Chapter 1: Introduction

Neurolinguistics, as the term suggests, is a research area which is the outcome of the interaction between two important branches of human science – neurology and linguistics. The present work is an attempt at the linguistic interpretation of a neurological deficit “Aphasia” – how this neurological damage can affect the working of language in human beings – the very fact that the damage to the CNS [Central Nervous System], particularly brain, affects language acts as an evidence for the contention that language is a biological phenomenon, which has been accepted.

Aphasia: An Introduction

The use of speech to communicate is unique to humans. When speech is impaired or absent, the impact on the person and his family is profound. One of the most heartbreaking and devastating disabilities is aphasia. Most people have not heard about aphasia, nor do they know the term until someone in their family or a friend acquires aphasia. Aphasia is an impairment of language, affecting the production or comprehension of speech and the ability to read or write. Aphasia is always due to injury to the brain—most commonly from a stroke, particularly in older individuals. But brain injuries resulting in aphasia may also arise from head trauma, brain tumors, or infections.

Aphasia can be so severe as to make communication with the patient almost impossible, or it can be very mild. It may affect mainly a single aspect of language use, such as the ability to retrieve the names of objects, or the ability to put words together into sentences, or the ability to read. More commonly, however, multiple aspects of communication are impaired, while some channels remain accessible for a limited exchange of information. It is the job of a professional to determine the amount of function available in each of the channels for the comprehension of language, and to assess the possibility that treatment might enhance the use of the channels that are available. Transient aphasia refers to a communication problem that lasts only a few hours or days. More than half of those who initially show symptoms of aphasia recover completely within the first few days.
No medicine or drugs have been known to cure aphasia, as yet. Surgery is successful in those occasions where pressure from a brain tumor or a hematoma, i.e. a swelling filled with blood, impacts a critical speech center. Surgery is not useful in cases of aphasia following stroke, which represent the vast majority of instances. Speech therapy is often provided to persons with aphasia, but does not guarantee a "cure". The purpose of speech therapy is to help the patient fully utilize remaining skills and to learn compensatory means of communication. Who are these speech therapists or if the question is reframed, it would be, ‘who should be worthy of being a speech therapist for these aphasics’? The answer is – none other than linguists with clinical experiences as only a linguist can understand the specific area of deficit in a language as no two patients with aphasia are affected in the same manner.

It has been estimated that about one million people in the United States have acquired aphasia. The majority are the result of stroke. About one third of severely head-injured persons have aphasia. The data pertaining to India is not available but this US data should be taken as somewhat equivalent to those in other countries as the factors leading to stroke are very common irrespective of age, sex, race, or nationality. Vocation and education are not determining factors and there are no definitive steps to prevent the onset of aphasia in the event of a stroke or head trauma. The degree of the pathological condition is determined by the location and size of the area of damage in the brain.

By responding quickly to the signs of stroke, one may greatly increase the likelihood of a successful recovery. A prompt response may give doctors the chance to administer a clot-busting drug, repair a bleeding vessel, or perform other interventions that can prevent debilitating brain damage.

Major warning signs of stroke are numbness, weakness, or paralysis of the face, arm or the leg, loss of speech, difficulty talking or understanding speech, dimming, blurring or loss of vision, in one or both the eyes, dizziness, difficulty walking, or loss of balance or co-ordination severe headache, without an apparent cause, nausea, vomiting, and fever unrelated to another illness, confusion or an episode of convulsions or unconsciousness. It should be noted that one or more of the signs may appear and that any given sign
might soon disappear. But even a transient sign warrants attention, for it may represent one’s opportunity to prevent a permanently disabling stroke so the neurologist should be contacted for the treatment quickly.

How Stroke Affects People

Each stroke is different, depending on the part of the brain injured, how bad the injury is, and the person's general health. Some of the effects of stroke are -

1. Weakness (i.e. hemiparesis) or paralysis (i.e. hemiplegia) on one side of the body – this may affect the whole side or just the arm or the leg. The weakness or paralysis is on the side of the body opposite the side of the brain injured by the stroke. For example, if the stroke injured the left side of the brain, the weakness or paralysis will be on the right side of the body.

