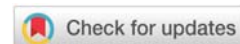




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Review Article

Aphasia: Definition, clinical contexts, neurobiological profiles and clinical treatments

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Abstract

Starting from the general concept of Aphasia, the present work focuses on the clinical, neurobiological, and functional aspects of the morbid condition, suggesting a multidimensional treatment between physiotherapy, psychotherapy, and rehabilitation exercises for lost skills.

Contents of the manuscript

The loss of the ability to understand, produce and reproduce a language, due to injuries to the areas of the brain responsible for its processing, is the definition of the neurological condition called “aphasia” (from the Greek ἀφασία, mutism). Therefore, speech disorders caused by primary sensory deficits, intellectual deficits, psychiatric disorders, or weakness of the musculoskeletal system are not included in aphasias [1-3].

In general, therefore, aphasia is caused by lesions of the areas of the brain primarily responsible for language processing (Broca area and Wernicke area) or other areas of connection with different brain centers variously involved in the function. These areas are generally located in the left hemisphere for right-handed subjects (in the rare cases in which they are located in the right hemisphere it is called “cross aphasia”). In left-handed people, in 60% of cases they are in the right hemisphere, while in the remaining 40% in the left hemisphere or both [3,4].

Some studies suggest that there is a lower incidence of aphasia in women because they may have more bilaterality of language function, although women have a higher incidence of Wernicke’s aphasia than men [4]. Common age can be considered an important factor in recovery: some studies

suggest that recovery from stroke aphasia has failed in patients over seventy years of age compared to younger patients; however, at any age, various degrees of recovery can occur even in remote times from brain injury. [5,6].

However, remaining on the etiological profile, it is important to underline that any brain disorder is capable of causing aphasia, as long as it affects the dominant hemisphere and the structures responsible for language processing. In this case we will speak of *acquired aphasia*. Among the most frequent causes, which can generate a temporary or permanent aphasic status, we find: [1,4,7-9].

- a) Cerebral infarcts (strokes) usually concerning softening of the territories of the Sylvian artery, of the left anterior cerebral artery or the posterior cerebral artery;
- b) Transient ischemic attack
- c) Cerebral hemorrhages: large central nucleus hemorrhages, lobar hematomas, but also small thalamic hemorrhages;
- d) Left, frontal or temporal hemispheric expansive processes (usually tumors) (mainly causing progressive aphasia);

- e) Degenerative processes (brain atrophies) characterized by progressive mental deterioration (in which speech disorders represent only part of the symptoms).
- f) Head injuries, which are responsible for intracranial hematomas especially in the left temporal lobe, brain contusions and post-traumatic arterial thrombosis (internal carotid artery and Sylvian artery);
- g) Infectious processes responsible for a brain abscess or encephalitis;
- h) Partial or complex epileptic crisis;
- i) Attack of migraine with aura, in its initial phase (within the first thirty minutes before the onset of migraine).

Aphasia in itself, as a pathological state, in its clinical sense may be able to negatively affect other psychological conditions, previous or concomitant, or feed the development of new psychopathologies, more or less acute in the manifestations, as happens in anxious states [10] and depressants [11], in post-traumatic stress events [12] caused by serious psychological trauma [13], in panic attacks [14], in sleep disturbances on a psychosomatic basis [15], in obsessive disorders- compulsive [16] and psychopathological personality disorders in general. Aphasia is also one of the causes that increases suicidal risk [17], especially concerning the subjective value of language for the patient.

The clinical alterations of cognitive functions related to language that fall within the term of aphasia include can concern both the understanding and the production, both the repetition and the structuring itself of the language, based on the brain areas affected by structural and functional damage. The aphasic phenomenon can manifest itself in various ways. Therefore, the ability to recognize a word or to choose the right word may be lacking. A word can be replaced with another of a different meaning but from the same family, or a wrong word with a sound similar to the right one or a completely different word and without any apparent link with the correct one can be used; furthermore, the disturbance can involve only speech, the ability to repeat a sentence, the structuring of a meaningful speech, or even the ability to write. In clinical observation, in particular, it has been observed on many individuals affected by this pathology that the forgetting of words follows a very precise order, without exception: the first words to be forgotten are proper names, then common names, follow the adjectives and finally verbs and prepositions [4].

