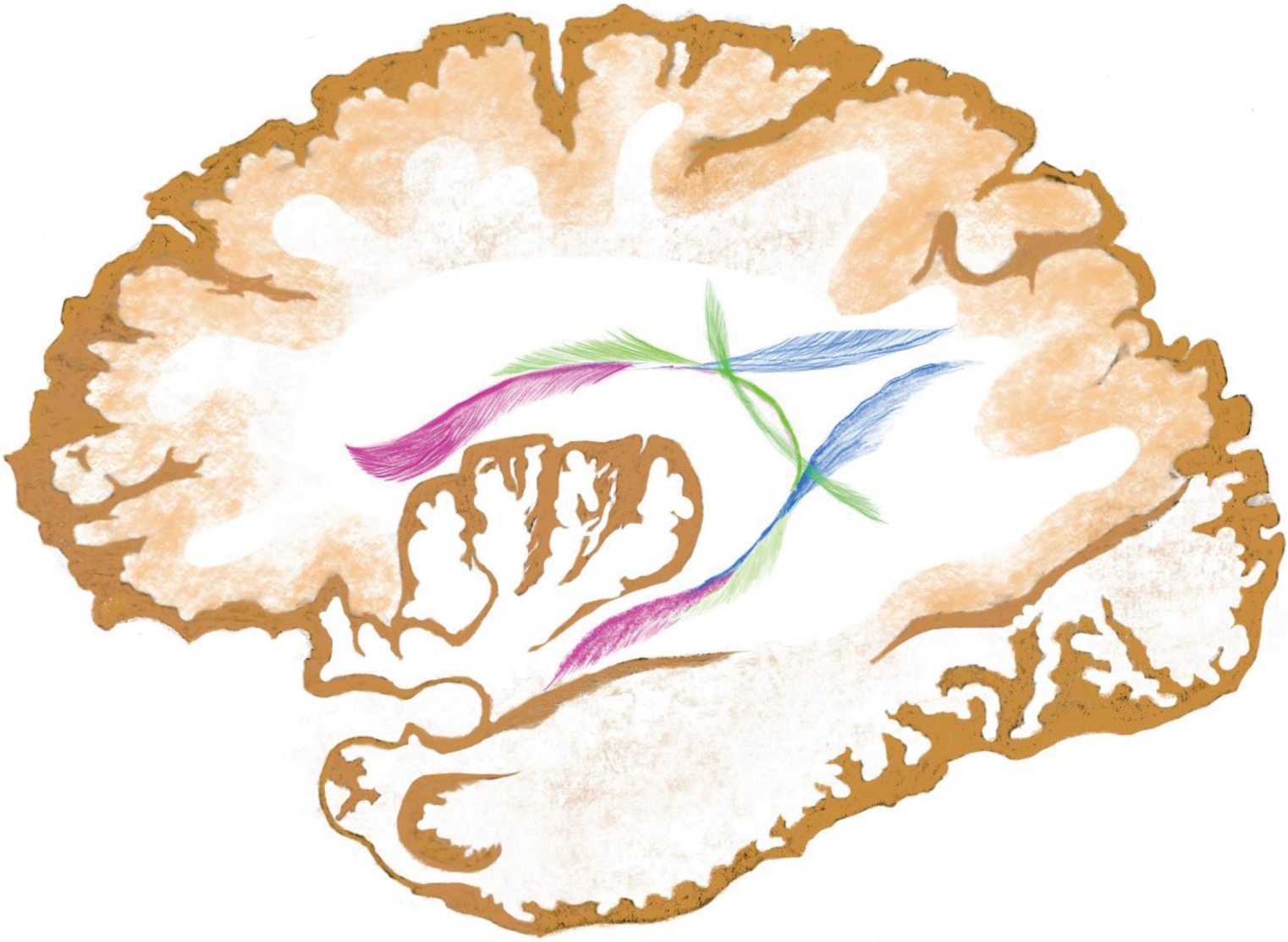
# Speech, Language And Human Brain



PN Tandon
P Sarat Chandra

## ABOUT THE COVER PAGE DIAGRAM

Schematic diagram depicting artistic representation of dorsal and ventral speech pathways connecting the Broca's, Wernicke's area and associative areas involved in speech comprehension and articulation.

Catani M, Jones DK, ffytche DH. Perisylvian language networks of the human brain. Ann Neurol. 2005 Jan;57(1):8-16. doi: 10.1002/ana.20319. PMID: 15597383.

Diagram created Dr Raghavendra M, fellow, Epilepsy surgery & functional neurosurgery, AIIMS, New Delhi using Procreate.