2. Problems with balance or coordination – these can make it hard for the person to sit, stand, or walk, even if muscles are strong enough.

3. Problems using language (aphasia and dysarthria) – a person with aphasia may have trouble understanding speech or writing. Or, the person may understand but may not be able to think of the words to speak or write. A person with dysarthria knows the right words but has trouble saying them clearly.

4. Being unaware of or ignoring things on one side of the body (bodily neglect or inattention) – often, the person will not turn to look toward the weaker side or even eat food from the half of the plate on that side.

5. Pain, numbness, or odd sensations – these can make it hard for the person to relax and feel comfortable.

6. Problems with memory, thinking, attention, or learning (cognitive problems) – a person may have trouble with many mental activities or just a few. For example, the
person may have trouble following directions, may get confused if something in a room is moved, or may not be able to keep track of the date or time.

7. Being unaware of the effects of the stroke – the person may show poor judgment by trying to do things that are unsafe as a result of the stroke.

8. Trouble swallowing (dysphagia) – this can make it hard for the person to get enough food. Also, care must sometimes be taken to prevent the person from breathing in food (aspiration) while trying to swallow it.

9. Problems with bowel or bladder control – these problems can be helped with the use of portable urinals, bedpans, and other toileting devices.

10. Getting tired very quickly – becoming tired very quickly may limit the person's participation and performance in a rehabilitation program.

11. Sudden bursts of emotion, such as laughing, crying, or anger – these emotions may indicate that the person needs help, understanding, and support in adjusting to the effects of the stroke.

12. Depression – this is common in people who have had strokes. It can begin soon after the stroke or many weeks later, and family members often notice it first. It is normal for a stroke survivor to feel sad over the problems caused by stroke. However, some people experience a major depressive disorder, which should be diagnosed and treated as soon as possible. A person with a major depressive disorder has a number of symptoms nearly every day, all day, for at least 2 weeks. These always include at least one of the following: feeling sad, blue, or down in the dumps or loss of interest in things that the person used to enjoy.

In addition, a person may also have other physical or psychological symptoms, including – feeling slowed down or restless and unable to sit still, feeling worthless or guilty,
increase or decrease in appetite or weight, problems concentrating, thinking, remembering, or making decisions, trouble sleeping or sleeping too much, loss of energy or feeling tired all the time, headaches, other aches and pains, digestive problems, sexual problems, feeling pessimistic or hopeless, being anxious or worried, thoughts of death or suicide. If a stroke survivor has symptoms of depression, especially thoughts of death or suicide, professional help is needed right away. Once the depression is properly treated, these thoughts will go away. Depression can be treated with medication, psychotherapy, or both. If it is not treated, it can cause needless suffering and also makes it harder to recover from the stroke.

**Childhood (Pediatric) Stroke: Not a Myth**

There are popular notions that "Strokes don't happen to children!" This has been told time and time again, yet there are cases of stroke deaths and stroke survivors with disabilities in children. Childhood stroke, which is still referred to as pediatric stroke, is no longer "rare". There are several statistics regarding childhood stroke. The latest statistics from Dr. John Kylan Lynch, from the National Institute of Neurological Disorders and Stroke (NINDS) in US, show that 25 out of 100,000 newborns will have an infant stroke. About 6 percent of those children will die, 20 to 35 percent will go on to have another stroke, and more than two-thirds of survivors will have neurological deficits or seizures. Strokes in children occur at the rate of 3 out of 100,000. 12% of children die from stroke each year.

The most common causes of stroke in children are heart disease, infection (meningitis / encephalitis), sickle cell disease, trauma, i.e. injury, and coagulation abnormalities and in 25-30% the reason is unknown. Other reasons could be arteriovenous malformation (AVM), birth injury, clotting or coagulation disorders, heart disease and child stroke, MoyaMoya disease, prematurity.

One of the main disabilities that result from child stroke is hemiplegia. Signs of hemiplegia or hemiplegic cerebral palsy may be, but are not limited to - behavioral issues, lack of control on the stroke affected side of the body, learning difficulties, little
use of one hand, drop toe, limping or other gait difficulties, muscle weakness or stiffness, poor balance, sensory issues, vision difficulties. Another main disability from child stroke is seizures. It is estimated that 90% of childhood stroke survivors have some type of seizure disorder. So a child may encounter these disabilities as a result of stroke – cerebral palsy, epilepsy, hemiplegia & hemiparesis, hypotonia (low strength), vision disabilities, hemianopia (blindness in one half of the visual field).