Modern neuropsychology bases the study of aphasia on the system of components of the language elaborated by psycholinguistics, identifying "specific deficits" about three levels of elaboration [18,19].

Phonological level: Evaluates the production of the fundamental units of words (phonemes). In verbal production, phonetic and phonemic level disturbances are therefore observed. The former is studied using instruments that detect the movements of the individual articulatory organs and the VOT (Voice Onset Time, or the interval of time between the

opening of the phonoarticulatory duct and the beginning of the vibration of the vocal cords), and mainly consist of distorted sounds, variations that do not exist in the patient's language and increased pronunciation times (visible more for consonants than for vowels); the latter are disturbances in the conversion of the sounds of words (which maintain a correct representation in the patient's mind) into forms appropriate for the articulation and manifest themselves with phonemic paraphasias, i.e. replacement, omission, repetition or addition of phonemes within the words, which in serious cases become unrecognizable, giving rise to neologisms and neologistic jargon (if not even a word is understood) or phonemic jargon (in case a few words are still understood).

Lexical-semantic level: Concerns knowledge related to the meaning of words, thus implying semantic memory. In verbal production there are naming and anomaly deficits. The first is the inability to produce the correct name of an object or situation upon request; the second concerns the inability to spontaneously produce a word during the speech, furthermore lacking any implicit knowledge about the word, such as the initial, the number of syllables, the position of the accent.

Syntactic-grammatical level: Studies the rules for the formation of sentences, and the rules for the selection of words and sounds that have a grammatical function (free grammatical morphemes and grammatical morphemes linked to the root). Among the syndromes concerning verbal production at this level, there are agrammatism and paragrammatism: in the first one there are mainly omissions of function words and substitution of bound morphemes and is generally associated with Broca's aphasia, therefore with a language not fluent, with the simplification of syntactic structures, reduction of the length of sentences and difficulty in producing the correct order of words in the sentence; Paragrammatism has been studied much less than agrammatism, and some authors even question its specificity, since it would be characterized by the substitution of bound morphemes (a feature that has recently been associated with agrammatism). In extreme synthesis however we could say that in the agrammatics there are errors for omission, while in the nosing errors and additions for substitution.

The traditional classification of aphasias has generated several problems and disputes. It is not always possible to position a subject in one of the traditional categories; moreover, patients classified in the same group can differ, even a lot, in the quality of the mistakes made. Finally, in several cases, the patient's pathology evolves and this no longer allows us to classify him in the same initial syndrome. For simplicity we can summarize them in the following forms: "Fluent aphasias" and "Not fluent aphasias". Fluent aphasics have a relatively productive speech, in fact they manage to generate twenty words per minute with sentences composed of 5-6 elements; they also have relatively normal prosody and intonation of the phrase. They generally don't seem to realize their deficits. Their language is characterized by the contemporary presence of appropriate words and words without connection; the sentences, often long, do not follow the rules of syntax and are made inaccurate by the frequent use of periphrases; however,

in severe cases, fluent aphasic can only produce meaningless words, generating an empty language. Fluent aphasias are caused by temporal-parietal lesions of the left hemisphere. Not fluent aphasics are characterized by a scarce spontaneous verbal production, they can produce only isolated words or very short sentences composed of two / three elements, reducing the expression in some cases to a stereotype or a verbal formula; they also have a strongly slowed down and abnormal prosody and intonation of the phrase. They use very simple syntactic structures: they use a few verbs, sometimes not even conjugated, and they use a telegraphic style, that is, they do not use particles as articles, prepositions, and pronouns. Often, noticing the difficulties in communication, patients become discouraged and give up speech or compensate for needs with non-verbal language. Not fluent aphasias are caused by frontal lesions of the left hemisphere [3].

