**Lesson 17 : Classification of Speech-Language Impairments – Part 03-**

**Aphasia :**

Aphasia is a disorder that results from damage to portions of the brain that are responsible for language. For most people, these areas are on the left side of the brain. Aphasia usually occurs suddenly, often following a stroke or head injury, but it may also develop slowly, as the result of a brain tumor or a progressive neurological disease.

The disorder impairs the expression and understanding of language as well as reading and writing. Aphasia may co-occur with speech disorders, such as dysarthria or apraxia of speech, which also result from brain damage.[[1]](#footnote-2)

Aphasia is often extremely frustrating for the client and his family/caregivers.

The client suffers because he has a decreased functional system of communication.

And because of this language breakdown, many family members feel their loved ones are now becoming mentally ill or senile. This could not be further from the truth.

As a language disorder, asphasia can affect different aspects of language. For example, a client might experience a receptive aphasia which impacts comprehension of spoken and written language, while an expressive aphasia affects a

client’s ability to produce spoken and written language. The Source for Aphasia Therapy covers receptive language skills, reading comprehension skills, and expressive language skills. The receptive and expressive sections deal heavily

with spoken language, while the reading comprehension section, naturally, contains

activities designed to increase reading single letters, words, sentences, and paragraphs.[[2]](#footnote-3)

**Aphasia etiologies:**

Aphasia is observed in about one-third of the patients with so-called **cerebrovascular disorders** or **cerebrovascular accidents** (**CVA**) (stroke).

However, aphasia subtype is variableand can change over time. In the acute stage of recovery, the most frequent aphasia is globalaphasia; however, aphasia profile often changes during the stroke evolution with the mostfrequent aphasia subtype one year later being anomic aphasia . As a matter of fact,anomia represents the most important aphasia symptom manifestation and long-term aphasiasequelae.

A CVA refers to a disruption in normal brain function due to any pathological condition of the

blood vessels: walls of the vessels themselves, accumulation of materials, changes in permeability, or rupture. Stroke can be caused either by a clot obstructing the flow of blood to the brain or by a blood vessel rupturing and preventing blood flow to the brain. Consequently, there are two major types of strokes: **obstructive (ischemic**) and **hemorrhagic**.

At the onset of the CVA, a sudden neurological deficit (e.g., hemiplegia, aphasia, etc.) is often

observed. In severe cases, CVA can be associated with coma. The development of the neurological deficit may take seconds, minutes, hours and occasionally even days, depending upon the specific type of CVA. Loss of consciousness is frequent in hemorrhagic CVAs, but infrequent in ischemic CVAs. Recovery is observed during the following hours, days, or weeks after the accident. As the results of decreases in edema (swelling) and diaschisis (extended impairment effect due to the broad connectivity of each brain area with the rest of the brain), symptomatology is progressively reduced to focal sequelae. The neurological or

neuropsychological residual deficit typically reflects the site and the size of the lesion .[[3]](#footnote-4)

**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.

**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 Silvian 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], 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.

**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?”.

**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.)

**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**.**

**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.

**Primary non-fluent progressive aphasia” :**

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-fluentagrammatic”, “semantic” and “logopenic”. “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.

**Bilingual aphasia:**

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 identifi ed classical language areas within the perislyvian 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. Italso 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 characterized 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 endstudent, 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.[[4]](#footnote-5)

**Guidelines for communication:**

Because of aphasia the way in which someone understands something or expresses himself or herself changes. By making the best possible use

of the remaining communication options people can still communicate with someone who suffers from aphasia. Someone with severe aphasia

often only understands the most important words from a sentence. He or she understands the ‘key words’. To understand things by means of key

words can cause misunderstandings, since because of the combination of key words and the general knowledge of things, the message can be

misunderstood. Sometimes we and someone who suffers from aphasia think we have understood each other well. A later reaction then sometimes

shows that this was not the case.[[5]](#footnote-6)

**How is aphasia diagnosed?**

Aphasia is usually first recognized by the physician who treats the person for his or her brain injury. Most individuals will undergo a magnetic resonance imaging (MRI) or computed tomography (CT) scan to confirm the presence of a brain injury and to identify its precise location. The physician also typically tests the person’s ability to understand and produce language, such as following commands, answering questions, naming objects, and carrying on a conversation.

