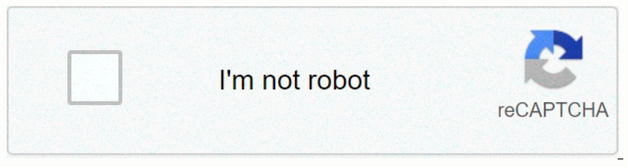

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Aphasia articles pdf

Aphasia is a violation of production and/or understanding of language caused by selective damage in specific areas of the brain, usually located in the left hemisphere (which is dominant to the tongue in the right and left hand of the people). From: Neurobiology of Brain Disorders, 2015H.S. Kirchner, in the Encyclopedia of Human Behavior (Second Edition), 2012Aphasia was classically described in connection with focal brain lesions. The most common such lesion is stroke, which has long led the etiology of aphasia in studies. Stroke can be seen as a nature experiment in which one area of the brain is damaged while the rest remains intact. Both major types of stroke, brain infarctions in which the brain is damaged by reduced blood flow, and hemorrhage, or bleeding in the brain, cause aphasia. Approximately 750,000 strokes occur annually in the United States. About 20% of stroke patients, or more than 100,000 people each year, develop aphasia after a stroke. The total prevalence of Afasic stroke in the United States is approaching 1 million. Traumatic traumatic brain injury often disrupts discourse and produces other language disorders that are often part of general cognitive dysfunction syndrome. Penetrating brain damage causes focal areas of damage that resemble strokes, except that the lesion sites are different, and the removed effects of swelling, increased intracranial pressure, and contrecoup injuries are more with traumatic brain injury than with stroke. Brain tumors, abscesses and other massive lesions can also disrupt the tongue. These syndromes usually develop gradually, within weeks or months, as opposed to the sudden onset of aphasia secondary stroke or head trauma. As with head injuries, there are long-term effects associated with mass effects and swelling, in addition to focal damage. For this reason, these disorders were less beneficial than stroke in studies of aphasia. Infectious brain damage from encephalitis and other infections also causes aphasia. Herpes simplex virus causes encephalitis, with an addiction to orbital frontal lobes and temporal lobes. Patients can pose with free aphasia as well as confusion, memory loss, seizures, and fever. Patients undergoing surgery to remove epilepsy also provided new knowledge of the language regions of the brain, as previously discussed. In addition to language disorders associated with focal brain lesions, generalized brain diseases produce language disorders. Aphasia accompanies diffuse syndromes such as encephalopathy, acute confusion, dementia and chronic memory syndromes and cognitive loss. These syndromes will be discussed after focal aphasia. Martha Taylor Sarno, in the Encyclopedia of the Human Brain, 2002 When aphasia persists for several months, full recovery to premorbid level of communication Unlikely. The temporal flow of aphasia from its sudden onset, acute stage and subsequent stages of recovery in separate ways, depending on whether a person is referring to a person's communication skills, psychological state, social functioning, compensatory skill level or level of adaptation. In the period immediately after the beginning, a degree of natural recovery called spontaneous recovery occurs in most people. There is a lack of confidence in how long this period lasts, ranging from 2 to 6 months after the start. It is generally accepted that, with some exceptions, significant improvements in communication occur in the first 12-24 months after the beginning. However, over the years since, there has been evidence of gradual changes, particularly in the area of compensatory, alternative skills, which are used more effectively over time. Age, gender, education and hands do not appear to affect recovery. Understanding tends to be restored more than expressive communication. Some people regain many communication skills but are left with an anomaly. Several studies report that for more severe aphasia, the recovery of communication skills begins later than for mild to moderate impairment.D. Kaplan, in the International Encyclopedia of Social and Behavioral Sciences, 2001The term aphasia refers to language disorders following brain disease. As in other articles in this encyclopedia the language is distinctly human system of symbols, which refers to a number of different types of forms (words, words formed from other words, sentences, discourse, etc.) to various aspects of meaning (objects, properties of objects, actions, events, causes of events, temporary order of events, etc.). Language forms and related meanings are activated in the processes of speaking, understanding speech, reading and writing. The processes by which these forms are activated are largely unconscious, obligatory after the beginning, quickly and usually quite accurate. Violations of language code forms and their connection to related values, as well as the processes that activate these views in these common language usage tasks, constitute afasic violations. By convention, the term aphasia does not refer to violations that affect the functions on which language processing is put. Lying (even a transparent, ineffective lie) is