There are several possible aphasia that can be found:

1) “*Global aphasia*”. It is a serious deficit in the production, understanding, and processing of linguistic messages: speech is limited to recurring syllabic fragments, comprehension, and repetition are seriously altered, reading aloud and writing is practically absent, the understanding of written words is only possible for frequently used words. It is therefore a non-fluent aphasia, generally caused by large lesions of the left hemisphere involving the pre and post-rolandic perisylvian cortex and the underlying deep structures. Some patients seem to realize their difficulties and react in two opposite ways: with expressions of despair, or they completely lose the ability to communicate.

2) “*Broca’s aphasia*”. It is a non-fluent aphasia and is caused by cortical lesions of the Broca area and a part of the territory of the Sylvian artery, but also by subcortical lesions affecting the putamen or the internal capsule. Patients with this disorder show problems of agrammatism, speech is not fluent, there are no function words (articles and prepositions) and morphology, it is void of intonation, has phonemic and phonemic paraphasias. At the level of language comprehension, there are disturbances at the syntactic-grammatical level (such as difficulty in recognizing a semantically reversible passive construction sentence (“the child chases the dog” and “the dog is chased by the child”) and at the phonological level (as difficulty distinguishing similar phonemes, /p/e/b/o/d/e/t/); even the repetition of sentences is compromised. In general, however, understanding is less damaged than production. The patient with Broca’s aphasia has the awareness of his situation and it is not uncommon for him to burst into tears easily feeling frustrated and depressed. In general, after a few weeks, aphasia can regress but in some cases this language can remain agrammatic or it remains a spoken with a foreign accent as it violates some phonetic laws typical of the original language (in fact, the possible similarity with some languages is completely random). This phenomenon is called accent syndrome foreigner [20], it is a rare event (just over twenty cases in the world) and has been known since 1919. In a third of these cases, the problem resolves within a year; in others, however, it remains for life.

3) “*Wernicke’s aphasia*” (or “*receptive aphasia*”). It is caused by a cortical lesion of the Wernicke area, the associative auditory cortex, and the inferior parietal lobule. Wernicke’s aphasia poses problems both in understanding language and in production. The ability to process a speech fluently is maintained; the speech is paraphrastic and rich in circumlocutions with neologisms. The patient does not realize that his language is incomprehensible and can appear choleric and paranoid. The only understanding preserved is when he is ordered movements that use axial musculature (getting up, closing his eyes, turning around) but he doesn’t understand the question “what’s your name?”.

4) “*Transcortical aphasia*”. It is a particular type of aphasia characterized by a relative saving of repetition. It is divided into three subgroups:

- a) The *sensory* type, in which all the skills of understanding, processing, and production of language are severely compromised, except for repetition. The lesions of this aphasic form are located in the areas adjacent to the Wernicke area;
- b) The *motor* type, in which the competence in the production of language is severely compromised, but with a relatively conserved understanding and elaboration. The lesion is located in the frontal cortex.
- c) The *mixed* type, particularly severe non-flowing form. Patients present completely incomprehensible language, while the only remaining skills are those of repetition and automatic language (songs, prayers, etc.).

5) “*Conduction aphasia*”. It is a fluent aphasia characterized by a serious deficit in repetition, that is, in producing a stimulus on imitation, and by numerous phonemic paraphasias. It was hypothesized by Wernicke and described for the first time by Lichtheim in 1885. The lesion that causes this type of aphasia is at the level of the left parietal lobe (angular gyrus) and of the arched file.

6) “*Anomic aphasia*”. It is a particular type of fluent aphasia where patients have difficulty finding the exact terms to express themselves with, yet they manage to pronounce those same words on imitation. Understanding and skills for written language are generally less compromised.