The treatments may include – speech therapy, assistants for daily living, Botox injections, early childhood or special education services, medications, music therapy, occupational therapy, physical therapy, rehabilitation, surgery.

**Concepts & Definitions of Aphasia: A Review**

**Paul Broca (1861)** defined “Aphemia”, i.e. aphasia, as a loss of speech consequent to lesion of the frontal lobe of the brain, the lesion usually localized in third frontal convolution and sometimes in the first convolution. Broca used the term speech rather than language which shows his preference for “articulated oral language” over oral comprehension of language. Broca (1865) noted that in all his cases, the damage is localized in the left hemisphere. Generalizing these findings, he said, “We speak with the left hemisphere”.

**Carl Wernicke (1874)** demonstrated that damage to an area in the back of sylvian fissure had caused linguistic deficits Unlike Broca’s patient, the patients he described had impaired comprehension and they frequently used elaborate descriptions in response to a single word, i.e. “circumlocution”. The patients also made phonemic substitutions. The collection of these symptoms described by Wernicke is known as Wernicke’s Aphasia.

**J. Hughlings Jackson (1879)** defined aphasia as impairment in linguistic formulation and expression. He was among the pioneers to highlight the need to understand the patients’ “premorbid personality” in order to appreciate the modifications that occur as

\[\text{As quoted in Ohler and Gjerlow (1999)}\]
‘sequelae of brain damage and overall aphasic involvements’). According to him, an aphasic person becomes “lame in thinking”.

Influenced by both Darwinian evolutionary theory and Hughlings Jackson’s writings on aphasia, S. Freud (1891) identified a “continuous cortical region ... between the terminations of optic and acoustic nerves and the areas of the cranial and peripheral motor nerves in the left hemisphere” as the central apparatus for speech. All the convolutions of sylvian fissure contribute in speech functions. Freud viewed aphasic disturbances as evolutionary retrogressions. Anyway, Freud was a little influence on his contemporaries and the reason was the unavailability of the translation of his manuscript on aphasia. In his “Project” he tried to break the myth of mind. Pierre Marie (1906) identified aphasia as a deficit of intellect related to the impairment in the language use. For Marie, Broca’s definition of aphasia is actually that of dysarthria. The intellectual deficit, described here, may be same as the impairment of language comprehension. Henry Head (1926) thought aphasia to be the disturbances of symbolic formulation and expressions. His concept of aphasia implies that all functioning in which some symbolization is involved is impaired in aphasic persons. He had some reservations against Jackson’s use of proposition and its implications although the cognitive school in the field of aphasiology was founded by Hughlings Jackson through his emphasis on the intellectual modifications of the aphasics. Wisenberg & McBride (1935) were one of the precursors to note the significance of handling the aphasic impairments individually, i.e. single case approach, and they advocated the need to consider the patient’s premorbid intellectual state while analyzing them. Kurt Goldstein (1948) further emphasizes the need to take the aphasics individually by considering their thought and behavior, post-onset. He also belonged to the cognitive school of aphasiology. He considered the significance of the impairment of abstract thinking and reasoning as the underlying

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2 Which was a draft until it was translated and published in 1895 by Basic Books under the title of “Project for a Scientific Psychology” but better known as “Project” after the publication.
3 As quoted in Lesser (1978)
4 As quoted in Eisenson (1984)
6 As quoted in Eisenson (1984)
substrate for verbal and non-verbal behavior of the aphasics. Weigl & Bierwisch (1973)\(^7\) presented the basis of neuropsychological research for the investigation of aphasics’ state and stressed on the need to consider the system of linguistic competence, which underlies the complex system of performance strategies, and performance – as the ‘aphasia must be considered ... as interference of those components...’ while the underlying competence remains intact. So these hypotheses are based on the claims that competence and performance are two psychologically distinct processes and in aphasia the performance is disturbed\(^8\). Damasio (1981) views aphasia as disturbances in comprehension and verbal encodings resulting from brain damage.

