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Lexical Access in Broca's and Wernicke's Aphasia: A Literature Review

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Abstract

Aphasia is a language disorder that results from brain injury, impairing the speaker's ability to produce and/or comprehend language (Arantzeta, 2021). The study of this specific language deficit is called aphasiology. Research in the area of aphasiology has been a matter of study for several years. Although various concerns about aphasia could be analysed and explained, as aphasic people can present impairments in different levels of the language such as phonology, lexicon, semantics, morphology, syntax, pragmatics and suprasegmental features (Arantzeta, 2021), it is the aim of this paper to analyse how aphasic people specifically access their lexicon. Throughout this paper, research concerning lexical access in Broca's and Wernicke's aphasia will be described, and the development and latest findings during the last years will be shown. With such an aim, classical and contemporary publications will be addressed first, and then what new neuroimaging techniques have contributed to the field will be detailed. In short, this paper analyses how advances in new technologies and the standardization of specific tests and tasks have resulted in the discovery of significant and relevant data within the field of Broca's and Wernicke's lexical access. The more classical studies reveal that semantic and phonological factors as well as automatic and controlled processing are worth considering in the lexical access of Broca's and Wernicke's aphasic people, based on their atypical results compared to healthy subjects (Baker et al., 1981; Milberg et al., 1987, 1988; Hagoort, 1993). Provided the use of advanced brain imaging techniques and other behavioral methodologies such as the Eye-Tracker, contemporary research has shown that both anterior and posterior brain areas are responsible for lexical access and that lexical competition could be the reason under Broca's and Wernicke's impairments in lexical access (DeLeon et al., 2007; Yee et al., 2008; Johnson et al., 2019). In fact, current research has shown that Wernicke's aphasic people have increased lexical activation, whereas Broca's aphasic people have reduced lexical activation (Yee et al., 2008).

Keywords: Aphasia, Lexical Access, Broca's Aphasia, Wernicke's Aphasia, semantics, phonology

1. Introduction

Language is a fundamental quality of being human. Not only do we use it to communicate and build relationships between one another every day, but we also employ it to create art such as literature or even human-machine interaction. The scientific study of language, linguistics, has been concerned with different issues of human language, and through conducting different studies and developing different theories, has attempted to understand the units, structure and origin of language (Diéguez-Vide & Peña-Casanova, 2012). Some of those theories have become prominent inside the field, such as the Universal Grammar (UG) theory proposed in the 1960s by the linguist Noam Chomsky. According to this theory, language is an innate capacity of humans and its components are genetically encoded in our DNA (Chomsky, 1986). Despite the disagreement that this theory may hold and the different perspectives that other researchers propose, it could be said that the existence of theories such as UG and the fact that language is a fundamental system of communication demonstrate the importance that language has in science and current research. It is not surprising, as a consequence, that a statement such as language is what makes us human has become slightly like a proverb in our society.

Within the science of linguistics, different branches such as psycholinguistics and neurolinguistics exist. These are two areas of research that share a close relationship, as they both aim to analyse language in our brain. In order to be more specific, psycholinguistics aims to comprehend the cognitive processes that govern the production and understanding of human language, and neurolinguistics tries to identify the brain structures and neural networks that underlie those various cognitive activities (Diéguez-Vide & Peña-Casanova, 2012). One might argue that these two disciplines are actually complementary, as trying to deconstruct the structure of the neural network of cognitive activities necessarily means the need for understanding the cognitive activities first. In fact, in order to understand how language works, a firm comprehension of the distinct linguistic modules (phonology, morphology, syntax…) is also essential. For the research of language in the brain, therefore, knowledge of linguistics, psycholinguistics and neurolinguistics is completely necessary (Diéguez-Vide & Peña-Casanova, 2012).

The studies that have been more commonly conducted in order to understand human language characteristics are those that involve brain damage (Ahlsén, 2006).

Those studies can be included inside the branch named aphasiology i.e., the study of aphasia. Aphasiology is responsible for characterising the altered language production and comprehension of a patient as a result of a brain injury, and tries to offer adequate rehabilitation for this impairment (Diéguez-Vide & Peña-Casanova, 2012). It is from the studies of these injured patients that researchers obtain some of the most relevant information about human language. In this way, in aphasiology, one of the most studied types of language impairment has been that one related to lexical access. Moreover, as stated by Arantzeta (2021), almost all the scientific knowledge about aphasia has been based on monolingual Anglophone speakers.

Taking into account the aforementioned literature, the present paper will give an account of the lexical access impairments of monolingual English speakers with aphasia. In fact, the aim of this paper is to describe the different neural characteristics and difficulties that these speakers present when attempting to access their lexicon. A general description of aphasia (see section 1.1), some basic notions about the clinical characteristics of the different types of aphasia (see section 1.2) and the access to the lexicon in healthy people who do not present any type of neurological damage (see section 1.3) will be explained beforehand.

1.1. Description and characteristics of aphasia

Aphasia is defined as a language disorder that is presented as a result of a neurological damage in which the production and/or comprehension of the speaker is impaired (Arantzeta, 2021). The main source of aphasia is a stroke or a cerebrovascular disease. In today's society 21-38% of people who have suffered a stroke present aphasia; being elderly people (Ellis & Urban, 2016; Engelter et al., 2006, as cited in Arantzeta, 2021) and women (Wallentin, 2018, as cited in Arantzeta, 2021) the most likely to suffer from it. The neurological injury usually occurs in the left hemisphere, and the specific location determines the type of aphasia and its particular symptoms (Henseler et al., 2014, as cited in Arantzeta, 2021). The severity of the injury is also determinant, as a patient can show different types of impairment that will dictate their diagnosis. A person with aphasia can show an impairment in all the levels of the language (phonology, lexicon, semantics, morphology, syntax, pragmatics and suprasegmental features) or they can only have one or some levels impaired. In any case, three different criteria are usually considered when describing and classifying aphasic syndromes: fluency, comprehension, and ability of repetition (Arantzeta, 2021).

As far as fluency is concerned, it encompasses the fundamental characteristics of verbal expression in relation to the difficulty of its articulation, the prosody, the length of the sentence and the lexical volume. An aphasic patient can present a fluent or non-fluent use of the language. A non-fluent person, on the one hand, presents a reduction in their capacity of expression with difficulty at starting sentences, complete or partial loss of the ability to articulate words and a decrease in the length of the sentence. A fluent patient, on the other hand, presents a typical eloquence or even an augmented one. Anatomically, the fluent/non-fluent dichotomy is reflected in the differentiation of anterior or frontal lesions from posterior or temporal-parietal lesions. Aphasia caused by anterior lesions of the language zones in the brain presents a non-fluent language, whereas aphasia caused by lesions of the posterior zones presents a fluent language. Considering the other two criteria, i.e., comprehension and ability of repetition, the former aims to distinguish a preserved comprehension from an altered one, and the latter aims to differentiate patients with a typical repetition ability from patients with an altered ability (Diéguez-Vide & Peña-Casanova, 2012). Taking these three criteria into account, several types of aphasia can be distinguished (see section 1.2).

1.2. Classification of aphasia

In 1861, Paul Broca examined a patient who, due to a brain condition, was only able to utter a particular swear word and the syllable “tan”. He was unable to speak. Broca found that this patient presented extensive damage to the frontal lobe on the left side of his brain, so he became intrigued by the idea that language could be localised in that area of the brain. The patient that Broca analysed had lost the ability to produce spoken language, but other aspects of language such as comprehension seemed to be perfectly preserved. For this reason, he examined other patients that presented the same loss of production and discovered that they all displayed damage to the same area of the cerebral cortex (Sedivy, 2014). This area of the brain was named Broca's area (Figure 1) and was associated with language production.

After these first examinations and findings, Carl Wernicke in 1874 studied a patient that presented just the opposite symptoms of Broca’s patients. This was a person who, due to a stroke, was able to speak fluently but did not understand anything that was said to him. In this case, this patient's lesion was also on the left side of the cortex, but it was in the temporal lobe rather than in the frontal lobe; farther back than the region Broca had already described (Sedivy, 2014). As a consequence, this new region in the brain was named Wernicke's area (Figure 1) and was associated with language comprehension. This pioneering work by Broca and Wernicke confirmed the idea that different types of damage in specific areas of the brain imply different kinds of language impairment.

