http://dx.doi.org/10.23925/2176-2724.2019v31i1p160-169

Proposal for the classification of oral language disorders in the adult and elderly

Proposta de apresentação da classificação dos transtornos de linguagem oral no adulto e no idoso

Propuesta de presentación de la clasificación de los trastornos de lenguaje oral en el adulto y en el anciano

*Bárbara Costa Beber**

Abstract

Introduction: Adult and elderly individuals that were affected by lesion or dysfunction of the Central Nervous System may present language disorders as consequence. The definition of the language disorder type is of relevance to the clinical and scientific practices. The literature reports a variety of types of language disorders but does not present a classification system with as many types of language disorders as possible. **Objective**: To propose a comprehensive presentation of the classification of oral language disorders that can affect adults and elderly. **Methods:** Non-systematic literature review. The presentation of language disorders was made by grouping the different types of language disorders already described in the literature, but with an organized taxonomy that considered criteria such as linguistic manifestation and etiology **Results:** The types of oral language disorders that can occur in this population were classified into three categories: the aphasic language disorders, non-aphasic language disorders and cognitive-communication disorders. The category aphasic language disorder received its own sub-classification. **Conclusion:** This classification might help the communication among professionals, might allow the characterization of patients, and might facilitate the clinical reasoning. It is expected that the scientific advance in this field leads to changes in the classification systems, and also in the paradigm of establishing medical and speech-language diagnoses.

Keywords: Classification; Language disorders; Aphasia; Communication; Brain injuries

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Received: 30/01/2018
Accepted: 12/11/2018

Resumo

Introdução: Indivíduos adultos e idosos acometidos por lesões ou disfunções do Sistema Nervoso Central podem apresentar como consequência quadros diversos de transtornos de linguagem. Definir o tipo de transtorno de linguagem que ocorre em cada paciente tem grande relevância clínica e científica. A literatura descreve variados tipos de transtornos de linguagem oral, porém não apresenta um sistema de classificação com o maior número possível de tipos de transtornos de linguagem. **Objetivo**: Propor uma forma de apresentação abrangente da classificação dos transtornos de linguagem oral que podem ocorrer em adultos e idosos. **Método**: Revisão não sistemática de literatura. A apresentação dos transtornos de linguagem foi realizada a partir do agrupamento dos diversos tipos de transtornos de linguagem já descritos na literatura, mas com taxonomia organizada considerando critérios como a manifestação linguística e etiologia. **Resultados:** Os tipos de transtornos de linguagem oral que acometem a população em questão foram classificados em três grandes categorias, os transtornos de linguagem afásicos, os transtornos de linguagem não afásicos, e os transtornos cognitivos da comunicação. O grupo de transtornos de linguagem afásicos recebeu uma subclassificação própria. **Conclusão**: A utilização desta classificação poderá auxiliar a comunicação entre os profissionais, permitir a caracterização dos pacientes e facilitar o raciocínio clínico. É esperado que o avanço científico na área acarrete em mudanças na classificação destas condições e no próprio paradigma de estabelecimento dos diagnósticos médico e fonoaudiológico.

Palavras-chave: Classificação; Transtornos da linguagem; Afasia; Comunicação; Lesões encefálicas

Resumen

Introducción: Individuos adultos y ancianos acometidos por lesiones o disfunciones del Sistema Nervioso Central pueden presentar como consecuencia cuadros diversos de trastornos del lenguaje. Definir el tipo de trastorno de lenguaje que ocurre en cada paciente tiene gran relevancia clínica y científica. La literatura describe variados tipos de trastornos de lenguaje oral, pero no presenta un sistema de clasificación con el mayor número posible de tipos de trastornos de lenguaje. **Objetivo:** Proponer una forma de presentación completa de la clasificación de los trastornos de lenguaje oral que pueden ocurrir en adultos y ancianos. **Método:** Revisión no sistemática de literatura. La presentación de los trastornos del lenguaje fue realizada a través de la agrupación de los diversos tipos de trastornos de lenguaje ya descritos en la literatura, pero con taxonomía organizada considerando criterios como la manifestación lingüística y etiologia. **Resultados:** Los tipos de trastornos de lenguaje oral que afectan a la población en cuestión, se clasificaron en tres grandes categorías, los trastornos del lenguaje afásicos, los trastornos del lenguaje no afásicos, y los trastornos cognitivos de la comunicación. El grupo de trastornos de lenguaje afásicos recebió una subclasificación propia. **Conclusión**: La utilización de esta clasificación podrá auxiliar la comunicación entre los profesionales, permitir la caracterización de los pacientes y facilitar el raciocinio clínico. Se espera que el avance científico en el área acarrete en cambios en la clasificación de estas condiciones y en el propio paradigma de establecimiento de los diagnósticos médico y fonoaudiológico.