The derangement of language must result from the pathology of the brain. The disorders of the language abilities resulting from functional disorders or psychiatric disorders are excluded. By a consensus aphasia could be defined as the impairment of linguistic competence or performance as a result of acquired (localized) brain damage.

### Aphasia Classification: An Overview

In aphasia brain injury results in language deficits and not all patients of aphasia have same deficit features. The idea of taxonomy begins with this differentiation.

Sometimes in aphasia the loss of communication is total (when the comprehension, auditory & written, and production are minimized) and this continues – then this aphasia is referred to as “global” (aphasia), which is usually a result of a larger damage in the left hemisphere of the brain. But not all the patients who cannot communicate through language at all immediately after brain injury are ‘global’ aphasics as the aphasics tend to recover over days, or weeks to a partial-deficit level. In few cases, the patients even recover fully from zero state and this is an evidence of preservation of linguistic

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\(^7\) In 1981 their paper ‘Neuropsychology and Linguistics: Topics of Common Research’ was published in a collected volume [Weigl et al, ‘Neuropsychology and Neurolinguistics: Selected Papers’, Mouton].

\(^8\) In Weigl & Bierwisch (1973) the viewpoint is that aphasia is the deficit to the access to the knowledge of the language, i.e. linguistic competence, which is intact.
competence even with severe problems in production/comprehension. There is language independent of all the language modalities and this may be preserved.

The main goal of the classification is description of the clinical features. Aphasia can be neuroanatomically and behaviorally classified into different types or syndromes of aphasia, involving the primary language cortex or other cortical/sub-cortical regions. **Paul Broca (1868)**, among the firsts to classify aphasia, presented his understanding of aphasia before “British Association for the Advancement of Science”. He classified the “disorders of speech into ‘aphemic’ and ‘amnesic verbale’” [Head (1926, 1963)].

An “aphemic” patient will have a decreased output or would be without speech except for some ‘monosyllables’/words, while an “amnesic” patient loses the associations between ideas and words, the memory (both of spoken/written words), although he can pronounce but cannot associate them to the concept.

**Wernicke-Licktheim’s** [Lichtheim (1885)] classification assumes the existence of seven forms of aphasia. The “Cortical Motor Aphasia” results from the destruction of Broca’s area where motor images of words are stored. “Sub-cortical Motor Aphasia” is due to an interruption of the conduction pathway between Broca’s area and the articulatory apparatus. “Trans-cortical Motor Aphasia” results from the dissociation of Broca’s area and other intellectual cortical areas. “Cortical Sensory Aphasia” results from lesions in the Wernicke’s area where auditory word images are stored. “Sub-cortical Sensory Aphasia” is produced by the interruptions between the projective auditory region and Wernicke’s area. “Trans-cortical Sensory Aphasia” is due to disrupted conduction pathways between Wernicke’s area and other intellectual cortical regions. “Conduction Aphasia” happens due to the dissociation of Broca’s area and Wernicke’s area.

A Cortical Motor aphasic preserves speech comprehension but the ability of spontaneous and repetitive speech is impaired. A Sub-cortical Motor aphasic can write too and this separates him from other Cortical Motor aphasics. Trans-cortical Motor aphasics can repeat speech they hear but can’t speak fluently and comprehension is preserved. A Cortical Sensory aphasic can neither comprehend nor repeat the utterances he hears but
he is able to speak fluently. Sub-cortical Sensory aphasics don’t grasp what they hear nor can they repeat or write from dictation. Spontaneous speech, reading, and writing are retained. In Trans-cortical Sensory Aphasia, the patient can repeat what he hears but doesn’t comprehend the utterances. This results from the fact that although the auditory impulses reach the intact Wernicke’s area, they are transmitted only to Broca’s area, thus making repetition possible, but don’t reach those areas where meaning is decoded (i.e. where words are assigned senses and concepts). A person with Conduction Aphasia has a typical symptom of disturbed repetition with intact comprehension and spontaneous speech. The naming ability is also preserved.