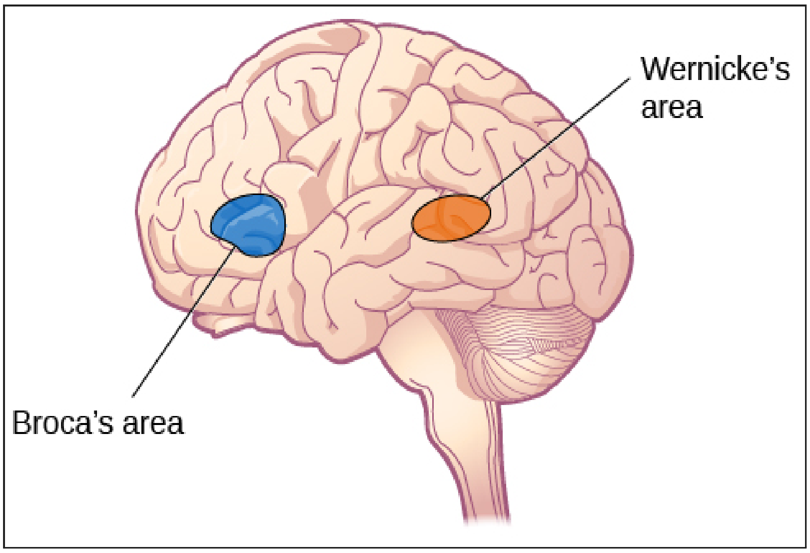

Figure 1. Left hemisphere of a human brain, with Broca's and Wernicke's areas shown in colour (taken from Guy-Evans, 2021).

Broca's and Wernicke's aphasia, nevertheless, are not uniquely associated with the areas of the brain that carry the same name. When diagnosing Broca's or Wernicke's aphasia, in many cases there is a lesion in Broca's or Wernicke's area plus lesions in the related white matter and in adjacent areas. Injuries only in Broca's area, for example, produce mild transitory aphasia and can be recovered with proper treatment (Diéguez-Vide & Peña-Casanova, 2012).

Broca's aphasia, therefore, is characterised by patients who present a non-fluent speech with a limited number of words, and a deficit in repetition. In addition, production requires great effort and is accompanied by poor articulation.

Comprehension, however, is typical or relatively typical.[1] Wernicke's aphasia, on the other hand, is identified for the important impairment on comprehension, even in simple tasks. Nevertheless, oral production also presents numerous alterations. Patients are characterised by a fluent expression without much articulatory effort, with typical prosody and often excessive verbal production. Repetition is also always altered.

Apart from these two types of aphasia, other types have been detected and explained in recent years (Diéguez-Vide & Peña-Casanova, 2012). These are: transcortical motor aphasia (similar to Broca's, but with the ability to repetition preserved), transcortical sensory aphasia (comprehension impaired but repetition preserved), mixed transcortical aphasia (only repetition preserved), conduction aphasia (inability to repeat whole sentences or words), global aphasia (the most severe aphasia where all aspects of language can be impaired), and anomic aphasia (severe lack of vocabulary) (Diéguez-Vide & Peña-Casanova, 2012) (see Figure 2).

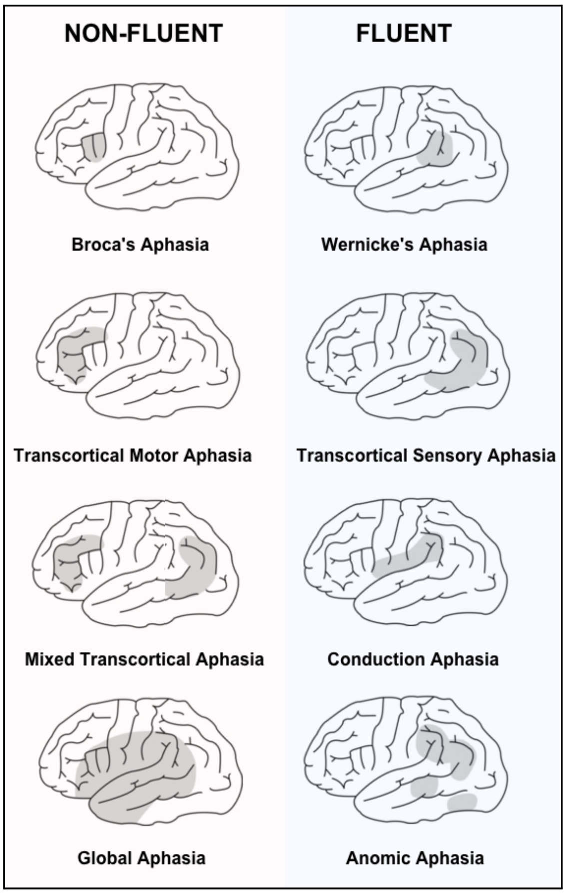

Figure 2. Different types of aphasia and the different brain regions that are injured in each of them (taken from PsychBD, 2021).

[1] The term "typical" will be employed when no neurological impairment is present.

It should be noted that there are other types of aphasia such as subcortical aphasia, aphasia in children and aphasia in bilingual and polyglot people but these types of aphasia go beyond the scope of this paper and will not be considered.

In order to attain a firm grasp of the notions on aphasia that will be described throughout this paper, a prior understanding of how the mental lexicon works in healthy people is necessary (see section 1.3).[2]

1.3. The mental lexicon in healthy people

Different studies have shown that the mental lexicon consists of semantic, phonological, orthographic, morphological, and other types of information (Diéguez-Vide & Peña-Casanova, 2012). According to the theory presented by Collins and Loftus (1975), concepts within the brain's semantic memory are represented as nodes connected in a network; following the principle of semantic proximity. That is to say, concepts that are semantically related are closer together in our semantic memory and share stronger ties. When accessing our lexicon, first of all, a concept is activated in our mind, which is known as the lemma access. This activation is extended to all other concepts connected to that concept in the network, so the approximate nodes are more active than the nodes that are farthest away. As an example, the concept of "dog" will provide to the concept of "cat" a higher activation than to the concept of "house", since the semantic connection between the first two is stronger than with the latter. However, as mentioned at the beginning of this section, the semantic network is also phonologically conditioned. This would be the second step into lexical access, known as phonological access. Here, the concept activated in the lemma access will have specific sounds that are attached to it. In this way, the concept "cat" will be linked to the concept "dog" because of its semantic proximity, but also to the concept "pat" because of its phonological proximity.

Collins and Loftus' theory (1975) illustrates the complexity of the lexical organisation and its relations, and induces to question the extent to which this lexical arrangement is challenged by people with aphasia (PWA). In fact, as reported by Diéguez-Vide and Peña-Casanova (2012) "in all aphasic cases exists a problem in denomination" (p. 88), but not all PWA present that impairment in the same way and

[2] The term "healthy people" or "healthy subjects" will be used to refer to people with no brain damage.

with the same frequency. For this reason, it is the aim of this paper to give an account of the different studies that have been conducted in order to understand the way in which monolingual PWA get access to the lexicon. To this end, section 2 will focus on classical publications and the data gathered in those experiments. Then, section 3 will describe the data obtained in more recent publications. Principally, the paper will address the different types of semantic and phonological factors that affect monolingual PWA's performance and competence, and the different neural networks and characteristics that these people present. The types of aphasia that will be mostly analysed and addressed throughout the paper will be Broca's and Wernicke's aphasia.

2. Early findings in lexical access in aphasia

As stated at the beginning of section 1.2, the first discoveries that set the starting point of aphasiology were the findings obtained by Broca and Wernicke during the end of the 19th century. These new discoveries implied the beginning of a systematic study of language pathologies focusing on the relation between language and the brain, and also the baseline of many questions regarding the language impairment that different patients presented. For instance, as reported by Dell et al. (1997), even the father of psychoanalysis, Sigmund Freud, was interested in the issue of aphasia and word retrieval. In his work *On aphasia: A critical study* (1981), he claimed that "the paraphasia in aphasic patients does not differ from the incorrect use and the distortion of words which the healthy person can observe in himself in states of fatigue or divided attention" (Freud, 1891/1953, p. 13). The concern of trying to comprehend language impairment in aphasic people started to become an ongoing debate and a matter of study. In the following sections 2.1 and 2.2, an account of the early findings concerning both phonological and semantic factors in lexical access in aphasia will be given, as well as a description of the types of errors that these patients present in section 2.3.