Palabras claves: Clasificación; Trastornos del linguaje; Afasia; Comunicación; Lesiones encefálicas

Introduction

The oral language communication disorders that occur in the adult and elderly population result from disruption in linguistic or cognitive domains that mediate the processing of language, due to injury or dysfunction of the Central Nervous System (CNS). However, it is not always possible to associate all symptomatic scenarios of language disorders with attestable neurological conditions. The knowledge of oral language disorders, their classification, symptoms and concepts are pivotal for the speech therapist to be able to evaluate, diagnose and rehabilitate the population affected by these health conditions.

The concepts and the classification of language disorders can vary according to the framework of thought of each author, and can change over the years according to the evolution of scientific evidence. The classification of classic aphasias, to give an example, is considered a "necessary evil" [1], because to plan the speech and language therapy the most important aspect is to know the nature of the linguistic deficit and not the "aphasia type". Nonetheless, the classification of aphasia in types and subtypes is a didactic way to guide the clinical reasoning of the language disorders and it facilitates communication between health professionals. With respect to the relevance of the research context, the symptom classification of such disorders allows associations to be made between the clinical presentation and the pathological findings. These associations are extremely necessary for advancing the understanding of the pathophysiology of neurological diseases that are still poorly understood, such as those with neurodegenerative causes. Consequently, such patients may be appropriately included in clinical trials to investigate the benefits of pharmacological and non-pharmacological therapies.

The earliest descriptions of language disorders come from the reports of the physicians and anatomists Paul Broca and Carl Wernicke.[2] Their findings paved the way for knowledge about brain neurobiology and language disorders. Currently, neuropsychology, which is an interdisciplinary science that studies the relationships between the brain and cognition, has greatly contributed to the advancement of knowledge on human language. Neuropsychology is also one of the specialties of the Speech and Language Therapy (SLT) profession, and it is essential for the clinical study and management of language disorders in adults and the elderly, since language disorders are the behavioral manifestation of CNS lesions or disturbances.[3] The growing advancement of the neuroimaging technologies has also allowed for a better understanding of the neural substrates of language disorders and consequently of the neurobiology of language.

Different types of oral language disorders are frequently described in didactic books or scientific articles. However, a classification model is necessary that presents the greatest possible number of language disorders types. To meet this need, the main objective of this non-systematic review is to propose a comprehensive presentation of the classification of all possible oral language disorders that may occur in adults and the elderly, in order to support the SLT clinical practice and the teaching of language disorders in the SLT undergraduation courses.

Method

The presentation of the oral language disorders in adult and in the elderly was organized from the grouping of the different types of language disorders previously described in the literature and based on the theoretical positioning of the author. The taxonomy of the three major categories of language disorders was organized according to the nature of the oral communication deficit, whether purely linguistic or cognitive. In the case of linguistic deficits, they were organized into aphasic or non-aphasic, depending on the impact of the linguistic deficit in communication. The other subcategories were taxonomised according to linguistic manifestations and their etiologies.

A non-systematic literature review was conducted to describe each type of language disorder, searching for scientific articles, books or documents that presented concepts and clinical characteristics. The search was made in scientific databases (Pubmed, Scielo, Lilacs, Google Scholar), in didactics books of the field, and in a free search on the internet. The keywords used in the search were not standardized, but some of the keywords used were "classification", "language disorder", "aphasia", and "communication disorder". No date or language of the publication was defined, and the criteria for inclusion was flexible, after considering the usefulness and relevance of each publication to describe each language disorder.

Results and discussion

Presentation of the classification of oral language disorders in the adult and elderly

The method of presentation of the classification that is proposed in this article covers types of oral language disorders that have already been described in the literature, but differentiates in the agglutination of those types in a single classification model. These disorders can be classified as: 1) aphasic – subdivided into classical aphasia, crossed

aphasia, subcortical aphasia and primary progressive aphasia (PPAs); 2) non-aphasic language disorders; and 3) cognitive communication disorders. Figure 1 shows the types of oral language disorders included in this model, which are described in detail below, according to the symptomatology, etiology and neuroanatomical correlates.