Head (1926, 1963) classified aphasic disorders into four broad categories. “Verbal Aphasia” is difficulty in forming words for external or internal speech. In this, written text comprehension and auditory comprehension is better than the ability to pronounce speech/writing. “Syntactical Aphasia” is a disorder of syntax, balance and rhythm. The patient has plenty of words but he/she can’t co-ordinate them into valid syntactic structures. “Nominal Aphasia” is difficulty in using names and the patient has severe problems in reading and writing. “Semantic Aphasia” consists in a “want of recognition of the full significance of words and phrases apart from their direct verbal meaning”. The patient may get a word or a phrase but can’t get its “ultimate significance” and this results into the disorder of comprehension of the intention of the speaker/ of the text. Head attempted to avoid the intake-output based dichotomy. His classification showed a bias against the notion of discrete or single (pure) aphasic disorders.

Like Head, Weisenburg and McBride (1935) classified aphasia without any commitment for the dichotomy. “Predominantly Receptive Aphasia” is the inability to comprehend spoken/written language. In this, the disturbance is in the ability to express in speech or writing. An ‘amnesic’ patient, suffering from expressive disturbance, couldn’t evoke the appropriate word as names (of objects, conditions, relationships etc.). In “Expressive-Receptive Aphasia”, a global type disturbance, the patient suffers from both expressive and receptive language disorders. Usually receptive disorders improve spontaneously over time to a greater degree than expressive disorders do. Like Head’s,
these groups were too broad, covering too many impairments under a single group and
did not intend to differentiate the aphasic patients according to syndromes of impairment.
Despite everything, they have placed a model for the development of aphasic inventories.

A. R. Luria (1964, 1966), a Russian neuropsychologist and an aphasiologist gave a
classification system based on Pavlovian neurophysiology. He classified aphasic
impairments according to their underlying disturbed function and the related defect in
language. “Sensory Aphasia” is a disturbance in the analysis and synthesis of speech
sounds which results in the defects in the comprehension of speech, defects in word
production in both spoken and written medium. “Acoustic-Amnesic Aphasia” is a
disturbance in the retention of audio speech traces resulting in impairment that increases
as the content is increased. A patient has problems in writing or speaking a non-fixed
series of verbal items. In “Afferent (Kinesthetic Motor) Aphasia”, the disturbance is in
the kinesthetic analysis of speech movements. The patient has a difficulty in the
production of fundamental units of speech, i.e. ‘articulemes,’ and basically the deficiency
is using the afferent stimulation as a control for articulatory production. The basic
impairment in “Efferent (Kinetic Motor) Aphasia” is in the kinetic analysis for a
sequence of speech movements. The patient can pronounce the individual sounds but
can’t pronounce sounds in sequence. Parallel defects are found in writing also. They can
write individual letters but problems in writing words come when they either repeat
letters or transpose them. In “Semantic Aphasia”, the primary disturbance is in the
simultaneous organization of the components of speech. The patient has difficulty in the
appreciation of meaning of a verbal formulation, of the “logico-grammatical structure of
speech”. In “Dynamic Aphasia”, the disturbance is in the inner speech which has a
“shortened structure”, a predicative function and serves as a fundamental means for the
transformation of a fore shortened idea into developed outer speech and for the change of
developed speech into a fore shortened scheme of thought’ [Luria (1964), as quoted in
Eisenson (1984)].

Roman Jacobson (1964), one of the pioneers to present a linguist’s point of view in his
classification system, noted that “the most striking symptoms of aphasia cannot be found
without the...assistance of linguistics”. He identified two broad categories – “similarity” (decoding) disorders and “contiguity” (encoding) disorders. His two types of disorder are in line with Luria’s six types – his contiguity disorder characterizes efferent, afferent and dynamic types of Luria with deterioration of the code and Luria’s sensory, semantic and amnesic display similarity disorder with damage to the code.

The Boston classification of aphasias by Goodglass and Kaplan (1983) uses a framework of component deficits rather than neurodynamics and Pavlovion system of analysers, as does Luria’s classification. In the Boston classification, the areas of deficit are--- articulation, fluency, word finding, repetition, seriatim speech, grammar, paraphasia, auditory comprehension, reading and writing. This Boston classification has been used in a largeumber of linguistic investigations of aphasic disorders in English language.