2.1. The effect of phonological factors in lexical access in aphasia

Some of the most relevant findings regarding word retrieval in PWA started to appear after the second half of the 20th century (Levelt, 2013). Prior to this, exploratory studies had already been conducted, but due to the inconsistent measures and the lack of specific instruments, results were contradictory and non-specific many times (Hagoort, 1993). The possibility that an interaction impairment between phonological and semantic factors in word processing existed in PWA was first explored by Baker et al. (1981). By designing a series of tasks that systematically increased the demand for semantic processing conditioned by phonological discrimination, the authors found that as phonological discrimination became more difficult, semantic processing for PWA became harder. In this experiment, both Wernicke's and Broca's aphasic patients presented similar results, but the effects of phonological discrimination dificulties were found to be significantly higher in those with Wernicke's aphasia (Baker et al., 1981, as cited in Milberg et al., 1988). This study marked the beginning of a series of works that questioned the role of phonological factors in word processing with PWA.

A highly contributing study that explored monolingual English PWA speakers' competence in lexical access was the one conducted by Milberg et al. (1988). Based on the phonological priming paradigm,[3] their main aim was to explore how PWA make use of the information provided by phonetic features in the course of lexical access.

In Milberg et al.'s (1988) experiment, both fluent and non-fluent PWA were auditorily tested with pairs of words in which the initial phoneme of the first word was systematically changed, e.g. "cat-dog", "gat-dog", "wat-dog". The aim of the experiment was to study whether semantic facilitation would occur in PWA with a nonword which was phonologically similar to a real word.[4] In the case of healthy subjects, the nonword stimulus that shared some phonetic features with an actual word in the vocabulary received a lexical interpretation. In the case of PWA, however, results were different. Non-fluent PWA showed priming only in the undistorted word conditions, whereas fluent PWA showed priming in all phonological distortion conditions (Milberg et al., 1988). Due to this difference between fluent and non-fluent PWA, a second task was conducted in which subjects had to make a lexical decision on the nonword primes presented individually. Under these new circumstances, both fluent and non-fluent aphasic patients presented phonological feature sensitivity. Taking these results into consideration, the authors claimed that in patients with aphasia, impairments in the use of phonological information to access the lexicon can emerge in various

[3] The priming paradigm in psycholinguistics is an effect by which a subject is exposed to a specific stimulus (a word) which influences the reaction to a subsequent target (Molden, 2014), i.e. "doctor" primes "nurse". It is usually unconscious.

[4] A nonword is a set of speech sounds that resembles a real word (for further information, go to section 2.3).

ways. In fact, they suggested that fluent PWA may have a lower lexical access threshold of sensitivity than nonfluent PWA. As a result, fluent PWA would be less sensitive to phonological distortion, allowing them to access more words in the lexicon than usual, whereas non-fluent PWA would be more sensitive to phonological distortion, allowing them to access fewer words in the lexicon than usual. These results indicated that changes in the ability to access word meaning via phonological information are crucial to the understanding of aphasia (Milberg et al., 1988). In the following section 2.2, the way semantic access is affected in lexical access in aphasia will be described.

2.2. The effect of semantic factors in lexical access in aphasia

As explained in section 1.3, the mental lexicon is conditioned by semantic factors as well as phonological factors. The semantic similarity between two words refers to the strength of the semantic relationship between the words, and words belonging to the same semantic field are strongly related and grouped together in networks inside our mental lexicon (Gómez-Vidal, 2022). The previous section 2.1 focused on how classical publications (such as Milberg et al., 1988) claimed that phonological factors were crucial for the understanding of aphasia when PWA access the lexicon. In this section, how semantic factors affect lexical access in aphasia will be explained, addressing the earliest and most relevant data gathered in up-to-date experiments.

2.2.1. The need for a distinction between automatic and controlled processing

According to Hagoort (1993), the studies that had been conducted in order to understand the lexico-semantic processing in PWA had primarily employed explicit semantic judgements up to that time; that is, subjects were presented with pairs or triplets of words, for example, and they were required to explicitly select the words that went together. In all of these types of experiments, subjects with Wernicke's aphasia were especially claimed to be the ones that displayed a deficit in the activation of the semantic information associated with the lexical item.[5] In contrast to Broca's PWA and healthy control subjects, subjects with Wernicke's aphasia were generally the ones

[5] As explained in section 1.3, a lexical item contains both phonological and semantic (meaning) information (in addition to other types of information).

incapable of grouping words according to their shared semantic features (Hagoort, 1993). Broca's PWA, on the contrary, were found to have a more or less intact semantic lexicon as a general rule (Zurif et al., 1974, as cited in Hagoort, 1993). However, a number of more recent studies suggested that the data gathered until that time could be inaccurate, as the account that a lexico-semantic deficit only occurred in Wernicke's PWA was disproved by new results. By adding the component of a priming effect in these more recent experiments, the results obtained were just the contrary to the data gathered in previous studies. For this reason, these inconsistent findings prompted researchers to investigate why individuals with Wernicke's and Broca's aphasia sometimes showed diminished capacity in the lexico-semantic processing and sometimes did not (Hagoort, 1993).

Milberg et al.'s *Processing of lexical ambiguities in aphasia* (1987) was the priming study of monolingual English speakers with aphasia that demonstrated the most significant difference between Broca's and Wernicke's PWA in the lexico-semantic processing and it also provided an explanation for the discrepancies obtained in previous research (Hagoort, 1993). In this study, a lexical decision task was designed in which Wernicke's and Broca's PWA had to judge whether the third stimuli of an auditorily presented triplet of stimuli was a real word or not. Three different types of triplets were used in the experiment: triplets where the first and third terms were related to (a different) one of the semantically ambiguous second word's meanings (e.g., money-bank-river), triplets where the first and third terms were both related to (the same) one of the ambiguous second word's meanings (e.g., shore-bank-river), and triplets where the first and third terms were related to neither of the second word's meanings (e.g., tent-bark-tree). In this experiment, Wernicke's PWA showed the same priming effects as the control subjects. They revealed selective access to multiple meanings of ambiguous words, as evidenced by the fact that the context provided by the first word influenced the semantic facilitation on the third word. Broca's PWA, on the other hand, demonstrated no semantic facilitation in any of the priming conditions; they showed a reduced ability. Milberg et al. (1987), as a consequence of these new results, proposed that the differences which arose between Wernicke's and Broca's PWA were due to the difference between automatic and controlled processing.[6] They claimed that

[6] Automatic processing is quick and unconscious; the subject's attention or awareness is not required. Controlled processing, on the other hand, is slow and conscious, and it is under the subject's deliberate control. In controlled processing, contrary to automatic processing, the subject has time to create expectancies and use strategies (Hagoort, 1993).

in the processing and access to the lexicon, some processes are automatic (priming) whereas others are controlled (explicit judgements). Thus, the authors asserted that Wernicke's PWA are in fact able to access word meanings automatically (they show priming), but are impaired in consciously analysing word meanings. For Broca's patients, within the same regard, the authors proposed just the inverse pattern: Broca's PWA have little or no trouble with controlled processing but they display a problem with automatic access to the lexico-semantic information (they show no priming) (Milberg et al. 1987, as cited in Hagoort, 1993). In section 2.2.2, the way PWA actually perform depending on automatic and controlled processing will be explained.

2.2.2. Testing both automatic and controlled processing in PWA

Hagoort (1993) suggested that the access to the lexico-semantic information in implicit task settings which do not make the subjects focus on the semantics of the presented words may differ from the access to the lexico-semantic information under explicit task conditions. The authors argued that the different types of experiments designed when testing lexico-semantic processing had to be conscientiously meditated as they could give rise to different types of processing and results. For this reason, and by claiming that "to date all priming studies with aphasic patients have used a fixed interval between primes and targets" (p. 193),[7] Hagoort (1993) designed an extension to Milberg et al.'s (1987) experiment in order to separate automatic from controlled lexico-semantic processing under exactly the same task conditions.