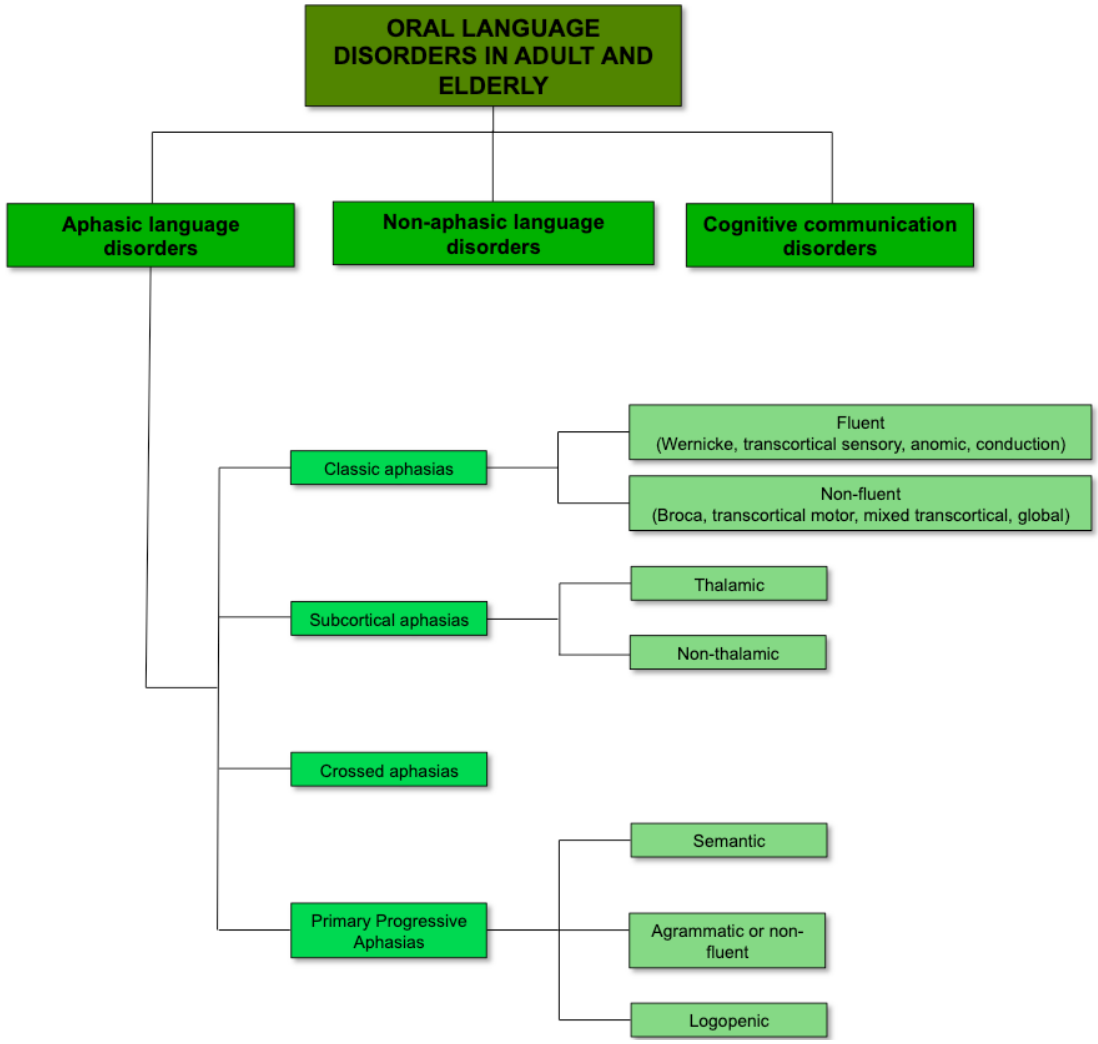

Figure 1. Classification of oral language disorders in the adult and elderly

The proper SLT diagnose of the disorders in question occur primarily through a comprehensive assessment of language and of other domains of cognition, history and medical diagnosis of the patient, and neuroimaging information. The language assessment should include investigation of speech fluency, repetition, oral comprehension, oral naming, grammatical aspects, reading and writing, pragmatics, prosody, and linguistic manifestations. The other cognitive domains that should be investigated are episodic memory, working memory, executive functions, attention, behavior, and visuospatial skills. Neural correlates are performed based on neuroimaging data, which can be obtained by computed tomography, magnetic resonance imaging, single photon emission tomography (SPECT), or positron emission tomography

(PET), depending on the neurological indication for each specific case.

It is important to note that due to recent literature recommendations, the terminologies "Broca´s area" and "Wernicke´s area" will be replaced in this paper by its respective neuroanatomical areas, posterior part of the left inferior frontal gyrus and left superior temporal gyrus.[4]

Aphasic language disorders

The aphasic language disorders are characterized by deficits in the emissive and/or comprehensive linguistic processes due to CNS lesions. They significantly limit the human communication and present with other cognitive functions relatively preserved.[5] Aphasic language disorders are subclassified here in classic aphasia, crossed aphasia, subcortical aphasias and PPA.