In the Boston classification, there are six types of aphasia—“Wernicke’s Aphasia” (lesion on the posterior portion of the first temporal gyrus); “Broca’s Aphasia” (lesion in the third frontal convolution); “Anomic Aphasia” (lesion in the temporal-parietal region—may extend to angular gyrus; “Conduction Aphasia” (lesion in the supramarginal gyrus deep to ‘arcuate fasciculus’, i.e. the connecting tissue between Broca’s area and Wernike’s area, making cortical & sub-cortical connections predominantly); “Trans-cortical Sensory Aphasia” or “Isolation Syndrome” (lesion in the band of infarcted tissues which cuts off an intact Wernicke’s-Broca area from rest of the brain); and “Trans-cortical Motor Aphasia” (lesion in the tissue of frontal lobe marginal to third frontal gyrus).

Features of Chief Types of Aphasia

Over a century of experience with the study of aphasia has taught us that particular components of language may be particularly damaged in some individuals. We have also learned to recognize different types or patterns of aphasia that correspond to the location of the brain injury in the individual case. Some of the common varieties of aphasia are:
Global Aphasia - This is the most severe form of aphasia, and is applied to patients who can produce few recognizable words and understand little or no spoken language. Global aphasics can neither read nor write. Global Aphasia may often be seen immediately after the patient has suffered a stroke and it may rapidly improve if the damage has not been too extensive. However, with greater brain damage, severe and lasting disability may result.

Broca’s Aphasia - This is a form of aphasia in which speech output is severely reduced and is limited mainly to short utterances, of less than four words. Vocabulary access is limited in persons with Broca’s Aphasia, and their formation of sounds is often laborious and clumsy. The person may understand speech relatively well and be able to read, but be limited in writing. Broca’s Aphasia is often referred to as a “Non-fluent Aphasia” because of the halting and effortful quality of speech.

Mixed Non-fluent Aphasia - This term is applied to patients who have sparse and effortful speech, resembling severe Broca’s Aphasia. However, unlike persons with Broca’s Aphasia, they remain limited in their comprehension of speech and do not read or write beyond an elementary level.

Wernicke’s Aphasia - In this form of aphasia, the ability to grasp the meaning of spoken words is chiefly impaired, while the ease of producing connected speech is not much affected. Therefore Wernicke’s Aphasia is referred to as a “Fluent Aphasia”. However, speech is far from normal. Sentences do not hang together and irrelevant words intrude-sometimes to the point of sounding like jargon, in severe cases. Reading and writing are often severely impaired.

Anomie Aphasia - This term is applied to persons who are left with a persistent inability to supply or name the words for the very things they want to talk about-particularly the significant nouns and verbs. As a result their speech, while fluent in grammatical form and output, is full of vague circumlocutions and expressions of frustration. They understand speech well, and in most cases, read adequately. Difficulty in finding words is as evident in writing as in speech.

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Other Features in Aphasia

In addition to the foregoing syndromes that are seen repeatedly by speech clinicians, there are many other possible combinations of deficits that do not exactly fit into these categories. Some of the components of a complex aphasia syndrome may also occur in isolation. This may be the case for disorders of reading (Alexia) or disorders affecting both reading and writing (Alexia and Agraphia), following a stroke. Severe impairments of calculation often accompany aphasia, yet in some instances patients retain excellent calculation in spite of the loss of language.

There are a variety of disorders of communication that may be due to paralysis, weakness, incoordination of speech musculature or cognitive impairment. Such impairment may accompany aphasia or occur independently and be confused with aphasia. It is important to distinguish these disorders from aphasia because the treatment(s) and prognosis of each disorder are different.

Apraxia is a collective term used to describe impairment in carrying out purposeful movements. People with severe aphasia are usually extremely limited in explaining themselves by pantomime or gesture, except for expressions of emotion. Commonly they will show you something in their wallet, or lead you to show you something, but this is the extent of their non-verbal communication. Specific examination usually shows that they are unable to perform common expressive gestures on request, such as waving goodbye, beckoning, or saluting, or to pantomime drinking, brushing teeth, etc. (Limb Apraxia). Apraxia may also primarily affect oral, non-speech movements, like pretending to cough or blow out a candle (Facial Apraxia). This disorder may even extend to the inability to manipulate real objects. More often, however, Apraxia is not very apparent unless one asks the patient to perform or imitate a pretended action. For this reason it is almost never presented as a complaint by the patient or the family. Nevertheless it may underlie the very limited ability of people with aphasia to compensate for the speech impairment by using informative gestures. Apraxia of Speech is frequently used by speech pathologists to designate an impairment in the voluntary production of articulation.
Fig A: Human Brain