7) “*Primary non-fluent progressive aphasia*” [21]. About dementias, atypical degenerative forms have been described since the nineteenth century, characterized by a non-amnesic focal onset but interesting in language, visual and visuospatial analysis, and face recognition. Pick (1892) and Serieux (1893) first described cases of subjects with progressive speech disorder associated with brain atrophy at the level of the frontal and temporal regions of the left hemisphere. In 1982, Marek-Marsel Mesulam described a series of six patients with a progressive reduction of verbal expressive abilities, coining the term “slowly progressive aphasia”. Snowden, in 1989, spoke of “semantic dementia”, then redefined in 2011 as “progressive non-fluent aphasia”. Primary progressive

aphasia (PPA) is a rare neurological syndrome characterized by polymorphic clinical aspects which however have language loss as a common element. It belongs to the picture of progressive focal cortical atrophies, in which the alteration of a cognitive function is found without dementia for at least two years. Linguistic competence can be deconstructed in its phonological, semantic, and syntactic levels, on the oral or written side; speech can be normal in fluency or non-fluent. Mesulam (2003) highlighted the difficulty in recognizing and diagnosing primary progressive aphasia, paying particular attention to the difficulties of differential diagnosis. In fact, in the first place there are multiple neurological diseases in which the loss of language can be present but included in a wide range of other cognitive deficits of an increasing nature (for example memory, attention, reasoning, apraxia-constructive, etc.). Furthermore, an aphasic picture can be progressive but not primary if inserted into a more complex syndrome. Finally, other clinical forms of a degenerative nature such as vascular dementia, in the Corps of Lewi, Creutzfeld-Jacob, and Alzheimer disease itself can manifest with speech disorders. In light of these claims, it follows that the differential diagnosis consists of and requires a long and complex long-term evaluation, supported by instrumental and laboratory accessory tests. Gorno-Tempini and colleagues, in 2011, provided a classification of primary progressive aphasia and its three main variants: “non-fluent-agrammatic”, “semantic” and “logopenic”.

8) “*Subcortical aphasia*”. Injuries of the left caudate nucleus and putamen determine transient fluent aphasia, characterized by the use of neologisms; thalamic lesions give transient aphasia similar to transcortical ones. In childhood and youth the most frequent causes of aphasia are traumatic, inflammatory and cancerous; in adulthood and old age vascular lesions prevail, with embolic or thrombotic occlusion of the middle cerebral artery (global aphasia) or its branches (selective aphasia). Transcortical aphasia electively appears in the infarcts known as the last meadows, due to pressure drops or systemic anorexia. The transient episodes of a. represent one of the manifestations of carotid TIAs.

9) “*Bilingual aphasia*” [22-25]. Language acquisition in multilingual subjects is conditioned by two factors: the age of language acquisition and competence. Specialization is centered in the Perisylvian cortex of the left hemisphere. Various regions both on the right and left hemisphere are activated during language production. Multilingual individuals constantly demonstrate similar activation patterns in the brain when using one or more languages they know fluently. Age of acquisition of the second or higher language, and competence of use determine which specific regions of the brain and pathways activate when using (thinking or speaking) the language. Unlike those who have acquired their different languages at different points in their lives, those who have acquired several languages when young, and practically at the same time, show similar activations in parts of the Broca area and left lower frontal lobe. If the second-or-higher language is acquired later in life, particularly after the critical period, the language becomes centralised in a different part of the Broca area than the mother tongue and other languages