Classic aphasias

The main cause of classic aphasias is acquired brain injury, such as stroke (which is the most frequent cause), traumatic brain injury, tumors and brain infections.[7] Different subclassifications of classic aphasias can be found in literature, and the most frequently used is the one that classifies those syndromes in two major groups, the fluent aphasias (Wernicke, transcortical sensory, conduction and anomic) and the non-fluent aphasias (Broca, transcortical motor, mixed transcortical and global).[2,6] However, the same types of aphasia can be subclassified in perisilvian (Broca, global, Wernicke, conduction) and extraperisilvian aphasias (transcortical motor, transcortical sensory, transcortical mixed and anomic);[7] or in emissive (Broca, conductive, transcortical motor), receptive (Wernicke, transcortical sensory, anomic) and mixed aphasias (transcortical mixed, mixed, global).[8] The subclassification used in this article is derived from the studies of the Boston Aphasia School, and divides the subtypes into fluent and non-fluent aphasias.[2]

Figure 2 shows a didactic flowchart that helps the SLT to define the subtype of classical aphasia, based on the characteristics of the triad: fluency, repetition and comprehension of oral language. Other aspects of language are important for the differential diagnosis between subtypes. Thus, Table 1 describes in detail each subtype of classic aphasias.[2,6,9] It is important to note that in clinical practice many patients do not fit adequately in any of these subtypes. When this difficulty of classification occurs, it is suggested that the speech-language assessment should aim to evaluate the cognitive nature of language impairment in order to define the affected domains. Those affected domains will be the targets for rehabilitation, and the diagnosis may be kept as "non-specified aphasic language disorder."

