansen and Goodglass (1998), there were 15% of correct verb tense inflections, while agreement relationship errors were much less frequent; 42% correctly produced agreement relationship. In other, highly inflectional languages, like Hebrew, Spanish etc. the difference was even greater, like 63.5% correct subject-verb agreement, but only 5.5% of correct verbal tense, for Spanish speaking agrammatics (Friedmann, 2006).

This supports the theory that agrammatism is not as dispersed a syndrome as it was previously thought to be. It provides solid proof that agrammatism is not an example of completely damaged functional categories, nor that it represents a complete loss of syntax, grammatical morphemes, or functional categories, as it was previously argued (Friedmann, 2006).

Furthermore, this encourages the need to find out why there is an obvious pattern of the different severity of impairment to the two different types of verb inflection, which are tense inflection and subject agreement. Tense inflection is usually severely hindered, while agreement is relatively intact (Friedmann, 2006).

Explanation for this interesting, recursive pattern lies in the hierarchical phrase markers, presented in the Figure 6. In the figure, the node for tense is above the node for agreement. TP node is above AgrP node, and this is what allows for such a selective impairment. In this case, TP is situated above AgrP, and subsequently harder to reach. Thus, a patient is trying to produce a coherent sentence, but keeps failing, because the highest they can reach is AgrP, therefore, agreement remains mostly preserved, while tense errors frequently occur (Friedmann, 2006). "Based on these results, Friedmann (1994, 1998, 2000, 2001; Friedmann and Grodzinsky, 1997) suggested the tree pruning hypothesis (TPH), according to which the syntactic tree of agrammatic aphasics is pruned and higher nodes are inaccessible in agrammatism" (Friedmann, 2006; pp. 67). After the TP and AgrP disassociation has been proved correct, the next step was testing whether the same disassociation appears with the CP node; climbing up the tree; with a presumption that if it is impaired the CP node should be inaccessible to agrammatic patients and, subsequently, sentences involving the need to access it should be erroneous.

Unlike some other languages, English demands for CP node to be accessible in order for yes/no questions to be properly formed. Therefore, under the above presumption, the sentence *Do you like cheese?* should, when produced by an agrammatic patient with an impaired CP node, sound something like *You like cheese?*

There were several studies regarding the yes/no question production in English language, all of which yielded similar result: patients with an impaired CP node produced ill-formed yes/no questions. In a study by Goodglass, Gleason, Bernholtz, and Hyde (1972), who

tested for various sentence production, including yes/no questions, the patient tested produced 0 out of 14 yes/no questions correct. Other studies report their patients producing questions without any movement of the verb in wh-questions, without initial do in yes/no questions. For example, in studies by Thompson, Shapiro, and Roberts (1993), Thompson, Shapiro, Tait, Jacobs, and Schneider (1996), Thompson, Shapiro, Ballard, Jacobs, Schneider and, Tait (1997), and Thompson and Shapiro (1995) the focus was on the treatment of wh-questions production, where all the patients exhibited a very poor ability of producing questions. In the study by Thompson and Shapiro (1995) all 17 patients were unable to produce wh-questions before their treatment started (Friedmann, 2006). The study by Thompson et al (1993) reported that patients consistently used intonation alone to indicate questions in spontaneous speech. Furthermore, a study by Friedmann (2002), presented an agrammatic patient with an inability to form both wh-questions and yes no questions, both of which demand access to CP node and the movement of elements. Tests in different languages support the consistency of the theory: if there is an impairment to a node, whenever there is a need to form structures that involve that impaired node agrammatic output is present, while the nodes below the impaired one remain intact, or almost intact (Friedmann, 2006).

All of the above data proves that the verb retrieving deficits that agrammatic aphasiacs present, in fact, stems from a deficient syntax, the inability to envision the hierarchical structure of sentences (Friedmann, 2006). Findings from treatments of agrammatic aphasiacs furthermore support this theory, as the pattern of recovery resembles climbing up a tree, the syntactic tree in this case. In the course of the treatment, and a larger period of time, the patients manage to access nodes higher than those prior inaccessible to them. Therefore, it is safe to say that agrammatism is not a matter of unrelated symptoms, or that all the elements are affected in it. On the contrary, this proves that, despite the opposite beliefs, agrammatic

impairment pattern moves in quite a regular line according to the phrase marker (Friedmann, 2006).