learned when young. A higher density of grey matter in the lower parietal cortex is present in multilingual individuals. It has been found that multilingualism affects the structure, and essentially, the cytoarchitecture of the brain. Learning more languages re-structures the brain and some researchers argue that it increases the brain’s capacity for plasticity. Most of these differences in brain structures in multilinguals may be genetic at the core. Consensus is still confused; it may be a mixture of both experiential (language acquisition during life) and genetic (predisposition to brain plasticity) conditions. Aphasia in multilingual (or bilingual) is commonly assessed through a bilingual (or BAT) Aphasia test. The BAT consists of 3 sections that patients are required to respond with continuously while test administrators record their responses. patient performance is then documented and processed with computer programs that determine the percentages of correctness given the specific language proficiency. With the bat many clinical contexts have a standardised system to determine the extent of aphasia in multilingual patients. Work in the field of cognitive neuroscience has identified classical language areas within the perisylvian cortex of the left hemisphere. This area is crucial for the representation of language, but other areas of the brain are shown to be active in this function as well. Language-related activation occurs in the middle and lower temporal circumvolution, the temporal pole, the fusiform circumvolutions, lingula, in the middle prefrontal areas (i.e. dorsolateral prefrontal cortex), in the insula. It also seems to us to be of activation of the right hemisphere during most of the language tasks. Linked linguistic areas are dedicated, for some components of language processing (e.g. lexical semantics). These areas are functionally characterised by linguistically relevant systems, such as phonology, syntax and lexical semantics, and not speaking, reading, and listening. In the normal human brain, associated linguistic processing areas are less rigid than previously thought. For example, greater familiarity with a language has been found to lead to reductions in brain activation in the left dorsolateral frontal cortex (Brodmann’s areas , 9, 10, 46). Bilingualism implies the use of two languages by an individual or a community. Neuroimaging studies of gender bilingualism focus on the comparison between areas activated when using the first language (L1) and second language (L2). Studies of language production using functional neuroimaging methods, investigate the brain representation of bilingual language activities. These methods (e.g. PET and fMRI) subjects distinguished mainly on the basis of age of acquisition L2 and not on the level of competence in L2. With the use of PET in the study of end-student, cerebral blood flow (rCBF) regional distribution was found to be comparable between L1 and L2. Word repetition engages overlapping neural structures across both languages; that, differences in neural activation are observed only in the left putamen when individuals repeat words in their second language. The putamen, therefore, plays a fundamental role, because the process of articulation places a greater demand on the brain’s resources when a second language learned in old age is being produced.

Investigating an aphasic patient is not easy, as the disorder presents a variety of different symptoms on various



neurocognitive levels of speech. After a brain injury, the deficits, which are not always evident, can however be highlighted using specific tests. Often, even when it is thought that the initial aphasia has regressed (the patient has returned to speak normally), it is still possible to find serious disorders evident only in the most complex tests. For a correct examination, it is important (first of all) to ensure the patient's collaboration and to exclude all those disorders that can alter the language but that come from a global disorganization of brain functioning (as in the case of dementia or mental confusion) or it comes from the sensory systems (blindness, deafness), the motor systems (dysarthria, dysphonia, respiratory problems) and serious and chronic psychiatric disorders. In patients with Alzheimer's disease, aphasic symptoms are one of the first symptoms to appear, along with memory impairment [9,26].

It should also be remembered that aphasia can also occur concurrently with other neuropsychological disorders, related to language (*stuttering*), writing (*agraphy*), reading (*dyslexia* and *alexia*, understood as the loss of the cognitive abilities needed to read a text), calculus (*acalculia*); therefore, the evaluation of the different levels must be done in a structured way, in reference to the expression (spontaneous language, repetition of words and phrases of increasing length, production of automatic series, naming of images, shapes and objects, description of a complex image, repetition of a story) that to understanding (indicating objects and images, executing simple orders, executing complex orders), passing through reading (identification of letters, syllables, words, reading aloud, understanding language written, execution of written orders, correspondence of words written with images, of sentences written with actions), writing (spontaneous, copied, dictated) and elaborate tests (definition of words, idioms and proverbs construction of a sentence with two or three words given to the patient, critique of absurd stories, interpretation of a text heard or read). The most used and useful language assessment tests to follow the functional recovery of the patient are CADL (Communicative Abilities in Daily Life), Token Test, MAE (Multilingual Aphasia Examination), BDAE (Boston Diagnostic Aphasia Examination) and AAT (Aachener Aphasia Test).

The best diagnosis, therefore, cannot be separated from an anamnestic evaluation that takes into account the medical condition and the use of a battery of specific tests, such as the Aphasic deficit analysis (BADA), which allows more easily to arrive at a functional diagnosis and approach with correct rehabilitation (speech therapy treatment), also taking into account cognitive-behavioral or strategic psychotherapeutic profiles [27], in case of need.

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