With this innovative experiment, the authors aimed to obtain the aforementioned distinction between automatic and controlled processing by manipulating the interstimulus interval (ISI) between the different priming and target words. They wanted to explicitly see what happened if they varied the intervals within the same experiment. They claimed that if priming happened at the shortest interval, it would be due to automatic processing (fast and unconscious processing) whereas if priming occurred at the longest interval, it would be because of controlled processing (as they would have more time to decide). Therefore, the authors varied the silent intervals between the triplet members, designing three different tests. In the first test, the terms in

[7] Hagoort (1993) claims that all previous studies used a silent interval of 500 milliseconds between primes and targets, a relatively long delay to safely differentiate automatic processing from controlled processing.

the triplets appeared after 100 milliseconds (ms) each. In the second, after 500 ms, and in the third after 1250 ms. In general, priming effects between the terms of each triplets were found with both Broca's and Wernicke's PWA at ISIs of 100 and 500 ms, but not at 1250 ms. In comparison, healthy controlled subjects did display the priming effect also at the longest interval. Apart from the experiments containing the priming paradigm, Hagoort (1993) designed a final test in which patients had to explicitly judge the semantic relations between the words (creating also a necessary need for controlled processing). In this case, as following the line established by Milberg et al. (1987), Wernicke's patients performed worse than Broca's ones and than controls.

By modifying the ISIs between the words and by adding a final test in which explicit lexico-semantic judgement was needed, the results obtained in Hagoort's (1993) experiment proved that both Wernicke's and Broca's patients can access the semantic lexicon automatically (as they showed a priming effect in the shortest interval), but may struggle to integrate lexico-semantic information into the context (as they failed to give accurate answers in longer ISIs). The authors explained that a shorter timespan of semantic priming may be given in PWA as the activation decays more quickly in the mental lexicon of aphasic individuals. This would be the reason why a shorter temporal scope for automatic spread is given in PWA. Finally, the authors argued that if controlled processes play a stronger role in priming effects with longer ISIs, the decrease in priming observed with longer ISIs could be due to a problem with controlled processing (Hagoort, 1993).

Under all the previously analysed assumptions that set worth considering differences between semantic and phonological factors and automatic and controlled processing in lexical access in aphasia, the subsequent experiments started to notice the specific types of errors that PWA made when accessing the lexicon (detailed in section 2.3).

2.3. Types of errors in lexical access in aphasia

As seen in sections 2.1 and 2.2, both phonological and semantic retrieval are part of lexical access, but each of these processes produces different results in experiments with PWA and accounts also for different types of processes inside the brain. In fact, as stated by Cappa el al. (1981), "(…) retrieval of the semantic word form and the phonological word form are distinct processes, supported by spatially distinct regions of the brain and hence subject to selective damage" (Dell et al., 1997, p. 802). This statement supports the idea that both phonological and semantic factors have to be considered when studying lexical access in PWA, but need to be analysed by using alternative meditated tasks as they are two complementary but independent processes. For this reason, the types of errors that PWA make in performance tasks also need to be classified concerning phonological or semantic factors. In the experiments that have already been discussed, results provided information and crucial ideas that need to be considered, but did not produce specific and consistent data. For this reason, a specific report on the types of errors that PWA make when accessing the lexicon is needed, as a set out point for the findings that the literature on aphasia has discovered. These types of errors were noticed in an experiment run by Dell et al. (1997). In their work, an account of specific phonological and semantic errors was given, with a differentiation between sublexical paraphasias and lexical paraphasias.

Sublexical paraphasias, on the one hand, were defined as errors that involve phonemic impairments; that is, impaired encoding for articulation of the phonological representation retrieved from the lexicon. Lexical paraphasias, on the other hand, involved impairments in the whole word level, such as the substitution of a word that bears a semantic relation to the target. Some naming errors were also claimed to be interpreted as "two-step" errors: *unicorn* uttered as "house";[8] *unicorn → horse* seen as a lexical error first that is then followed by a phonological access error that transforms *horse* into "house" (Martin et al., 1994, as cited in Dell et al, 1997). Considering this, Dell et al. (1997) distinguished five types of errors concerning lexical access in aphasia: semantically related word errors, formally related word errors, mixed semantic and formal errors, unrelated word errors and neologisms or nonwords.

A semantic error occurs when the two concepts that are confused share semantic nodes (i.e. "dog" for *cat*). As explained in section 1.3, during lemma access (word activation/recognition in our lexicon), the shared semantic nodes of *cat* immediately activate also the word node for *dog.* In order to select *cat* and not *dog,* however, the activation of *cat* must be higher than that of any other noun, including *dog*. A semantic error, therefore, may happen due to the noise in the activation level.

[8] Italics will be used to represent that the word written is the target, i.e. the intended word the speaker wanted or had to say. Double quotation marks will be used to represent that the word written is the actual word the speaker had uttered.

A form-related error (i.e. "mat" for *cat*) is a sublexical paraphasia where phonemic impairment plays a role. When this type of error happens, the target phonemes to the word layer activate nodes for words that share phonemes with the target. As a result, words such as *mat* or *sat* would become more active. A form-related error, therefore, occurs during phonological access because the proper word, *cat*, may have been selected during lemma access, but one or more phonemes of *cat* may be replaced by alternative phonemes due to the interference from other activated words or noise.

Mixed semantic-formal errors happen due to both an error in lemma (as the error can be a word which carries semantic similarity with the target) and phonological access (as it can be a word which shares a phoneme with the target). When a mixed semantic-formal error happens, these two steps or processes are impaired and distorted. When uttering "rat" for *cat,* for example, the node of "rat" receives activation from shared semantics but also from feedback from shared phonemes.

Unrelated word errors are neither semantically nor formally related to the target. This category includes mistakes like "log" for *cat*, which can indicate a long-distance connection (*cat* is related to *dog*, which is related to *log*). Unrelated word errors are regarded as "two-step" errors that happen due to both lemma and phonological access respectively.

Finally, there are two types of neologisms or nonwords: the ones that resemble the target and the ones which do not. The first type of neologisms (i.e. "lat" or "cag" for *cat),* indicate a problem at phonological access. The nonwords that do not resemble the target, on the contrary, could be the result of a significant disruption of phonological access or problems with both lemma and phonological access.

All things considered, Dell et al.'s (1997) paper presented the prototypical errors that PWA are expected to make when speaking. Wernicke's and Broca's patients, therefore, were expected to present all of these types of errors to some extent in their speech. Some more recent papers (Swanberg et al., 2007), however, claim that Wernicke's PWA typically show more paraphasias than Broca's PWA, being semantic paraphasias the most frequently found type. In Wernicke's aphasia, phonemic paraphasias and neologisms have also been found to be more frequent than in Broca's aphasia (Swanberg et al., 2007).

Along section 3 of this paper, an account of the recent discoveries concerning lexical processing in PWA will be presented, where the types of brain imaging techniques that are used today will be explained together with the data obtained thanks to these new neuroimaging techniques.

3. Current neuroimaging techniques and findings in lexical access in aphasia

The main challenge of neurolinguistics inside the lexical retrieval process these days is the identification of its components, their localisation in specific brain areas, and the description of the information-flow between them across time (Friedmann et al., 2013). For this purpose, in recent years various brain imaging techniques have been developed that have led to different discoveries and identification of concrete brain areas responsible for specific tasks. Reasonably, these new techniques have represented a great advance in understanding language impairments suffered by aphasic patients. In section 3.1 of the paper, various brain imaging techniques that are used today will be presented to better understand lexical retrieval in PWA. Then, in section 3.2, what these techniques and recent research has contributed to the understanding of lexical retrieval access in Wernicke's and Broca's PWA will be described.

3.1. Neuroimaging techniques

In order to know exactly where and when we process language in our brain, within the study of language processing, several brain imaging techniques have been developed and used. Electroencephalography (EEG), Magnetoencephalography (MEG) and Functional magnetic resonance imaging (fMRI), for example, are time-sensitive technologies for tracking brain activation with millisecond precision; that is, techniques that show the brain in real time action. These three techniques are used nowadays to study lexical access in both healthy and unhealthy subjects. On the one hand, EEG records the changes in the electrical voltage generated by groups of neurons by placing electrodes on the subject's scalp (Zipse, 2008) (Figure 3). The results obtained are waves that show the electrical charge of our brain cells' activity. Then, the electrical activity of our brain is transformed into a picture that shows the neurons' level of electrical voltage on the surface of the scalp (Sedivy, 2014) (Figure 4).