not considered a form of aphasia, nor is the garrulousness of old age or the inconsistency of schizophrenia. Language consists of a complex system of representations, and its processing is no less complex, as described in other records in this encyclopedia. Simply presenting minimally linguistically relevant elements of sound - phonemes - and processing involved in the recognition and production of these units is a very complex area of operation. When all levels of language and interaction are considered, language processing is considered extremely complex. It is therefore to be expected that the Afas violations will be just as complex. Researchers are slow a very significant range of these disorders. Nina F. Dronkers, Jennifer Ogar, in the Encyclopedia of Neurological Sciences, 2003Textbook descriptions of aphasia localize Brock's deficiency to lesions in the Brock area and those of Wernicke aphasia in the Wernicke area, as envisioned by the Wernicke-Geschwind model. Recent work with modern neuroimaging techniques and more complex behavioral testing has changed this model of localization. For example, lesions only in the Brock area are known to produce only transitional mutism, which is allowed in 3-6 weeks. Much greater defeat is needed to produce symptoms that lead to Brock's lingering aphasia. Brock's chronic aphasia occurs as a result of lesions, which usually covers the Brock area, as well as the adjacent frontal areas underlying the white matter, in zero, the anterior upper temporal gyrus, and the arquaat/upper longitudinal fascicle in more severe cases. Similarly, the site of the defeat associated with Wernicke's aphasia was also controversial. Recent studies have shown that the persistence of Wernicke aphasia is not due to lesions in the Wernicke area, but from the near-total destruction of the middle temporal gyrus and the significant involvement of the underlying white matter. Patients with such lesions have a poor prognosis for recovery. Defeats limited to the Wernicke area are usually resolved by a milder form of aphasia, usually aphasia holding. Aphasia Of Acute Wernicke, whose lesions spare the upper and middle temporal weights, as well as the underlying white substance, are usually brought into abnormal aphasia with a tendency to semantic paraphase error. Attempts to localize aphasia syndromes have probably failed because these syndromes are collections of many individual language deficits or symptoms. Brock's aphasia, for example, syndrome with symptoms such as speech apraxia, deficiency in word search, relatively spared auditory understanding, and poor repetition skills. Instead of localizing whole syndromes, it was more fruitful to study the relationship of lesions with individual symptoms that exist in different types of aphasia. For example, any lesion that involves an excellent portion of the precentral gyrus isula will result in speech apraxia, a planning articulation disorder. Patients with such lesions have difficulty in coordinating the articulation of movements so that the tongue, lips and larynx make the correct movement at the appropriate time. Speech apraxia may be present in isolation or in combination with Brocky aphasia, abnormal aphasia and aphasia. Regardless of the type of aphasia, defeat always includes this part of isula. Whether the apraxia speech is part of the deficit depends on whether this area has been injured. Similarly, the site of the defeat can predict other specific deficits. Patients with problems in repeating low-frequency words and phrases tend to have damage involving the posterior upper and, in more severe cases, also the lower parietal lobula. Because the most obvious deficiency in aphasia pursued is the recurrence of the disorder, it is not surprising that all of these patients have lesions involving these two regions. However, some patients with Brock aphasia whose lesions include these regions also demonstrate the same pattern of recurrence of deficiency in addition to their other deficits in fluency, auditory understanding and naming. In general, larger lesions tend to cover some of these critical areas and therefore lead to more extensive upset. Although anomaly is common in all types of aphasia, lesions in different areas of the brain lead to different patterns of symptoms. Defeats of dorsolal frontal areas can lead to difficulties in obtaining words from the lexicon, as evidenced by the opposition of naming tasks, as well as in free conversation. Recognize the correct word when it is provided, instantly. This pattern is observed with chronic abnormal aphasia with frontal lesions, as well as acute transcortic motor aphasic patients, which are also commonly solved in abnormal aphasia. Frontal abnormal patients are likely to improve their naming skills relatively quickly, suggesting that the frontal areas of the brain may help in finding words, but not in lexical storage per se. On the other hand, rear damage, especially involving the middle temporal gyrus and the underlying white matter, tends to cause the wrong name of objects with less chance of recognizing the correct name when proposed. These patients have a more central deficiency in the language system than their frontal counterparts. Their deficit is