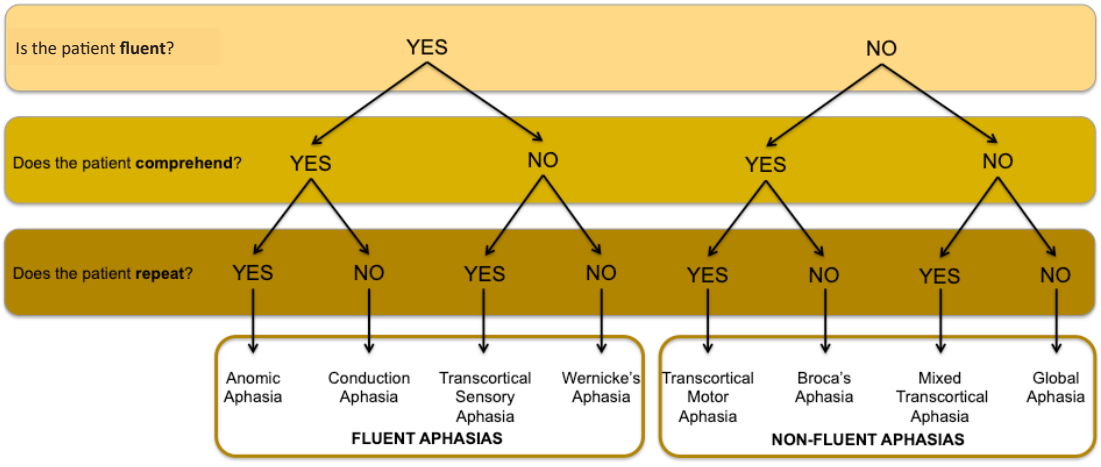

Figure 2. Flowchart for the classification of classical aphasias

Chart 1. Description of classical aphasias

	Triad fluency, repetition and comprehension	Other characteristics and linguistic manifestations	Neural substrates in left hemisphere
Fluent Aphasias			
Wernickes´s Aphasia	Fluency: preserved Repetition: impaired Comprehension: impaired	Jargon; logorrhea; anomia; neologisms; Circumlocutions; phonemic and formal paraphasias.	Superior temporal gyrus, middle temporal gyrus, inferior parietal lobe, angular gyrus, Heschl gyrus, temporal pole, putamen.
Conduction Aphasia	Fluency: preserved Repetition: impaired Comprehension: preserved	Phonemic and formal paraphasias.	Arcuate fasciculus (especially the posterior segment), cortical areas of the posterior perisylvian region (left supramarginal gyrus and adjacencies), Heschl gyrus.
Transcortical Sensory Aphasia	Fluency: preserved Repetition: preserved Comprehension: impaired	Verbal, phonemic and semantic paraphasias; anomias, echolalia, neologisms.	Proximities of the junction of the temporal, parietal and occipital lobes, middle part of the posterior temporal gyrus, inferior temporal gyrus, inferior angular gyrus.
Anomic Aphasia	Fluency: preserved Repetition: preserved Comprehension: preserved	Anomias, frequent pauses, semantic substitutions. Greater difficulty in certain semantic categories.	It is not reliably associated with a specific region. Anomias for nouns may be associated with lesions in temporal pole, middle and inferior temporal gyrus. Anomias for verbs are associated with lesions in the frontal lobe (inferior frontal gyrus and connections).
Nonfluent Aphasias			
Broca´s Aphasia	Fluency: impaired Repetition: impaired Comprehension: preserved	Slow and effortful speech, phonetic and phonemic paraphasias, anomias, verbal stereotypies, agrammatism, telegraphic speech, prosody changes. Difficulty understanding sentences with greater grammatical complexity, and greater difficulty in verbs than nouns.	Posterior portion of the inferior frontal gyrus, frontoparietal operculum, anterior portion of insula.
Transcortical Motor Aphasia	Fluency: impaired Repetition: preserved Comprehension: preserved	Reduction of spontaneous speech and speech initiative. Naming is better than spontaneous speech.	Lesions in the prefrontal cortex and adjacencies, with inferior frontal gyrus preserved.
Mixed Transcortical Aphasia	Fluency: impaired Repetition: preserved Comprehension: impaired	Emission severely impaired, anomias, echolalia.	Extensive cortical lesions that maintain the perisylvian cortex preserved.
Global Aphasia	Fluency: impaired Repetition: impaired Comprehension: impaired	Severe impairment of all language domains, slow and laborious speech, hesitations, mutism, severe anomia. Often, the improvement in the condition progresses to Broca's aphasia.	Extensive cortical lesions that compromise much of the perisylvian regions and their subcortical connections.

Subcortical aphasias

Subcortical aphasia can be described as changes in language emission and/ or comprehension resulting from lesions in subcortical areas of the brain, with cortical areas preserved. The clinical presentation is quite heterogeneous in terms of linguistic phenotype and severity. In addition, anatomic-clinical correlations are less consistent than in aphasias due to cortical lesions, and this is mainly due to imprecision regarding the function of subcortical structures in the language processing. Despite these inconsistencies, the subcortical aphasias seem to have as a common characteristic, the preserved repetition.[10]

The most commonly used classification for subcortical aphasias divides them into thalamic and non-thalamic.[6] In the former, as the name refers, lesions occur in the thalamus. The linguistic profile

depends on the combination of the injury site and the topography of the language within the innumerable pathways involved. The mechanism of brain diaschisis may play an important role in the clinical presentation of thalamic subcortical lesions.[6] Most studies on thalamic aphasia report selective impairments in lexical-semantic functions with preserved speech fluency. However, less frequent findings also point to the occurrence of phonetic paraphasias and some grammatical impairment.[6] In non-thalamic subcortical aphasias, the affected areas are the basal ganglia. The linguistic profile tends to be characterized by impairment in speech fluency, occurrence of literal paraphasias, and preserved comprehension and naming.[10] Mechanisms of hypoperfusion, ischemic damage, disconnection, and pressure effect may be associated with the clinical findings of this subtype of subcortical aphasia,[6,11] calling attention to the importance of a cautious interpretation of the anatomic-clinical correlations.

Crossed aphasia

More than 95% of right-handed individuals have the left hemisphere dominant for language. In this population, when brain lesions occur in the left hemisphere and damage brain areas that are involved in the processing of language, the manifestation of aphasia occurs. In the rest of the right-handed population, less than 5%, language processing occurs predominantly in the right hemisphere. In this group of people, lesions in the right hemisphere may lead to aphasia syndromes which are called crossed aphasia.[12] Thus, the definition of crossed aphasia can be summarized as being an aphasic language disorder in consequence of right hemisphere lesions in right-handed individuals;[12] or an aphasic disorder resulting from unilateral hemispheric brain injury ipsilateral to the individual's manual dominance.[13]

Crossed aphasia accounts for less than 4% of aphasic disorders and approximately 70% of cases manifest as a "mirrored" version of classic aphasias.[5]

Primary Progressive Aphasias

PPA is a group of neurological diseases identified by selective language impairment due to a neurodegenerative process in frontal and temporal brain areas that are involved in language processing.[14] The knowledge about this progressive and fatal disease is relatively recent and to date there is no pharmacological treatment to cure or change the course of the disease.[15] Nonetheless, there is evidence that non-pharmacological treatments such as SLT may delay the development of language disorder and improve the quality of life of these patients.[16,17]

Arnold Pick made the first description of an aphasia case due to neurodegeneration in 1892. About a century later, Marsel Mesulam described a number of cases of progressive speech disorders, which he named PPA. For two decades, APPs were classified into fluent (or semantic dementia) and non-fluent subtypes.[18] Over the years, it was found that many cases did not meet the criteria for either subtype, and in 2004 Gorno-Tempini and his colleagues described a third subtype of PPA called logopenic aphasia. In 2011, the diagnostic criteria for the 3 variants of PPA were published and they are currently used today.[18] The 3 PPA variants are described below, according to the publication mentioned above.