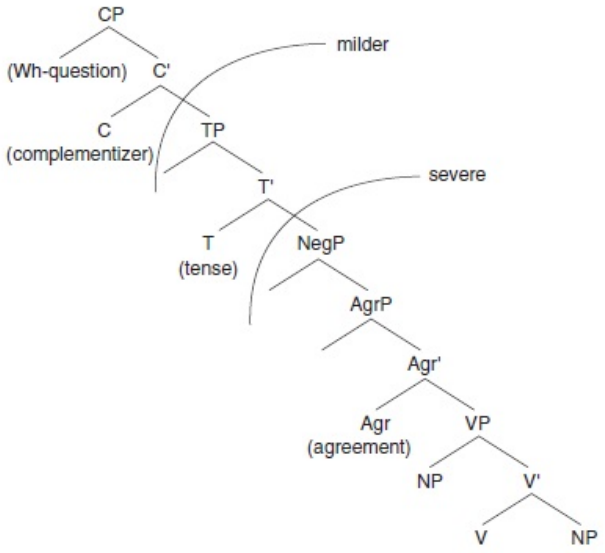

Figure 7. Severe and mild agrammatic impairment

Besides the variation in the ability to access differently impaired nodes, the agrammatic patients all present with the same pattern of non-fluent, agrammatical speech - short phrase length, ungrammatical sentences, and the lack of ability to embed or ask questions.

One segment that is lacking is the matter of open question of whether the AgrP can also be inaccessible. A study conducted on a patient named S.B., whose brain injury was very severe, proves that in the early stages of severe agrammatic aphasia, AgrP is also inaccessible, leaving the speech of the subject patient so erroneous that even the subject-agreement relationship, previously shown to be quite well preserved, is significantly impaired (Friedmann, 2006).

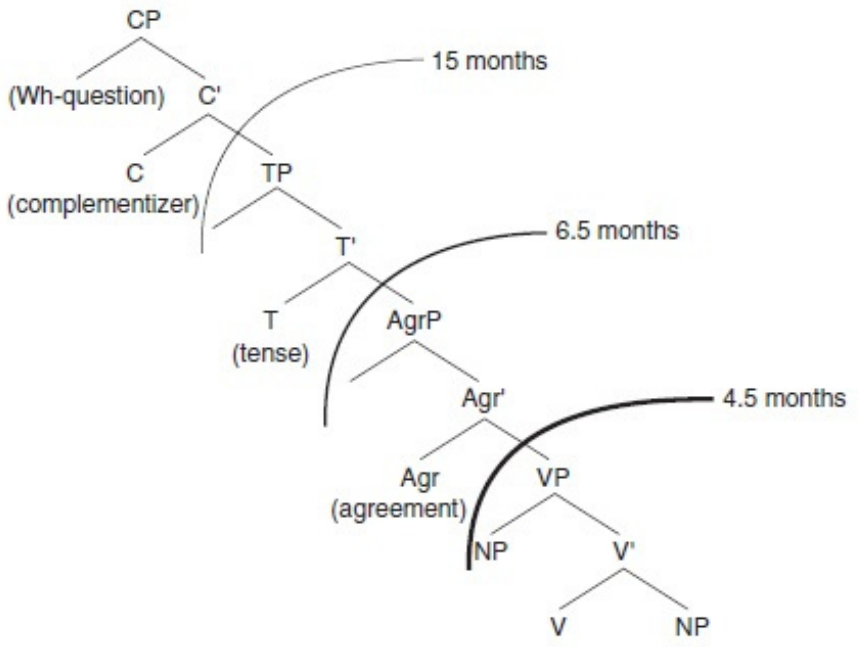

Figure 8. The course of recovery of S.B.

The case of S.B. neatly fits into the pattern. The impaired lowermost node disables access to any higher nodes, and results in an almost completely unintelligible speech. Through a period of time, as the brain healed, and her speech abilities were trained, the access to higher nodes was restored, subsequently, the patient's speech recovered too.