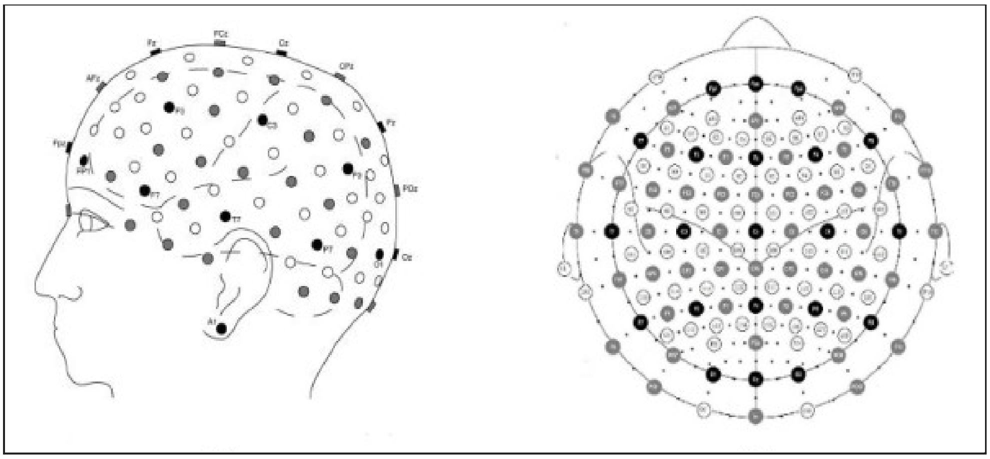

Figure 3. Distribution of EEG electrodes on the scalp (taken from Oostenveld & Praamstra, 2000)

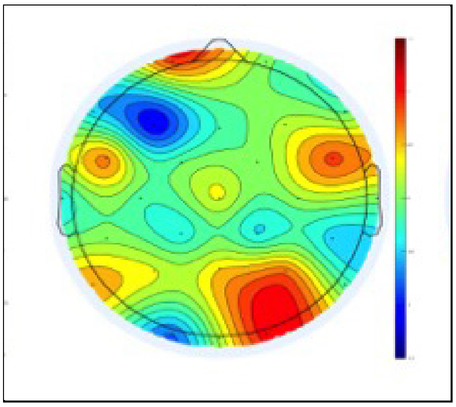

Figure 4. Representation of semantic violation EEG waves in an aphasic patient (taken from Barbieri et al., 2016).

On the other hand, MEG measures the magnetic signals created by the brain's electrical activity. Unlike EEG, MEG localises the signals generated by neurons as they are activated, as they communicate and as activity spreads through them. By combining those magnetic signals obtained through MEG with anatomic images obtained via Magnetic resonance imaging (MRI), the results obtained in MEG are images that show the magnetic activation in a specific area of the brain (Zipse, 2008) (Figure 5).

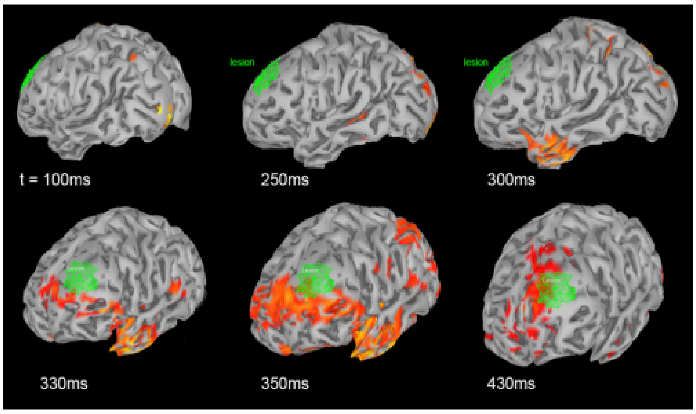

Figure 5. Magnetic signals obtained of language-related processes in a brain tumour patient during a word-reading test. The green colour represents the lesion (taken from McConnell Brain Imaging Centre, n.d.).

Finally, fMRI records the hemodynamic changes in the brain (the changes in the blood oxygen level and the direction of the blood flow). fMRI's results are also specific images of the brain, but in this case, images that show a comparison between different brain activities. The areas which are shown in colour in fMRIs are the ones in which a higher and more oxygenated blood flow is taking place (Sedivy, 2014) (Figure 6).

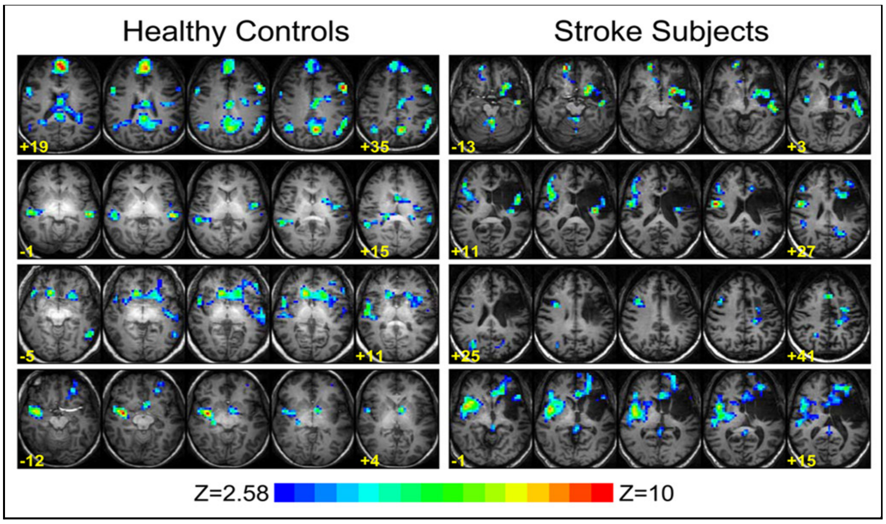

Figure 6. fMRI results of the activation level of a picture-naming task of healthy subjects (left) and post-stroke aphasic subjects (right) (taken from Szaflarski, 2010).

Each of the aforementioned methods, the EEG, MEG and fMRI, has its own set of benefits and drawbacks. In some ways, EEG allows for more activity visualisation, but it is less clear than MEG and fMRI (Cohen & Halgren, 2003, as cited in Zipse, 2008). This is because EEG receives electrical waves through the scalp, and the bone and skin distort electrical signals, but not magnetic signals. As a result, source localisation with MEG is often superior to EEG. In the case of fMRI, source localisation is optimal as it directly shows the blood-difference in specific areas of the brain. It is vital to remember, therefore, that although these methods provide similar information in some cases, EEG, MEG and fMRI are likely to reflect slightly different parts of the same complex of activity in many circumstances (Zipse, 2008).

Within lexical access, EEG, MEG and fMRI are often used in conjunction with behavioural paradigms (i.e., lexical decision tasks, priming effects…) in order to obtain additional and the most accurate information. This is because brain imaging methods allow researchers to keep track of cognitive subprocesses that participants may not even be aware of in behavioural tasks. Furthermore, most behavioural studies track a single-dimensional variable, such as reaction time, whereas EEG, MEG and fMRI typically allow for complex analyses with several dimensions. In addition, these imaging techniques are also effective in clinical groups where participants may be unable to carry out sophisticated tasks (Zipse, 2008).

EEG, MEG, and fMRI, consequently, have been used in the literature to characterise a number of electrophysiological and neuroanatomical information related to speech and language processing, as well as to plot brain activity in PWA during lexical processing activities. The majority of the research done through brain-imaging techniques concerning aphasia has focused on analysing picture-naming tasks. Moreover, compared to the number of studies using EEG and fMRI, there has been little research utilising MEG in the study of lexical access in PWA (Zipse, 2008).

A quite recent example of a lexical access experiment of PWA using a neuroimaging technique was the one conducted by Johnson et al. (2019). They used a fMRI picture-naming task to examine 16 specific regions of the brain in 26 patients with chronic aphasia. The fMRI picture-naming task was used before and after 12 weeks of semantic naming treatment. The results showed that the primary effect of the beneficial naming treatment in PWA was an upregulation of traditional language areas in their brain (Johnson, 2019). Brain imaging techniques, therefore, have enabled researchers to acknowledge the exact neural changes and the brain activity that PWA present inside their brain, being a tool that has consequently brought several improvements in the study of aphasia. In section 3.2, the specific findings that have recently been discovered about Wernicke's and Broca's lexical processing impairments will be addressed.