- cerebrum
- sylvian fissure
- brainstem

Human Brain

Fig B: A nerve cell (neuron)

- dendrites
- nucleus
- Myelin sheath
- Axon

Neuron
and prosody (the rhythm and timing) of speech. It is characterized by highly inconsistent errors.

Dysarthria refers to a group of speech disorders resulting from weakness, slowness, or incoordination of the speech mechanism due to damage to any of a variety of points in the nervous system. Dysarthria may involve disorders to some or all of the basic speech processes: respiration phonation, resonance, articulation, and prosody. Dysarthria is a disorder of speech production, not language (e.g., use of vocabulary and/or grammar). Unlike Apraxia of Speech, the speech errors that occur in Dysarthria are highly consistent from one occasion to the next.

Dementia is a condition of impairment of memory, intellect, personality, and insight resulting from brain injury or disease. Some forms of Dementia are progressive, such as Alzheimer's disease, Pick's disease, or some forms of Parkinson's disease. Language impairments are more or less prominent in different forms of Dementia, but these are usually overshadowed by more widespread intellectual loss. Since Dementia is so often a progressive disorder, its prognosis is quite different from aphasia.

**Human Brain (Fig. A)**

It is divided into a higher section (cerebrum), and a lower section (brainstem or primitive brain). The cerebrum -- which is not essential for life but its purpose, a very important one, is "to integrate an animal with its environment" [Aitchison (1989)] -- is divided into two halves vertically - left and right hemisphere, each made up of four lobes namely frontal, parietal, occipital and temporal. Though some specific functions are assigned to each of the lobes, overlapping has been witnessed\(^9\). These are not entirely separate but very closely connected by nerve fibre bundles, the most important of them being 'corpus callosum' [200 million in number].

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\(^9\) Though frontal lobe is primarily known for performing intellectual functions but the role and the involvement of other lobes have also been established beyond doubt.
Nerve fibres are bundles of nerve cells with specific functions. A ‘neuron’ [Fig. B] is a nerve cell which consists of a cell body and one or more ‘extensions’ that are similar in function to the electrical cords that connect power sources to the appliances and these extensions can be extremely long and carry impulses toward or away from the cell body. These extensions are ‘axons’ and ‘dendrites’. Axons of a cell carry impulses away from the cell body and dendrites carry impulses toward the cell body. ‘Myelin’ is a layer encasing (and insulating) medullated nerve fibers. One single axon may control a number of muscle fibres. A nerve cell and the muscle fibres (controlled by the axons of the nerve cell) constitute one ‘motor unit’.

The surface of cerebral hemispheres is called ‘cortex’ which is distinguished by its convolutions – hill ['gyrus' / 'gyri' (pl.)] and valley ['sulcus' / 'sulci' (pl.)]. The ‘Rolandic fissure’ separates the frontal and parietal lobes. ‘Sylvian fissure’ cuts through the language area with temporal lobe below and parietal lobe above. The outer layer or the cortex consists of nerve cell bodies and is referred to as ‘gray matter’, and beneath it, i.e. the ‘subcortical region’, the nerve cell fibre layer is called ‘white matter’. Although cortex is most crucial for language but subcortical regions also play an important role in this. Structures in the brainstem – thalamus and hypothalamus – are primarily involved with more basic functions like sleeping, and appetite. Thalamus, made up of gray matter, gives a channel to all the sensory impulses to cerebral cortex. Hypothalamus takes care of the emotions, consciousness and breathing.

**Localization & Lateralization (Lateral Dominance)**

Lateralization is localizing the (linguistic) functions into one hemisphere of the brain. The two similar looking cerebral hemispheres are not identical in the performance of functions. The left hemisphere controls the right side of the body and the right hemisphere the left side of the body. Despite the debate of “Localizationists versus Holists,” the issue of lateral dominance has been widely accepted.