However, the thing with aphasias in general is that there always has to be room for more questions, yielding more interpretations, more studies, and possible results, to either support or disprove the existing theories. Despite being fascinatingly consistent, and accurate, the Tree Pruning Hypothesis is still very limited by its own predictions of impaired agrammatic speech (Friedmann, 2006).

5.2. Agrammatic Comprehension

Further research of agrammatism enters into the sphere of agrammatic comprehension, which was primarily thought to be intact. However, this was proven wrong. This, therefore,

demands an introduction of the second most important linguistic theory that accounts for the errors in agrammatic comprehension, the Trace Deletion Hypothesis (Grodzinsky, 1990, 2000a, 2000b; Balogh and Grodzinsky, 2000; Edwards, 2005). The main goal of these theories and studies conducted was moving away from the volatile nature of agrammatism as an underlying syndrome, and focusing on finding its common features.

Comprehension errors are present mostly with passive sentences and sentences that exhibit a similar type of movement of its constituents. Namely, it is a problem of grasping the movement of constituents that happens when active sentences are made passive; that is the problem of interchangeable theta-roles of NPs (Edwards, 2005).

In sentences like *John slapped Tom,* and *Tom was slapped by John,* for a non-aphasic person, it is easy to differentiate between the doer and the receiver, that is, in this case, between the Agent and Patient roles of the NP which remain the same no matter where they are placed. For an aphasic patient, on the other hand, this interpretation is hindered, because in the last two sentences, both *John* and *Tom* are animate, thus both can take the role of the Agent in the sentence above (Edwards, 2005).

This is not the case with non-animate NPs. The comprehension of the sentence *the soup was eaten by Mary* is preserved in an agramamtic patient, because of the logical understanding of the fact that a soup cannot eat a human being.

This led to a conclusion that it is not the grammatical deficit only that is the cause of this inability to comprehend such a sentence, but also an inability to properly map and coordinate meaning and sentence structure (Edwards, 2005).

The idea behind this is that, when moved, like it happens in passive sentences, NPs leave a trace which connects the old with the new position, and this trace is what is crucial for understanding the thematic role of the NP in question. According to Grodzinsky and his colleagues, (Grodzinsky, 1990, 2000a, 2000 b; Balogh and Grodzinsky, 2000) the agrammatic

patients demonstrate deletion of this trace needed for the interpretation of the thematic role. That is why when both NPs are animate and the NP moves from one place to another, the agrammatic patients demonstrate a random choice making performance on recognizing the thematic roles of NPs, which is mostly based on the knowledge of thematic roles the NPs had before the movement.

The consistency of the theory is further supported by the fact that the problem with passive sentence extends to other sentence structures that behave in a similar way, like object-cleft sentences, object relatives, and certain questions (Edwards, 2005).

With active sentences, comprehension problems do not arise since there is no movement of NP. In the sentence *John slapped Tom,* the first NP takes the role of Agent, while the second one takes the role of Patient, and there are no complications. The comprehension of this sentence remains the same with both aphasic and non-aphasic persons. The problem arises when the same sentence is made passive, as in *Tom was slapped by John.* Here, agrammatic patients demonstrate deletion of the trace which is the previous location of the NP *Tom*. They also try to apply the same logic as in the active form of the same sentence. Subsequently, they end up with having two Agent roles in one sentence, and they randomly decide which one is which (Edwards, 2005).

The case with object-cleft sentences operates on the same principle, despite the fact that the structure of object-cleft sentences is different than that of passive sentences. Here, the NP movement also occurs. The following sentence, *It was the cat the dog was chasing,* demonstrates a movement of object to the position before the Agent, therefore, an agrammatic patient is facing the same problem again, perceiving both of the NPs as Agents (Edwards, 2005).

This is considered to be symptom specific for Broca's aphasia or agrammatism, not occurring in Wernicke's aphasia. However, further studies showed that this is not a rule that

can be generally applied to any agrammatic patient, and that the error pattern in comprehension depends on the severity of comprehension disorders. Nevertheless, the above mentioned pattern does occur in a significant number of cases. The paper will continue with an overview of Wernicke's aphasia.