3.2. Recent findings concerning Wernicke's and Broca's PWA

Wernicke's and Broca's areas have been specifically analysed through brain-imaging techniques, so today, the specific function of these areas has been precisely set and discovered. DeLeon et al. (2007), for example, conducted a study with PWA in which they found the correspondence between brain lesions and specifically set deficits. Section 3.2.1 will focus on DeLeon et al.'s (2007) study, where the particular areas of the brain responsible for word processing will be shown, and section 3.2.2 will address the specific impairments Wernicke's and Broca's PWA present. Finally, in section 3.2.3, a factor that nowadays' research has considered key to understanding these two aphasic groups' disabilities will be presented and explained: lexical competition.

3.2.1. Specific brain areas for lexical access that include Wernicke's and Broca's areas

DeLeon et al. (2007) found that lesions in Brodmann areas 21 (which corresponds to the middle temporal gyrus (MTG) in the brain)[9] (see Figure 7) and 22 (superior temporal gyrus (STG), including Wernicke's area) (see Figure 7) were most frequent in the group of patients with semantic/conceptual deficit.[10] It was concluded that these areas were the ones that serve the conceptual/semantic system inside the brain. In other words, the authors claimed that Wernicke's area corresponded to the conceptual system and the semantic lexicon. In this study, the authors also found that a post-lexical deficit was associated with lesions in areas 44 and 45 (inferior frontal gyrus (IFG) and Broca's area) (see Figure 7). The authors of the experiment asserted that these two areas were involved in later motor and articulatory processes (DeLeon et al., 2007, as cited in Friedmann et al., 2013).

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[9] The term gyrus (pl. gyri) refers to the folds which comprise the cerebral cortex of the brain (Deng et al., 2013).

[10] Korbian Brodmann was the first to describe the human brain's cortex. He provided precise subdivisions of the brain. These subdivisions were defined by the different structure and organisation that neurons displayed in each of them. In total, he described 52 different regions, including language network regions. Today, brain regions are named "Brodmann 1-52" (Friederici, 2011).

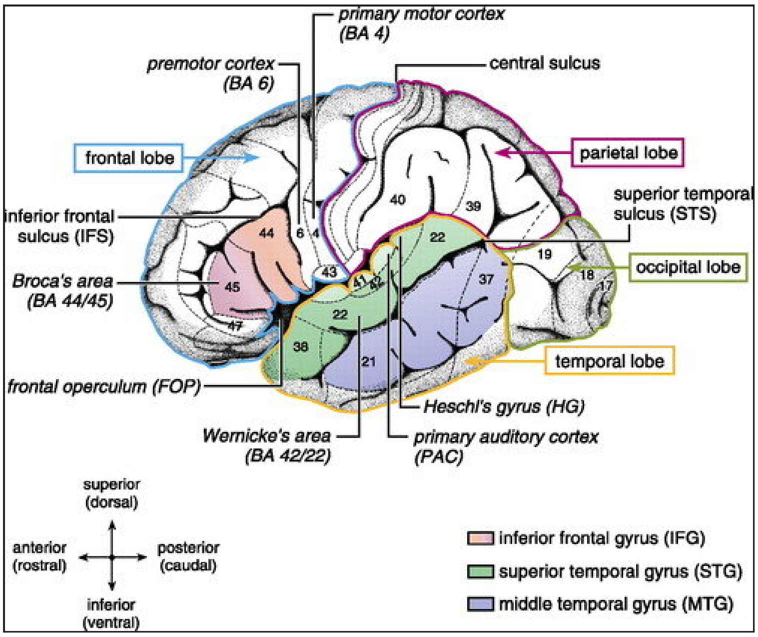

Figure 7. Language-relevant areas and Brodmann areas in the left hemisphere of a human brain (taken from Friederici, 2011).

All things considered, DeLeon et al.'s (2007) findings proved that both posterior and anterior brain structures are involved in the process of lexical access. In fact, it was shown that the superior and middle temporal gyri (STG/MTG), which are in the posterior brain structure, are involved in the lexico-semantic processing. As far as the anterior brain structure is concerned, the inferior frontal gyrus (IFG) has been found to be the region involved in the later motor and articulatory processing (DeLeon et al., 2007, as cited in Friedmann et al., 2013). With this neuroimaging experiment, DeLeon et al. (2007) proved that both Wernicke's and Broca's areas are involved in the command of lexical access and processing, accounting that a lesion in either or both of these areas is what creates the lexical impairments that happen to Wernicke's and Broca's PWA. In fact, sections 3.2.2 and 3.2.3 focus on the current discoveries that describe Wernicke's and Broca's PWA's deficits on lexical access.

3.2.2. Specific impairments in Wernicke's and Broca's PWA

Recently, a deficit in three areas concerning lexical access have been attributed to Wernicke's and Broca's PWA. These areas are graded activation, competition, and interactivity (Yee et al., 2008). Concerning the three of them in healthy individuals, the speech-lexical processing system is first activated in a graduated manner; that is, activation patterns are not all-or-none, but rather have a graded level of activation. Second, there is a lot of competition among lexical candidates, and the degree of competition affects the timing and patterns of activation at each level of representation, as well as at the overall system's performance. Lastly, processing stages interact with one another, causing activation patterns at one level to affect activation patterns at subsequent levels (Yee et al., 2008).

Conditioned by the three characteristics mentioned above, Yee et al. (2008) claim that the nature and origin of the lexical processing disorders in Broca's and Wernicke's PWA could be due to two primary hypotheses. According to the first hypothesis, the degree of activation of lexical candidates is the source of the impairments present in Broca's and Wernicke's PWA. Within this regard, total lexical activation is reduced in Broca's PWA while it is raised in Wernicke's PWA. Two examples of this would be experiments conducted by Milberg et al. (1987, 1988) (see sections 2.1 and 2.2.2). The findings obtained in those two studies demonstrated precisely what the first hypothesis argues: that total lexical activation is reduced in Broca's PWA while it is raised in Wernicke's PWA. The second hypothesis, on the contrary, claims that the temporal course of lexical activation is the source of Broca's and Wernicke's impairment. According to this view, the time course of lexical activation is thought to be delayed in Broca's PWA, resulting in a later than typical rise time (see Swinney et al., 2000). By this hypothesis, Wernicke's PWA, conversely, have a delay in lexical deactivation; that is, Wernicke's PWA show a difficulty in suppressing once-activated word candidates (see Prather et al., 1997 as an example, as cited in Yee et al., 2008).

Although the two hypotheses provide different reasons as key factors to understanding the deficits that Wernicke's and Broca's PWA show, as far as lexical competition is concerned, the two hypotheses agree on the facts that lexical competitors are active for longer than usual in Wernicke's PWA and shorter than usual in Broca's PWA (Yee et al., 2008). In order to explore how lexical competition affects Broca's and also Wernicke's PWA, Yee et al. (2008) designed an experiment which tested both aphasic groups in lexical competition. In section 3.2.3, Yee et al.'s (2008) research will be described along with the findings they obtained which are determinant to comprehend lexical access impairments in Wernicke's and Broca's PWA.

3.2.3. Lexical competition: key to understanding Wernicke's and Broca's aphasia

One of the first ideas claimed by Yee et al. (2008) is that until that date, only Broca's PWA had been tested within the concern of lexical competition. Despite the fact that it was clear that Wernicke's PWA showed lexical processing impairments, it was unclear if they would also show lexical processing impairments in the presence of lexical competition, as Broca's PWA showed. For this reason, Yee et al. (2008) investigated if the role of lexical competition also affected Wernicke's PWA. For this aim, they used the Eye-tracking method, which is a common methodology to measure word recognition in experimental subjects. Specifically, participants in Eye-tracking experiments are exposed to linguistic stimuli via reading or listening, with the notion that language mediates eye-movements, that is, linguistic processing causes changes in participants' eye movements (Gómez-Vidal, 2022). Most importantly, this Eye-tracking methodology allows both the activation of a lexical target and its competitor to be tracked over time. Therefore, eye-movements have the potential to provide extensive information regarding lexical activation and, as a result, regarding the role of the anterior and posterior brain areas in lexical processing in Broca's and Wernicke's aphasia (Yee et al., 2008).