If the physician suspects aphasia, the patient is usually referred to a speech-language pathologist, who performs a comprehensive examination of the person’s communication abilities. The person’s ability to speak, express ideas, converse socially, understand language, and read and write are all assessed in detail..[[6]](#footnote-7)

**Treatment of aphasia:**

Many people who suffer from aphasia have been hospitalised for some time. That hospitalisation often took place after the brain damage occurred. After being dismissed from the hospital many people with aphasia still need further treatment. It is not always clear who they can turn to for help.

The treatment of aphasia is almost always given by speech therapists. In principle anyone suffering from aphasia is eligible for speech therapy. The duration of the treatment is, among other things, connected with the recovery from the aphasia, and with the possibilities and regulations in the country. [[7]](#footnote-8)

Following a brain injury, tremendous changes occur in the brain, which help it to recover. As a result, people with aphasia often see dramatic improvements in their language and communication abilities in the first few months, even without treatment. But in many cases, some aphasia remains following this initial recovery period. In these instances, speech-language therapy is used to help patients regain their ability to communicate.

Research has shown that language and communication abilities can continue to improve for many years and are sometimes accompanied by new activity in brain tissue near the damaged area. Some of the factors that may influence the amount of improvement include the cause of the brain injury, the area of the brain that was damaged and its extent, and the age and health of the individual.

Aphasia therapy aims to improve a person’s ability to communicate by helping him or her to use remaining language abilities, restore language abilities as much as possible, and learn other ways of communicating, such as gestures, pictures, or use of electronic devices. Individual therapy focuses on the specific needs of the person, while group therapy offers the opportunity to use new communication skills in a small-group setting.

Recent technologies have provided new tools for people with aphasia. “Virtual” speech pathologists provide patients with the flexibility and convenience of getting therapy in their homes through a computer. The use of speech-generating applications on mobile devices like tablets can also provide an alternative way to communicate for people who have difficulty using spoken language.

Increasingly, patients with aphasia participate in activities, such as book clubs, technology groups, and art and drama clubs. Such experiences help patients regain their confidence and social self-esteem, in addition to improving their communication skills. Stroke clubs, regional support groups formed by people who have had a stroke, are available in most major cities. These clubs can help a person and his or her family adjust to the life changes that accompany stroke and aphasia.

Family involvement is often a crucial component of aphasia treatment because it enables family members to learn the best way to communicate with their loved one.

Family members are encouraged to:

1. Participate in therapy sessions, if possible.
2. Simplify language by using short, uncomplicated sentences.
3. Repeat the content words or write down key words to clarify meaning as needed.
4. Maintain a natural conversational manner appropriate for an adult.
5. Minimize distractions, such as a loud radio or TV, whenever possible.
6. Include the person with aphasia in conversations.
7. Ask for and value the opinion of the person with aphasia, especially regarding family matters.
8. Encourage any type of communication, whether it is speech, gesture, pointing, or drawing.
9. Avoid correcting the person’s speech.
10. Allow the person plenty of time to talk.
11. Help the person become involved outside the home. Seek out support groups, such as stroke clubs.

1. NIDCD Fact Sheet | **Voice, Speech, and Language,** **Aphasia, p01** [↑](#footnote-ref-2)
2. Lisa A. Arnold : **The sources of Aphasia therapy, Printed in the U.S.A., 1999, p05** [↑](#footnote-ref-3)
3. **Alfredo Ardila :Aphasia Handbook** , **Florida International University, 2014, p29** [↑](#footnote-ref-4)
4. **Giulio Perrotta\* : Aphasia: Defi nition, clinical**

   **contexts, neurobiological profi les and clinical treatments,** Annals of Alzheimer’s Dementia Care, 27 June, 2020, 22, 23 [↑](#footnote-ref-5)
5. **What is aphasia?, Association Internationale Aphasie**  [↑](#footnote-ref-6)
6. NIDCD Fact Sheet | **Voice, Speech, and Language,**

   **Aphasia** [↑](#footnote-ref-7)
7. **What is aphasia?, Association Internationale Aphasie**  [↑](#footnote-ref-8)