6. **Wernicke's Aphasia**

The main characteristic of Wernicke's aphasia is fluent speech without meaning, characterized by a large amount of paraphasias, neologisms, and repetition. This type of speech is referred to as paragrammatic speech, or simply paragrammatism. The amount of morphological errors is significantly smaller than in the agrammatic speech pattern, because in paragrammatic speech, the difficulties are of lexical-semantic nature, rather than morphosyntactic, as it is the case in agrammatic speech (Kent, 2004). Patients with Wernicke's aphasia are mostly not aware of their speech errors in the initial phase of the disorder. The awareness develops later, which, subsequently, leads to development of self-correction attempts, most of which are mostly long and unsuccessful (Kent, 2004).

6.1. Characteristics

Even though, from a distance, speech of paragrammatic patients seems normal, its deficits become obvious after just a sentence or two of more careful listening. Below is an example of paragrammatic speech. The patient was presented the cookie theft picture.

"These were [ɛkspɾɛtʃəz], [əgræʃnz] and with the type of mechanic is standic like this … and then the … I don't know what she [gðin] other than [?]. And this is [dɛli] this one is the one and this one nad this one and … I don't know.

I mean, she is a beautiful girl. And this is the same with her. And now it is coming there and [?]. Now what about here or anything like that … what any.

This is a boy, this is a boy. I forget the boy and a boy. This one ever which ever one is right and a boy. Then this one is right here, right here. And… nice night in here.

Well, this is a little girl boys. And that's a little girl, he's a [trə traksər] candy. And, my lights are oh, [kæθəl dunət], [kænə donət]. And he was up on the [raksər], but it's a wonder he wasn't [ɔfə] fell [ɔfə] there.“ (Buckingham, 1981; pp. 54-59)

People who have encountered such patients report their speech to be somewhat hasty, super-fluent. To the listener, it seems that fluent aphasic patients speak faster. This characteristic of fluent speech is called the press of speech (Edwards, 2005).

Different studies, however, showed that the word-count in non-fluent aphasic patients is the same as the one in non-aphasic patients. Speech of non-fluent aphasics, however, gives the impression of being faster. This is mostly due to the fact that the structure of such impaired speech is incorrect, thus, it is hard, almost impossible, to understand it.

In a study by Edwards and Garman (1989), a non-aphasic and an aphasic patient, with a very similar career and educational background performed a verbal task in which they had to speak about their previous work experience. What became evident first is the difference in the flow of conversation. The non-aphasic person conversed without difficulties, allowing for interruptions and questions, showing respect for their interlocutor. The aphasic person showed no such trait. This is mainly because the speech of this aphasic patient is unclear, resembling a jargon-like monologue rather than a conversation. Thus, for the interlocutor there is not much to ask the aphasic patient, other than repeatedly asking for clarification. This impression that the non-fluent aphasics are somehow pressed for speech might therefore stem from this inability to converse in a proper manner (Edwards, 2005).

6.2. Phonological and Lexical Deficits

Fluent aphasic patients exhibit a problem with finding words while maintaining a proper syntactic structure (Davis, 2000). This disassociation approach is the most common in literature about fluent aphasia (Edwards, 2005).

The most common error patterns in fluent aphasia are phonological errors. These errors involve incorrect selection of phonemes in a word, also known as phonemic paraphasias. For example, a patient may produce *kog* instead of *fog*, and the sentence *All he could see was kog*, might be intelligible, since only a minor phonological substitution happened, forming an easily understandable non-word. However, such examples are almost never present in fluent aphasic patients' speech. The severity of disorder is usually greater, and the output is usually so impaired that it is hard to discern whether the paraphasia is a whole-word substitution, or just phonemic substitution. Besides, the case with phonemic substitutions, no matter how simple they can appear, is that they can yield an actual word that was not the desired word. Had the imaginary aphasic patient produced *log* instead of k*og.* It would have been harder to say whether the uttered word was the desired one, or just a phonemic substitution (Edwards, 2005).