Yee et al. (2008) employed the visual world paradigm, in which participants are exposed to auditory linguistic stimuli while observing visual stimuli on the screen. This is used under the assumption that participants glance at specific things such as images or words if those are related to the auditory stimuli they are listening to (Gómez-Vidal, 2022).

Yee et al.'s (2008) experiment consisted of three different tests where the visual stimuli that appeared on the screen were either different pictures or pairs of words. Subjects, therefore, depending on the auditory stimuli, glanced at different pictures or words and fixated their eyes on a specific stimulus. In the first test, the authors examined whether Broca's and Wernicke's PWA preferentially fixated on items semantically relevant to the uttered word. For example, given the auditory presentation of "hammer," the authors wanted to explore whether fixations to a picture of a *nail* would increase. The findings obtained in this test would demonstrate if patients' ability to transfer sound structure onto the lexicon and access the lexico–semantic network was impaired or not. In the second test, whether Broca's and Wernicke's PWA exhibited competing effects from words with similar phonological onsets to the uttered word was investigated. Given the auditory presentation of "hammer" again, the authors now wondered if fixations on a picture of *hammock* would increase. In this case, the findings would give proof of subjects' specific ability to choose a word candidate as regards lexical form competition. Finally, with the third test, the authors investigated the extent to which Broca's and Wernicke's PWA activated words semantically related to the uttered word's phonological onset competitor. They gave an auditory presentation of "hammock" and in this case they questioned if increased fixations to *nail* due to activation of the onset competitor "hammer" would occur. Subjects' ability to activate the lexico-semantic network of words that are phonological competitors of a heard lexical candidate would be tested.

In the first test, the results obtained suggested that both Wernicke's and Broca's PWA presented typical semantic fixations as healthy subjects. Both aphasic groups could fixate on an object semantically related to the target; when presented with an audio of "hammer" they fixated on "nail" as healthy subjects did. However, the results obtained in tests 2 and 3 were different to those of healthy subjects, demonstrating that Wernicke's and Broca's PWA have deficits in the dynamics of lexical activation. In test 2, Wernicke's PWA showed increased fixations to onset competitors (*hammock → hammer*) while Broca's PWA failed to specifically fixate on them. In test 3, Wernicke's PWA also showed increased fixations to semantically related words that shared their phonological onset with the target (*hammock → nail)* and Broca's PWA did not fixate on them.

Taking into account the data gathered in the three tests of the study, Yee et al. (2008) suggested that Wernicke's and Broca's lexical impairments were specific: Wernicke's PWA had increased lexical activation, whereas Broca's PWA had reduced lexical activation. Yee et al. (2008) claimed that the reason for all subjects displaying a typical semantic fixation in test 1 went along with recent neuroimaging results; they stated that, as demonstrated via neuroimaging experiments, both anterior and posterior areas of the brain were responsible for lexical access in the brain (see section 3.2), so lesions involving only one of those areas (as in Wernicke's and Broca's aphasia) could be the reason why the subjects in Yee et al.'s (2008) study showed typical semantic relatedness. Yee et al. argued that "(…) it is possible that neither region is necessary or sufficient to result in abnormal performance if lesioned" (Yee et al., 2008, p. 609).

Relating the experiment and the results obtained by Yee et al. (2008) to the previously described study by Milberg et al. (1988) (see section 2.1), the claim proposed by Yee et al. (2008) is proved to be coherent and plausible. In Milberg et al.'s (1988) study, typical lexical priming effects were found in both Wernicke's and Broca's PWA, which were regarded as non-expected data because they did not explain the source of the impairments of both groups compared to healthy subjects. Based on Yee et al. (2008), Milberg et al.'s (1988) results become understandable. In short: Broca's and Wernicke's PWA show non-impaired lexical priming, because either Broca's or Wernicke's regions can be damaged and still present no lexico-semantic processing impairment, taking into account that both anterior and posterior regions of the brain together are responsible for it.

4. Conclusions

The impairments present in Broca's and Wernicke's aphasia concerning lexical access have been a matter of study for many years. The discoveries of Paul Broca and Carl Wernicke at the end of the 19th and beginning of 20th centuries were a starting point where the first advances in aphasiology began. These two scientists found anatomical evidence of people with two types of brain lesions that produced two different types of language disturbances; one that showed impairments in language production and another that showed impairments in language comprehension. As a consequence of these findings, research regarding linguistic impairments began to be relevant and abundant in the literature, where one of the most studied types of language impairment was the one related to lexical access. Little by little, advances and discoveries in the lexical access of Broca's and Wernicke's PWA began to be seen.

On account of the theory presented by Collins and Loftus (1975) that explained how lexical items are semantically conditioned and connected in the brain and because of the fact that the mental lexicon is also organised phonologically, some of the first works concerning lexical access in aphasia investigated the role of both phonological and semantic factors in word processing in PWA. In this way, studies such as the ones conducted by Milberg et al. (1987, 1988) showed that phonological factors as well as semantic factors were crucial for the understanding of aphasia when accessing the lexicon, as the results in the experiments showed atypical performances of PWA comparing them to healthy subjects. However, in some of the cases, the results also showed typical semantic relatedness, which was something that could not be explained by the authors. A number of more recent studies, therefore, suggested that the data gathered until that time could be inaccurate, and that there was a need to design experiments with specific instruments and tasks in order to obtain reliable and consistent data. Hagoort (1993) explicitly found that changes in the ISI of target words triggered different results in Broca's and Wernicke's PWA, inferring that a distinction between automatic and controlled processing was needed in order to properly study lexical access in aphasia. Specifically, Hagoort (1993) observed that a shorter timespan of semantic priming may be given in PWA as the activation decays more quickly in the mental lexicon of aphasic individuals.

In addition, current neuroimaging techniques have enabled researchers to accurately observe and locate the brain areas which are responsible for language processing, as well as to acknowledge the exact neural changes and the brain activity that PWA present inside their brain when accessing their lexicon. Precisely, experiments using brain imaging techniques, such as EEG, MEG and fMRI, have found that Wernicke's area corresponds to the conceptual system and the semantic lexicon, whereas Broca's area corresponds to later motor and articulatory processes. DeLeon et al. (2007) proved that both Wernicke's and Broca's areas are involved in the command of lexical access and processing, accounting that a lesion in either or both of these areas is what creates the lexical impairments that happen to Wernicke's and Broca's PWA. Therefore, it was shown that both posterior and anterior brain structures are involved in the process of lexical access.

Taking the aforementioned literature into account, Yee et al.'s (2008) experiment provided detailed evidence to comprehend which impairments actually occur in the lexical access of Wernicke's and Broca's PWA. Determined by the neuroimaging results that claimed posterior and anterior brain areas to be in control of the lexico-semantic processing, Yee et al. (2008) explained the reason why semantic relatedness can be intact in both Broca's and Wernicke's PWA (as observed in classical publications such as Milberg et al., 1988). In addition, the authors argued that the impairments exhibited in these aphasic patients could be determined by the specific deficit Wernicke's and Broca's PWA present when considering lexical competition, stating that Broca's PWA have reduced lexical activation while Wernicke's PWA have increased lexical activation.

All in all, this paper has shown that research in lexical access in Broca's and Wernicke's PWA has had an evolution where, at first, innovative but inconsistent data were obtained, but based on building specific experiments and taks, and with the advancement in neuronal imaging techniques, studies have been consolidated and and have proved reliable, specific and consistent discoveries. Specifically, this paper has shown how lexical access in PWA is conditioned by the impairments patientes present in lexical competition, as well as by the differences PWA present when considering distinct types of processing (automatic vs. controlled processing), giving researchers insight into the brain processes that underlie lexical processing in PWA.

However, a fact that has to be taken into account is that this paper does not explore the alterations in the lexical access in all types of aphasia, as the paper focuses mainly on Broca's and Wernicke's PWA. In order to have a complete view of how lexical processing occurs in all PWA, a specific analysis of each type would be needed, as done in this paper with Wernicke's and Broca's PWA.

After having analysed the studies and experiments described in this paper, and led by the fact stated by Arantzeta (2021) that says that almost all the scientific knowledge about aphasia is based on monolingual Anglophone speakers, it is important to highlight that an extensive study of only one type of linguistic profile is not enough to understand how PWA access their lexicon in holistic terms. Taking this idea into consideration, more research is required in the literature concerning bilingual PWA, specifically, Broca's and Wernicke's PWA, in order to expand on the knowledge of their lexical processing. Moreover, other languages apart from English, such as minority languages, also need to be studied and taken into account, as every different aspect that a patient shows in their mother tongue will be crucial for the development and treatment of the lexical impairment that they present.