Semantic variant: in this variant, spontaneous speech is fluent, but there is evident anomia and difficulty in understanding single words. In the initial phases, there are errors in naming that are more frequent in unfamiliar and low-frequency words, verbal and semantic paraphasias, circumlocutions, generalizations (eg speaking "animal" for any species of animal) and omissions may occur. This variant also presents with surface dyslexia and dysgraphia, which are, respectively the difficulty of reading and writing irregular words due to the impairment of the reading and writing lexical route. Other cognitive domains are preserved, such as episodic memory, praxis, visuospatial functions, and executive functions. This clinical scenario is due to atrophy in the temporal pole of the left and/or right hemisphere. Postmortem studies have found pathological findings indicating the accumulation of certain altered proteins in the brain. The most frequent pathology was the frontotemporal degeneration by tau protein and TDP protein, as well as pathological findings of Alzheimer's disease (accumulation of beta protein amyloid and tau protein neurofibrillary tangles).

Agrammatic or non-fluent variant: this is characterized mainly by non-fluent speech. There is the occurrence of apraxia of speech and agrammatism that may appear in isolation or not. Agrammatism presents with the emission of simple and short sen-

tences, morphological changes in verbs and nouns, and changes in the order of words. Apraxia of speech is characterized by slowed speech; abnormal prosody; substitutions, additions, repetitions and/or prolongations of speech sounds; and possible difficulty to initiate the emission with attempts and errors in the articulatory movements. The neural substrate of this variant is atrophy or hypoperfusion in the frontal or insular brain regions of the left hemisphere. The most frequent pathological finding is frontotemporal degeneration due to alteration in tau protein.

Logopenic variant: the logopenic variant has been described more recently and is characterized by difficulty in repeating sentences and finding words (called the "tip of the tongue" effect). Phonological errors may occur in speech, but there is no agrammatism or difficulty of oral comprehension. The classical neuroimaging findings in individuals with this variant of PPA are atrophy at the left temporoparietal junction. Pathological findings are mostly those of Alzheimer's disease pathology, i.e., tau protein neurofibrillary tangles and senile plaques due to the accumulation of beta-amyloid protein.

Non-aphasic language disorder

The non-aphasic language disorder is characterized by a normal or near to normal performance in the formal aphasia assessments, despite the occurrence of language deficits in higher order or high complexity language tasks.[19] In addition, it is possible to add to this definition subtle language difficulties, often perceived by the individual and by the evaluator, but that are difficult to detect in the evaluation.

The etiology may be diverse and may result from progressive or non-progressive cerebral disorders from left or right hemisphere lesions.[19,20] Such disorders are very common in cases of traumatic brain injuries, and many cases present with acute aphasia of which the condition may improve, remaining only with non-aphasic language disorder that still limits communication.[21] The most frequent language deficits in cases of traumatic brain injury occur in higher order language skills, such as discursive skills.[20] Non-aphasic disorders may occur in cases of Alzheimer's disease, manifesting mainly through lexical-semantic and oral comprehension deficits.[22,23]

It is possible that in cases of left hemisphere lesions, the lesions are located outside the main areas of language processing but interrupt connections with language networks, or affect these areas in small proportions.[19] In these cases, the basic functions of language (lexical-semantic processing, grammar, phonology) may be altered, but at a lesser level of severity that does not characterize an aphasic syndrome.