Another type of impaired output is when the substituted words seem syntactically well positioned, however, semantically, they are impaired. One such example is the sentence "*I couldn't hear the pain"* (Edwards, 2005). The sentence is correct, however, illogical; pain cannot be heard. Thus, here, the assumption is that the verb pain must have been substituted for feel. Patients with fluent aphasia rush to fill the missing slot in the sentence, subsequently, such substitutions occur. Here the substitution is verb for verb, which implies that the representation of a syntactic structure inside of the head of such patients is intact, while lexicon seems to randomize words based on a pattern of similarity (Edwards, 2005).

Lexical substitutions tend to respect grammatical category. However, when it comes to verbs, the verb argument structure requirements in a sentence are not always preserved (Edwards, 2005).

The best way of explaining the functional pattern of such whole-word impairments is comparing the task solving methods to those of non-aphasic speakers of a foreign language. When conversing in a non-native tongue, a pattern which is very often is that, in lack of an appropriate word, the speaker finds the second best option and uses it (Edwards, 2005).

Fluent speech could be explained as an annoying inability to produce desired words. Judging by the fact that in the starting phases of the disorder speakers are often unaware of their speech impairments, it could be argued that, what is perceived to have been said by the speaker and what the interlocutor hears differ greatly, which would furthermore support the view that concept representation remains intact. The brain of the patients perceives their speech as correct, probably because at the first stages of the disorder there is no awareness of the presence of the disorder and the mind of the aphasic patients tricks them into believing that their speech is normal. Therefore, the initial frustration when the interlocutor is unable to understand the message. When and if the awareness of the errors appears, the need to correct it appears which leads to an even more unintelligible output. They often cannot control the need to correct themselves so they might continue producing strings of words and non-words in order to eventually get to the desired word, which rarely happens.

7. Jargonaphasia

Jargonaphasia was first defined as “a series of speech sounds without meaning“(Brown, 1981). This definition makes it similar to that of Wernicke's aphasia. This is roughly what jargonaphasia is, a severe syndrome of Wernicke’s aphasia.

In jargonaphasia, the term neologism is not used in its denotative sense, which requires a neologism to be an intelligible, acceptable new word or a new meaning of an already existing word. "The word neologisms designates deviant segments that are uttered as if they were single words or locutions although they do not occur in the dictionary, and that can neither be positively identified as phonemic paraphasias, because the listener cannot recognize target words, if any, nor as morphemic deviations, because they are not made of bona fide[3] morphemes"(Lecours et al., 1981).

The human mind and language operate in a hierarchical, compositional way, that is to say, the words are not stored in the brain like they are in the dictionary. They are, however, retrieved in forms of roots and stems to which affixes are attached (Buckingham, 1981). This means that a non-aphasic person possesses a creative ability to coin new words, and employ wordplay. Patients with jargonaphasia maintain this ability, since their brain still somehow tries to employ this approach to forming words, however, the outcome of it is altered (Buckingham, 1981).

According to Buckingham, inflectional morphemes are the most affected ones. The neologisms are inserted into well-formed grammatical matrices abiding morphemic and morphosyntactic rules (Buckingham, 1981).

From this it follows that the neologism is formed first, than this neologism is assigned affixes and, subsequently, inserted into a sentence (Buckingham, 1981).

Determining the categories of the neologisms is not always possible, however, since syntactic constructions remain sufficiently correct, categories can be assigned (Christman and Buckingham, 1989).

[3] Real or genuine

Miller and Ellis (1987), tried to explain the occurrence of neologisms by frequency of occurrence. The higher the frequency of occurrence of a word, the lower the rate of neologisms. Therefore, function words, which occur the most in speech, should, according to Miller and Ellis, be the least susceptible to neologisms (Christman and Buckingham, 1989).

Phonologically, neologisms can resemble a word in the patient's target language; they can remain partially recognizable because of the preserved parts of the original word; or they can be completely unintelligible. This, highest level of distortion is labeled as an abstruse neologism, where no connection to the patient's native language can be found at all (Christman and Buckingham, 1989). The anomic element affects the neologisms in a way that it might be the stem of the problem, because the word the patient has in mind is not the desired one, but the one anomically encouraged into a process of neologization. The word a patient has in mind is an object for which they cannot find a name, then, in order to articulate it somehow the patient forms an unintelligible neologism (Christman and Buckingham, 1989).