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10 Discussed in Chapter 3.
Fig. C: left hemisphere of the human brain
Tests for Lateral Dominance

It is very difficult to determine the hemispheric dominance for language and for it a test requiring no structural brain damage is needed. There are techniques for this determination but none of these are satisfactorily proved and safe.

1. In Sodium Amytal Test [Wada & Rasmussen (1960)\(^{11}\)] the patient is asked to count out aloud while ‘barbiturate’ (sodium amytal) is injected in the artery\(^{12}\) carrying blood to one side of the brain. If this is the hemisphere used in speech, he will be disrupted by the brain and severe language difficulties will appear for some minutes, and if not, the patient will resume counting. Although the test is effective, because of the risk involved this is used when surgery of the brain is to be performed (as in the cases of severe epilepsy) as the surgeon wishes to know whether the speech areas are likely to be disturbed.

2. In Dichotic Listening Test [Kimura (1967)\(^{13}\)] a subject is asked to wear the headphones and then two different words are played, one into each ear. Most people can report the word played to the right ear more accurately than the word played into the left ear. It is because right ear has a preference for words and the left ear is better for non-linguistic sounds. A person will identify the tune played into the left ear better than that into the right ear. So, the conclusion is left hemisphere is better at processing linguistic signals.

3. Electrodiagnosis [Rosenfield & Goree (1975)\(^{14}\)] In this process—electrodes are attached to the skull in order to measure the amount of (energy) electrical activity in the area beneath. Various experiments have shown that spoken words produce greater response in left hemisphere whereas noises such as mechanical clicks arouse a greater response in the right hemisphere.

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\(^{11}\) As quoted in Benson (1979)
\(^{12}\) Carotid artery is the principle artery on each side of the neck.
\(^{13}\) As quoted in Benson (1979)
\(^{14}\) As quoted in Benson (1979)
These tests observed that as many as 90% of the human beings have speech located in the left hemisphere. Therefore localization may not be acceptable to most aphasiologists and neurologists but lateralization has been accepted by almost all of them. The table (Table 0.1) given below suggests the findings of the different cerebral dominance tests.

<table>
<thead>
<tr>
<th>Speech Location</th>
<th>In Right-Handed People</th>
<th>In Left-Handed People</th>
</tr>
</thead>
<tbody>
<tr>
<td>Left Hemisphere</td>
<td>90% OR MORE</td>
<td>70-90%</td>
</tr>
<tr>
<td>Right Hemisphere</td>
<td>10% OR LESS</td>
<td>10-30%</td>
</tr>
</tbody>
</table>

Table 0.1: Language Localization and Handedness

In addition to this, Benson (1979) provides a ‘neuroanatomical’ basis for language dominance. The anatomical asymmetries between the two hemispheres have been demonstrated subtly. There is an asymmetrical crossing of pyramidal motor fibres in the medulla oblongata and also an asymmetry in the size of occipital horns of the lateral ventricles, the left being both longer and broader [Mohr (1989)]. The left sylvian fissure is consistently longer and more horizontally placed, suggesting there is more cortex at the left temporo-parietal junction [LeMay & Culebras (1972)]. The most suggestive cortical asymmetry is in the size of temporal planum15. The left planum is usually larger in most of the brains [Geschwind & Levitsky (1968)]. These observations indicate significant asymmetry between the two cerebral hemispheres.

Single Case & Group Studies

There has been a debate regarding single case vs. group-studies over the past two decades. There are strong claims for single case method16 that single case-studies are

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15 The portion of the auditory association cortex lying on the superior surface of the temporal lobe.
justifiable and are the only source of valid inferences about the structure of the normal cognitive system. Neither anatomical impairments nor cognitive-linguistic impairments are same for two patients, hence no group study is justified in such cases. The variables observed in the group of patients are too great in number to justify the groupings and the extensive variability displayed by the patients, which reflect meaningful and very specific patterns of dissociations, would be lost in case they are grouped together in the name of agrammatism [Miceli et al (1989)]. Averaging over the findings in a group of the patients increases the probability of chance and therefore the preferred methodology should be multiple single-case studies rather than group studies. The present study also comprises multiple single-case studies.