In cases of language disorders due to right hemisphere lesions, in individuals who do not have language dominance in this hemisphere, the deficits may occur in functions of pragmatics, prosody, lexical-semantic processing, and textual and discursive skills.[6,24] These patients may present with difficulties in producing and perceiving emotional and linguistic prosody, in performing tasks of semantic judgment, and in understanding implicit information and inferential content.[6]

Cognitive-communication disorder

The diagnosis of cognitive-communication disorder is established when communication difficulties (emission, comprehension, reading, writing or social interaction) emerge from deficits in non-linguistic cognitive functions which intermediate between the functioning of language, such as memory, attention, and executive functions. These deficits may occur as a result of any neurological or even psychiatric condition and are common in dementia, mild cognitive impairment, stroke, brain tumors, and traumatic brain injury.[25-27]

To clarify the concept of cognitive-communication disorder, it is possible to take as examples the deficits in the following cognitive domains and their possible consequences in communication: *1) episodic memory* - the individual does not remember that he has already made a certain question or affirmation and repeats it constantly, reducing the effectiveness of her/his communication; *2) attention* - when the individual is unable to maintain attention during the conversation and the loss of information occurs which may impair the comprehension of the message. In this case, the deficit is not in the function of oral comprehension, but in attention, which can cause confusion between the two; *3) inhibitory control* – it causes difficulties in respecting turn takings and can leads to inappropriate speeches for the social/communicational context in question.

Although Alzheimer's disease may present with non-aphasic language disorder, as described in the previous section, it may also present with cognitive-communication disorder.[25] Alzheimer's disease is mainly characterized by impairment in episodic memory. Because episodic memory is important for the organization of discourse, it has a direct impact on communication. The main characteristics of the cognitive-communication disorder in Alzheimer's disease are: oral comprehension difficulties due to inability to access the information of the conversation in the episodic memory, repetition of sentences and questions, and difficulty remembering the topic of the conversation. Such difficulties can lead to behavioral problems such as depression, irritability and even aggressiveness. Cognitive-communication disorder may also occur in cases of vascular dementia.[25] The clinical presentation of vascular dementia depends on the affected areas which may be diverse and difficult to specify. However, common to all, difficulties of attention and executive functions are frequent. The impact on communication is manifested by a reduction in speech fluency, difficulty in understanding because the person is unable to store the information heard in the working memory, and confused speech.

In the cases of traumatic brain injury, difficulties of episodic memory, attention, working memory, speed of information processing, executive functions, social cognition and self-awareness may occur.[28,29] Discursive difficulties may then emerge, presenting discourse with insufficient information, redundancy of information , failures to structure discourse, and inadequate utterances for specific situations.[30]

Final considerations

This article described a proposal for the presentation of oral language disorders that most frequently affect adults and the elderly, classifying them into three broad categories. The first category is composed of aphasic language disorders. These are characterized by changes in language processing as a consequence of CNS damage, which significantly limit communication but keep other cognitive functions intact. This category includes the classic aphasias, the subcortical aphasias, the crossed aphasias and the PPAs. The second category is the non-aphasic language disorders, which is established when there is normal or near-to-normal performance in aphasia assessments, with deficits in higher order or high complexity language tasks, or when there are subtle language difficulties. The third category, cognitive-communication disorders, is characterized by communication difficulties due to deficits in non-linguistic cognitive functions caused by neurological or psychiatric conditions.

The usage of this classification has the objective of facilitating communication among professionals, allowing the characterization of patients and facilitating clinical reasoning. However, it is known that many cases are not possible to be classified, because they present characteristics that are common to more than one type of disorder, or because they are atypical cases. The impossibility of classifying language disorders still allows for adequate therapeutic planning and its execution. In these situations, the results of the speech-language assessment should guide the patient's rehabilitation, independent from the speech-language diagnosis. The language and/or cognitive deficits may be the goal of rehabilitation, while the preserved functions may be used in therapy as facilitators.

It is expected that the scientific advance in the area of language disorders allow for better understanding of language dysfunctions, neurological diseases and of language neurobiology itself. Naturally, this scientific advance may lead to changes in the classification of these conditions and in the paradigm of establishing medical and speech-language diagnoses.

References

1. Damásio AR. Signs of aphasia. In: Sarno MT. Aquired aphasia. 3rd ed. San Diego: Academic Press; 1998. 687 p.

2. Kemmerer D. Cognitive neuroscience of language. New York: Psychology Press; 2015. 623 p.

3. Brandão L, Fonseca RP, Ortiz KZ, Azambuja D, de Salles JF, Navas AL, et al. Neuropsychology as a specialty in Speech Language and Hearing Sciences: Consensus of Brazilian Speech Language Pathologists and Audiologists. Distúrb Comun. 2016; 28: 378–87.

4. Tremblay P, Dick AS. Broca and Wernicke are dead, or moving past the classic model of language neurobiology. Brain Lang. 2016; 162: 60–71.