severe and persists and usually leads to the classification of Wernicke or, if the frontal areas are also involved, global aphasia. When the lesion is small and involves only part of the middle temporal gyrus, transcortic sensory aphasia can result, with temporary problems of auditory understanding and intact repetition skills. However, as problems are resolved with understanding, the classification changes to abnormal aphasia. The lesions leading to symptoms of conduction aphasia are thought to be associated with arquaat fascicle. It is now known that damage to this fiber optic tract can lead to a much more serious production deficit. It appears that information processed in the posterior areas cannot pass through the motor speech mechanisms in the frontal lobe. When the fasciculus arc is struck, the result is repetitive repetitive words or phrases with little or no productive product. When all of the aforementioned areas of speech and language are damaged, global aphasia results. This is usually due to a large middle cerebral artery heart attack that affects most of the left hemisphere. Areas of Brocky and Wernicke, Insula, Arkuat Fascicle, frontal temporal gyrus, posterior temporal gyrus and lower dark lobe, middle temporal gyrus and underlying temporal temporal the question is usually all participants. Somatosensor and auditory cortices may also be affected. When some of the previously mentioned areas are spared, an atypical picture of performance can result, with the preservation of certain speeches or language functions. In general, all patients of aphasia will present with a deeper language deficit acutely, which, given the time, solved in a less severe form of aphasia. Knowing the relationship between the site of the lesion and the specific behavioral deficit can help in more accurate predictions. It is also important to note that although the most dramatic recovery occurs in the first year, Atasic patients continue to make language benefits well beyond the first 12 months. Age, etiology, general health, motivation and other patient variables are also predictive indicators. Elizabeth E. Galletta PhD,... Peggy S. Conner PhD, in Stroke Rehabilitation, 2019Aphasia is an acquired language disorder secondary stroke or other acquired traumatic brain injury. Most people with aphasia present with some degree of oral language disturbance, which includes expressive and/or auditory lack of understanding. A distinctive feature of aphasia is the lack of word search. There are different behavioral approaches to language intervention to rehabilitate aphasia. Aphasia treatments can be widely classified as restitutive violations of oriented or substitution-compensation-oriented approaches. While reshitutive therapy is a mechanistic approach that focuses directly on linguistic function, the methods of rehabilitation of substitution aphasia are aimed at compensating for the lack of language aphasia and include functionally oriented treatments. The restoration of the language of aphasia varies depending on the person. Most people who acquire aphasia live with disorders and never recover their pre-stroke abilities. However, most experience constant improvement in their language and communication abilities, especially after intervention. This chapter provides information on the classification of aphasia and type, and discusses existing approaches to treatment of aphasia rehabilitation. Examples of behavioral interventions based on impaired treatment approaches in the oral language, approaches to reading and writing treatment, and approaches to treatment in the field of social communication are described. Pharmacological interventions and non-invasive methods of brain stimulation to rehabilitate aphasia are also generalized. Finally, we comment on the use of behavioral interventions to rehabilitate aphasia for multilingual and multicultural individuals with aphasia.N.F. Dronkers, J.V. Baldo, in the Encyclopedia of Neurology, 2009Aphasia is a violation of speech and language after brain injury. This is usually the result of defeats in the Peri-Sylvia region of the left hemisphere. Depending on the affected areas of the brain, the structure of speech and language deficit varies. Aphasia classifications include: aphasia Brockie, Wernicke Wernicke aphasia, global aphasia, abnormal aphasia and transcortic aphasia. These types of aphasia differ in the pattern of lack of fluency, understanding, naming and repetition. This article examines the clinical, cognitive-linguistic and neural correlates of these aphasia syndromes. Hal H. Nguyen, Stephen K. Kramer, in The Neuroscience of Language, 2016Aphasia is a consequence of brain injury. Many different forms of pathology can produce aphasia, including ischemia, degenerative diseases, infections and injuries, with the most common cause of ischemic stroke; about 1 in 4 stroke patients have some degree of aphasia at acute presentation. The natural course of aphasia varies depending on the pathogenesis. When the cause is ischemic stroke, there is often some degree of spontaneous behavioral recovery. However, this is generally incomplete and aphasia remains an important source of disability. This chapter explores the potential for stem cell therapy to