References

Ahlsén, E. (2006). *Introduction to Neurolinguistics.* John Benjamins Publishing Company.

Arantzeta, M. (2021). Afasiaren diagnostikoa eta terapia pertsona elebidunetan: eraginkortasuna eztabaidan. In K. Erdozia, M. Santesteban & A. Zawiszewski (Eds.), *Bi hizkuntza garun bakarrean: Euskal psikohizkuntzalaritzaren ekarpenak eta erronkak* (pp. 65-87). Euskal Herriko Unibertsitatea.

Baker, E., Blumstein, S. E., & Goodglass, H. (1981). Interaction between phonological and semantic factors in auditory comprehension, *Neuropsychologia, 19,* 1-15.

Barbieri, E., Walenski, M., Hsu, C., Bovbjerg, K., Dougherty, B., Mesulam, M., & Thompson, C. K. (2016). Electrophysiological signatures of semantic and syntactic processing in Primary Progressive Aphasia: integration and re-analysis processes during auditory sentence comprehension. *Frontiers in Psychology. Conference Abstract: 54th Annual Academy of Aphasia Meeting.*

Cappa, S., Cavallotti, G., & Vignolo, L. A. (1981). Phonemic and lexical errors in fluent aphasia: Correlation with lesion site, *Neuropsychologia, 19,* 171-177.

Chomsky, N. (1986). Knowledge of Language: Its Nature, Origin and Use. Praeger.

Cohen, D., & Halgren, E. (2003). Magnetoencephalography (Neuromagnetism). In G. Adelman & B. H. Smith (Eds.), *Encyclopedia of Neuroscience, 3rd Edition.* Elsevier.

Collins, A. M., & Loftus, E. F. (1975). A spreading-activation theory of semantic processing. *Psychological Review, 82*, 407-428.

DeLeon, J., Gottesman, R. F., Kleinman, J. T., Newhart, M., Davis, C., Heidler-Gary, J., & Hillis, A. E. (2007). Neural regions essential for distinct cognitive processes underlying picture naming, *Brain, 130,* 1408-1422.

Dell, S. G., Martin, N., Saffran, M. E., Schwartz, M., & Gagnon, A. D. (1997). Lexical Access in Aphasia and Nonaphasic Speakers. *Psychological Review, 104,* 801-838.

Deng, F., Jiang, X., Zhu, D., Zhang, T., Li, K., Guo, L., & Liu, T. (2013). A functional model of cortical gyri and sulci. *Brain Structure and Function*, *219*(4), 1473–1491.

Diéguez-Vide, F., & Peña-Casanova, J. (2012). *Cerebro y Lenguaje: Sintomatología y neurolingüística.* Editorial Médica Panamericana S. A.

Ellis, C., & Urban, S. (2016). Age and aphasia: a review of presence, type, recovery and clinical outcomes. *Topics in Stroke Rehabilitation, 23,* 430-439.

Engelter, S. T., Gostynski, M., Papa, S., Frei, M., Born, C., Aidacid-Gross, V., Gutzwiller, F., & Lyrer, P. A. (2006). Epidemiology of aphasia attributable to first ischemic stroke: incidence, severity, fluency, aetiology, and thrombolysis, *Stroke, 37,* 1379-1384.

Freud, S. (1953). *On aphasia: A critical study* (E. Stengel, Trans.). International University Press. (Original work published 1891).

Friederici, A. D. (2011). The Brain Basis of Language Processing: From Structure to Function, *Physiological Reviews, 91,* 1357-1392.

Friedmann, N., Biran, M., & Dotan, D. (2013). Lexical retrieval and its breakdown in aphasia. In C. Boeckx & K. Grohmann (Eds.), *The Cambridge Handbook of Biolinguistics* (pp. 350-374). Cambridge University Press.

Gómez-Vidal, Beatriz (2022). Unit 4: Finding and using words. [Class handout]. University of the Basque Country, 25366.

Guy-Evans, O. (2021, July 18). *Wernicke's Area Location and Function.* SymplyPsychology. https://www.simplypsychology.org/wernickes-area.html

Hagoort, P. (1993). Impairments of Lexical-Semantic Processing in Aphasia: Evidence from the Processing of Lexical Ambiguities, *Brain and Language, 45,* 189-232.

Henseler, I., Regenbrecht, F., & Obrig, H. (2014). Lesion correlates of patholinguistic profiles in chronic aphasia: comparison of syndrome-, modality-, and symptom-level assessment, *Brain, 137,* 918-930.

Johnson, J., Meier, E., Pan, Y., & Kiran, S. (2019). Treatment-related changes in neural activation vary according to treatment response and extent of spared tissue in patients with chronic aphasia, *Cortex, 121,* 147-168.

Levelt, W. (2013). A History of Psycholinguistics. The Pre-Chomskyan Era. Oxford University Press.

Martin, N., Dell, G. S., Saffran, E. M., & Schwartz, M. F. (1994). Origins of paraphasia in deep dysphasia: Testing the consequences of a decay impairment to an interactive spreading activation model of lexical retrieval, *Brain and Language, 47,* 609-660.

McConnell Brain Imaging Centre. (n.d.). *Magnetoencephalography (MEG) at the BIC.* https://www.mcgill.ca/bic/meg-unit

Milberg, W., Blumstein, S. E., & Dworetzky, B. (1987). Processing of lexical ambiguities in aphasia, *Brain and Language, 31,* 138-150.

Milberg, W., Blumstein, S. E., & Dworetzky, B. (1988). Phonological Processing and Lexical Access in Aphasia, *Brain and Language, 34,* 279-293.

Molden, D. C. (2014). Understanding priming effects in social psychology: What is "social priming" and how does it occur? In D. C. Molden (Ed.), *Understanding priming effects in social psychology* (pp. 3-13). The Guilford Press.

Oostenveld, R., & Praamstra, P. (2000). The five percent electrode system for high-resolution EEG and ERP measurements. *Clinical Neurophysiology*, *112,* 713-719.

Prather, P. A., Zurif, E., Love, T., & Brownell, H. (1997). Speed of lexical activation in nonfluent Broca's aphasia and fluent Wernicke's aphasia. *Brain and Language, 59,* 391-411.

PsychBD. (2021, December 29). *Approach to Aphasia.* https://www.psychdb.com/neurology/approach-aphasia#approach-to-aphasia

Sedivy, J. (2014). *Language in Mind: An Introduction to Psycholinguistics.* Oxford University Press.

Swanberg, M., Nasreddine, Z., Mendez M., & Cummings J. (2007). Speech and Language. In C. G. Goetz (Ed.), *Textbook of Clinical Neurology (Third Edition)* (pp. 79-98). WB Saunders.

Swinney, D., Prather, P., & Love, T. (2000). The time-course of lexical access and the role of context: Converging evidence from normal and aphasic processing. In Y. Grodzinsky, L. P. Shapiro & D. Swinney (Eds.), *Language and the brain: Representation and processing* (pp. 273–292). Academic Press.

Szaflarski, J. P., Eaton, K., Ball, A. L., Banks, C., Vannest, J., Allendorfer, J. B., Page, S., & Holland, K. S. (2010). Poststroke aphasia recovery assessed with functional magnetic resonance imaging and a picture identification task. *Journal of Stroke & Cerebrovascular Diseases, 20,* 336-45.

Wallentin, M. (2018). Sex differences in post-stroke aphasia rates are caused by age. A meta-analysis and database query, *Plos One, 13.*

Yee, E., Blumstein, S. E., & Sedivy, J. C. (2008). Lexical-semantic activation in Broca's and Wernicke's aphasia: evidence from eye movements. *Journal of cognitive neuroscience*, *20*(4), 592–612.

Zipse, L. R. (2008). *A MEG Investigation of Lexical Access in Aphasia.* Health Sciences and Technology Program in Speech and Hearing Bioscience and Technology. Massachusetts Institute of Technology.

Zurif, E. B., Caramazza, A., Myseron, R., & Galvin, J. (1974). Semantic feature representations for normal and aphasic language, *Brain and Language, 1,* 167-187.