5. Davis GA. Aphasiology: Disorders and Clinical Practice. 2nd ed. Boston: Pearson; 2007.

6. Stemmer B, Whitaker HA. Handbook of neuroscience of language. London: Elsevier; 2008. 490 p.

7. Benson DF, Ardilla A. Aphasia: A Clinical Perspective. Oxford University Press; 1996. 441 p.

8. Ortiz KZ. Distúrbios Neurológicos Adquiridos Linguagem e Cognição. Manole; 2010. 484 p.

9. Yourganov G, Smith KG, Fridriksson J, Rorden C. Predicting aphasia type from brain damage measured with structural MRI. Cortex. 2015; 73: 203–15.

10. Kuljic-Obradovic DC. Subcortical aphasia: three different language disorder syndromes? Eur J Neurol. 2003; 10: 445–8.

11. Hillis AE, Barker PB, Wityk RJ, Aldrich EM, Restrepo L, Breese EL, et al. Variability in subcortical aphasia is due to variable sites of cortical hypoperfusion. Brain Lang. 2004; 89: 524–30.

12. Kim WJ, Yang EJ, Paik N-J. Neural Substrate Responsible for Crossed Aphasia. J Korean Med Sci. 2013; 28: 1529.

13. Mastronardi L, Ferrante L, Maleci A, Puzzilli F, Lunardi P, Schettini G. Crossed aphasia. An update. Neurosurg Rev. 1994; 17: 299–304.

14. Mesulam MM. Primary progressive aphasia. Ann Neurol. 2001; 49: 425–32.

15. Tsai RM, Boxer AL. Treatment of frontotemporal dementia. Curr Treat Options Neurol. 2014; 16: 319.

16. Cadório I, Lousada M, Martins P, Figueiredo D. Generalization and maintenance of treatment gains in primary progressive aphasia (PPA): a systematic review: Treatment gains in PPA: a systematic review. Int J Lang Commun Disord. 2017; 52: 543–60.

17. Tippett DC, Hillis AE, Tsapkini K. Treatment of Primary Progressive Aphasia. Curr Treat Options Neurol [Internet]. 2015 [cited 2018 Jan 6];17. Available from: http://link.springer.com/10.1007/s11940-015-0362-5

18. Gorno-Tempini ML, Hillis AE, Weintraub S, Kertesz A, Mendez M, Cappa SF, et al. Classification of primary progressive aphasia and its variants. Neurology. 2011; 76: 1006–14.

19. McDonald S. Viewing the brain sideways? Frontal versus right hemisphere explanations of non-aphasic language disorders. Aphasiology. 1993; 7: 535–49.

20. Vas AK, Chapman SB, Cook LG. Language impairments in traumatic brain injury. In: Handbook of Clinical Neurology [Internet]. Elsevier; 2015 [cited 2018 Jan 6]. p. 497–510. Available from: http://linkinghub.elsevier.com/retrieve/pii/B9780444635211000315

21. Prigatano GP, Roueche JR, Fordyce DJ. Nonaphasic language disturbances after closed head injury. Lang Sci. 1985; 7: 217–29.

22. Klimova B, Maresova P, Valis M, Hort J, Kuca K. Alzheimer's disease and language impairments: social intervention and medical treatment. Clin Interv Aging. 2015; 10: 1401–8.

23. Verma M, Howard RJ. Semantic memory and language dysfunction in early Alzheimer's disease: a review. Int J Geriatr Psychiatry. 2012; 27: 1209–17.

24. Cummings L. Pragmatics and Adult Language Disorders: Past Achievements and Future Directions. Semin Speech Lang. 2007; 28: 096–110.

25. Bayles KA, Tomoeda CK. Cognitive-communication disorders of dementia. Definition, diagnosis, and treatment. San Diego: Plural Publisching Inc.; 2014. 271 p.

26. Hewetson R, Cornwell P, Shum D. Social participation following right hemisphere stroke: influence of a cognitive-communication disorder. Aphasiology. 2018; 32: 164–82.

27. Togher L, Wiseman-Hakes C, Douglas J, Stergiou-Kita M, Ponsford J, Teasell R, et al. INCOG Recommendations for Management of Cognition Following Traumatic Brain Injury, Part IV: Cognitive Communication. J Head Trauma Rehabil. 2014; 29: 353–68.

28. Gardner RC, Langa KM, Yaffe K. Subjective and objective cognitive function among older adults with a history of traumatic brain injury: A population-based cohort study. Brayne C, editor. PLOS Med. 2017; 14: e1002246.

29. Azouvi P, Arnould A, Dromer E, Vallat-Azouvi C. Neuropsychology of traumatic brain injury: An expert overview. Rev Neurol (Paris). 2017; 173: 461–72.

30. Snow P, Douglas J, Ponsford J. Conversational assessment following traumatic brain injury: a comparison across two control groups. Brain Inj. 1997; 11: 409–29.