improve deficiency and disability from aphasia. M Mr. Gorahl, ... M.L. Albert, in the Encyclopedia of Gerontology (Second Edition), 2007Aphasia (see Language Disorders: Aphasia) refers to the language of specific deficits resulting from brain damage, usually in the left hemisphere of the brain. In most cases, not all language skills are equally affected. Several types of afasic syndromes have been identified. The classification of aphasia has traditionally been based on the type of observed language deficiency, as well as on the location of brain damage. While the link between the type of deficiency and the place of defeat has been identified, these relationships are complex and are still being discussed. Disturbance of the language in aphasia can affect predominantly the production of the language or understanding of the language, one of the conditions (e.g. spoken language) or all conditions, and can be mild or severe. Most physicians and researchers in speech language today distinguish between two main categories of language disorders. Free aphasia is usually characterized by a cursory but empty (senseless) speech, problems of word search, speech errors, semantic deficit and impaired understanding. Nonfree aphasia is characterized by light speech and language production and relatively untouched understanding. In most cases, atasic disorder is not progressive, and time, practice and treatment lead to a gradual improvement of language skills. Several epidemiological studies of aphasia in adulthood show that some forms of aphasia are associated with certain age ranges. In particular, nonfree aphasia is associated with a younger age range than running aphasia. In one study, for example, non-free aphasia individuals were between the ages of 30 and 60 with an average age of 52, while those with free aphasia tended to be older, with ages 40 to 80 and an average age of 63. This is may be associated with brain changes associated with aging. Attitude Attitude the age of onset of aphasia and the degree of recovery from aphasia, however, is less consistent. The hypothesis that older people with aphasia are less able to regain their language skills than young adults has not received unequivocal research support. Martha Taylor Sarno, in Acquired Aphasia (Third Edition), 1998Aphasia Rehabilitation has been in such a state of flow since the publication of the second edition of acquired aphasia that it is difficult to describe his current state with precision and confidence. Dramatic changes in the provision of health services have led to a sharp decline in the availability of aphasia rehabilitation for all individuals except those who can support treatment costs. If there is a refund to third parties, its frequency, intensity and duration are significantly limited. As a result, the practice of aphasia therapy has shifted to address the problem associated with this modified health care system. In addition, new categories of patients with communication disorders are looking for services such as primary progressive aphasia. This chapter outlines the approaches and trends that have historically characterized the treatment of aphasia, recovery factors,

research and the philosophy of aphasia rehabilitation. A detailed overview of many of the methods used in the rehabilitation of aphasia is not possible in this volume, but can be found in a wide range of textbooks and journal articles. David Myland Kaufman MD, ... Mark J. Milstein MD, in Kaufman Clinical Neurology for Psychiatrists (Eighth Edition), 2017While aphasia is not equivalent to dementia, it can mimic dementia. For example, when aphasia impairs routine communication - by telling a date and place, repeating a series of numbers, and following requests - it mimics dementia. At times, patients with aphasia seem so strange that they seem incoherent. Because people think in words, aphasia is also a cloud of cognition and memory. Dementia and aphasia also vary in time. Dementia develops slowly, but aphasia starts suddenly, except in rare cases when it foreshadows a neurodegenerative disease. Nonfluent aphasia is even more different from dementia by the accompanying physical aspects: disarthria and obvious lateral signs such as right-sided hemiparesis and homonym hemianopia. In addition, paraphasia is often found in free aphasia, but rarely with dementia. However, patients sometimes have both aphasia and dementia. This combination occurs with one or more strokes inlaid on neurocognitive disorder due to Alzheimer's disease. It regularly develops in front of the lobar degeneration. Such situations are unclassified because aphasia usually cancels standard cognitive testing. The difference between aphasia and recognition when two conditions coexist more than academic exercise. Diagnosis of aphasia usually suggests that the patient had a discrete dominant hemisphere brain injury. Because stroke or or or structural lesions will be the most likely cause, the appropriate assessment will include COMputed tomography (CT) or magnetic resonance imaging (MRI). In contrast, a diagnosis of dementia suggests that the most likely cause will be a neurodegenerative disease and the evaluation will include various blood tests as well as CT or MRI scans. Mri.