Jargonaphasic patients have problems with the phonological representation of words, while the meaning of these words remains intact. Syntax seems to remain well preserved, just like the relationship of language and real world, and relations inside the language, as well. Their sentences, compound or coordinate, may be correct, while their syntactic morphology, although it may present with an impaired predication semantic context relationship or incorrect affixation, still remains quite coherent (Christman and Buckingham, 1989).

8. Other Syndromes of Aphasia

8.1. Anomia

Anomia, literally meaning without words, is largely similar to the feeling of words being constantly on the tip of one's tongue. This is the feeling that anomic patients have, just

that they cannot get the word off their tongue. This subtype of aphasia is important because it is present in all aphasic syndromes (Goldfarb and Harpen, 1989).

Patients can have different issues, like, for example, an inability to identify and name the object when confronted with it or with naming in spontaneous speech, where the problem arises during the speech itself. Fascinating enough is the fact that it happens that patients that present with issues in naming object at confrontation have fluent spontaneous speech. Syntax and fluency are preserved in anomic patients. Patients are prone to hesitation, giving periphrastic definition of the presented object, or the object they are talking about (Goldfarb and Harpen, 1989).

The following is how one patient tried to describe a ruler when he was presented one. "According to what size you get – a long one, and you get narrow ones. You measure the inches and so forth" (Goldfarb and Harpen, 1989; pp. 35).

Anomia is considered a fluent type of aphasia, however, localizing it is not possible since it can appear as a result of damage to any part of the brain in charge of linguistic tasks. This happens because of the fact that the patient, despite not being able to produce the desired word, can perceive it clearly enough to assign it a similar specter of meaning, therefore, they use circumlocutions in order to name it, or describe it (Goldfarb and Harpen, 1989).

8.2. Conduction Aphasia

After Wernicke's aphasia and paragrammatism, this is the most frequently researched fluent aphasia syndrome. Originally, it is thought to be a result of damage to the structure connecting Broca's and Wernicke's area, thus resulting in a disassociation between the two areas. The main characteristic "is the inability to repeat spoken language." (Gjerlow and Obler, 1999; pp. 43) Since Broca's area, in conduction aphasia, remains relatively spared, this accounts for the good spontaneous speech. The relatively intact Wernicke's area is the reason

why comprehension remains good. However, repetition demands a quick exchange of data between the two areas, thus, damage to the area connecting the two results in conduction aphasia. Repetition is not always the only thing that is impaired in these patients, however, it is the most prominent feature of disorder (Gjerlow and Obler, 1999).

More recent research on conduction aphasia yielded better, more accurate results. Besides the impaired repetition, patients also present with phonemic paraphasias, that is, the substitution of morphemes in target word (Gjerlow and Obler, 199). An example of this is found in Goodglass' patient where his patient constantly fails to name a whistle, and produces the following: "tris … chi … twissle" (Goodglass, 1993; pp. 142). The patient keeps failing to properly assemble the word, despite the fact that it seems that the patient knows which word they are supposed to produce. The key to such a scrambled word output lies in a failed attempt at coordinating the desired word with muscles needed to produce it. Conduction aphasiacs have problems with positioning phonemes inside a phonemic string (Gjerlow and Obler, 1999).

9. Conclusion

No doubt, aphasia is a difficult and demanding area of research. The vastness of the field further encumbers those that intend on studying this phenomenon. However, despite the difficulties, studying aphasia is as rewarding and as interesting as it is demanding, if not even more. It provides a working ground on understanding language, and this is what makes it so significant and interesting, at least from a linguist's point of view.

It usually happens that people tend to notice things to which they were oblivious before only when they start malfunctioning, or disappear completely. Similar to this peculiar human custom, aphasia provides those that decide to delve into its depths with an open window into how the disorders on different linguistic levels of comprehension and production, help explain the functioning of a non-impaired language system. It helps understand how language is structured inside of the brain.

As this paper presented, the theories and approaches to analyzing aphasia are numerous, however, the errors produced by aphasic patients are somewhat less volatile than the approaches to their interpretation. Through history, many researchers dealt with this topic, taking Wernicke's and Broca's findings as their guidelines or parting from their findings in order to prove the opposite or provide a groundbreaking shift in directing the further researchers.

Conclusion that can be drawn from this paper, and studying different theories, and case study reports on aphasic patients is that there is enough evidence that language is structural and componential. That is that it functions like a puzzle that our brain starts solving as early as in the first stages of life. In the course of time, the brain accumulates quite an imposing, however, delicate mechanism for the production of language. This mechanism works automatically and empirically. It takes pieces from different parts of storage available

in order to form meaningful strings through which human beings express themselves. The puzzle is made of syntax, morphology, phonology, semantics etc. and each of this parts consists of its own parts. Thus, this delicate mechanism operates, most often, subconsciously, because seldom is any special attention given to it, unless the person using the mechanism is a linguist, a curious individual, or the mechanism itself starts malfunctioning.

Finally, it is important to emphasize one more time that applying any theory to researching a normal-functioning language through an impaired one, can be equally productive and counterproductive, because it can be limiting. Since aphasia syndromes are very volatile, in that they often overlap, making it hard to draw strict lines between the types, the approaches to studying should adapt to its nature. Preferring one theory or approach over another, if it yields better results, is logical and reasonable, however, stubbornly complying to the conditioning of a theory or approach can hinder the progress in discovering more about language disorders, and language itself.

Therefore, there must always be room for innovation, be it the application of an altered, reworked approach to studying aphasia, or providing a completely new, groundbreaking approach to studying it. While studying aphasia, both thinking inside of the frameworks, and outside of the frameworks, should be employed.

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11. Abstract

The main goal of this paper is to give an insight into the wide term of aphasia. The paper provides an overview of symptoms and syndromes of aphasia, along with providing the location of the damage in the brain that causes the specific case of aphasia. Paper starts with a brief introduction to one of the main contributors, who highlighted the importance of studying aphasia; Carl Wernicke and Paul Broca. Then the focus shifts onto the two main subtypes of aphasia, Broca's and Wernicke's aphasia, their most common syndromes: agrammatism and paragrammatism and their linguistic characteristics along with presenting some most accepted linguistic theories that try to provide a reasonable, consistent explanation of the cause and effect of these syndromes. Furthermore, some of the less researched, however still important aphasia syndromes are shortly presented. The volume of this paper is too small for the vastness of the subject matter, therefore, only the most prominent, basic elements are presented and analyzed.

Key words: agrammatism, paragrammatism, jargonaphasia, aphasia, fluent aphasia, non-fluent aphasia, Wernicke, Broca,

12. Sažetak

Afazija i agramatizam

Cilj ovog rada je pružiti uvid u ono što spada pod široki pojam afazije. U radu je predstavljen pregled simptoma i sindroma afazije, te povezanost lokacije oštećenja mozga i afazije koju isto uzrokuje. Rad započinje kratkim upoznavanjem s dvama najpoznatijim doktorima koji su uvelike doprinjeli isticanju važnosti proučavanja afazije: Paul Broca i Karl Wernicke. Zatim se pažnja usmjerava na dvije glavne vrste afazije: afazija Broca i afazija Wernicke, njihove najčešće sindrome: agramatizam i paragramatizam i njihova lingvistička obilježja. Također, predstavljene su i neke od najpriznatijih ligvističkih teorija koje su pokušale pružiti jasno, konzistentno i razumno objašnjenje uzroka i posljedica ovih sindroma. Nadalje, ukratko su prikazani neki od sindroma afazija koji su, unatoč tome što su istraživanji manje nego prethodnici, idalje vrlo važni. Opseg ovog rada premalen je za razmjer teme, stoga su samo najvažniji, najstaknutiji djelovi predstavljeni i analizirani.

Ključne riječi: agramatizam, paragramatizam, žargonafazija, afazija, afazija Broca, afazija Wernicke