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# SPEECH, LANGUAGE AND HUMAN BRAIN

## ACKNOWLEDGEMENTS

It is obvious that a book on a subject continuously studied over centuries and further revolutionized over the past few decades with the advent of new technologies cannot be comprehensively produced without input from diverse sources. This has been attempted with the help of several knowledgeable persons who must be gratefully acknowledged. Professor Pankaj Seth of the National Brain Research Centre (NBRC) provided the reprints of many publications with unbelievable willingness and promptness. This helped me advance my knowledge of the subject and broaden my concepts. Interaction with Prof Nandini Singh Chatterjee (NBRC) revived my interest in the neurobiology of speech, resulting in a paper, "Educational Neuroscience" as an Editorial, published in Ann Neurosci 23,63-65-2016. We revised this paper by adding new knowledge. My dear friend Professor Bruce Alberts, former President of the National Academy of Sciences USA and later Editor-in- Chief of Science, reviewed this paper with his well-known interest in science education. He provided valuable input and encouraged us to further elaborate on this subject, in which format it now appears in the book. It would not be enough to say "Thank you" to him. Prof Soumaya Iyenger (NBRC) and her colleagues, with their long-standing pioneering works on "Neurobiology of Bird Song", have kindly agreed to summarize current knowledge on the subject. Prof. Aiyesha Kidwai of the Jawaharlal Nehru University, New Delhi, a distinguished linguist, graciously provided inputs with which I was unfamiliar. This stimulated me to study at least the basics of the subject. She then reviewed several chapters—my heartfelt thanks to her.

Professor P. Sarat Chandra, a distinguished neurosurgeon at the AIIMS, New Delhi, and a long-term Editor of Neurology India offered to be an Associate Editor of this volume. Earlier, we had published a monograph, "Evolution of Neurosciences" (Neurology India 70 (suppl 1) 2022). He did the final pre-editing of the book and painstakingly corrected any mistakes and deficiencies in the text. Without his help, it would not have been possible to provide the final acceptable version of the book.

Pooja Gosain (NBRC) provided administrative support to me throughout the period required to produce the book. Kuldeepfi Kumar (NBRC) produced my hand-written drafts' repeated soft and hard copies with unique patience and ef ciency. He deserves very special thanks. This work was carried out when I was an Emeritus Professor at AIIMS and at the National Brain Research Centre, Manesar and also a National Research Professor of the Ministry of Human Resource Development, Government of India.

In a lighter vein, I conclude this book was written "behind the back" of my dearest wife. Confined to bed due to various disabilities, always lying on her left side, I provided her my constant company on the other side of the bed During the last couple of years. I did all my reading and writing for this work while attending to all her "beck and calls". It is deeply regretted that she will not be there to look at the find product with her usual "Charming Smile" and a "mystic very good".

I often wondered, at my age past 90, if I would be able to say "Thank Mata Saraswati, it is over with your kind grace".

PN Tandon

Editor

## ACKNOWLEDGEMENTS

Speech and Language and its correlation with the human brain have been a subject of long-standing intrigue and fascination.

For me, it has been a task of sacredness.

For many reasons, but first and foremost, it allowed me to interact meaningfully with my mentor and teacher of teachers, Prof PN Tandon.

When Prof Tandon told me two years back that he would write a book on "Speech, Language and Human Brain", I was overwhelmed.

Not just because this topic, even though discussed and discussed extensively, very little is still known about it…..

Not just because very few books have extensively and comprehensively discussed this topic to be of use for the practicing clinician….

Not just because I was taken aback by the courage of my teacher to undertake this momentous task.

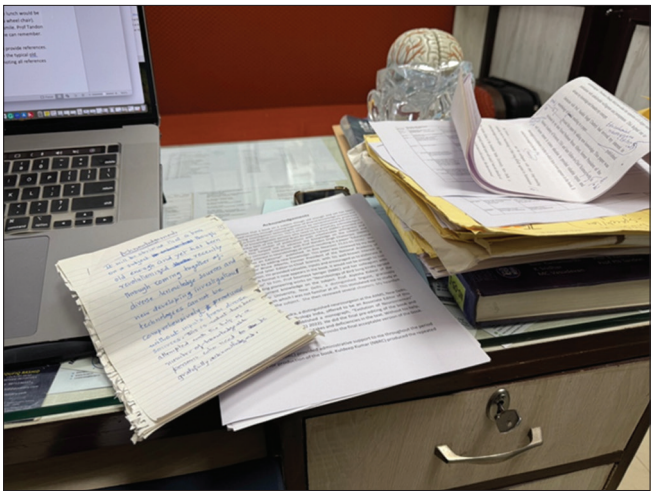
But, by my wonder, to appreciate his continuous thirst for knowledge, his inability to say "I have done enough", and his never-ending curiosity to continue learning how the brain functions.

Figure 1: Prof PN Tandon with Prof P Sarat Chandra at the former's residence while working on the book

Amid all this, I do have a confession to make.

My contributions have only been to make the corrections and provide references. Prof Tandon has single-handedly written the entire book, in the typical old-fashioned manner (Figure 2), with a pen on paper, meticulously noting all references himself. This would be later typed and corrected.

Figure 2: Prof PN Tandon's notes while writing the book



Prof Tandon is one of the original teachers, a rare jewel in the crown of academics. I feel fortunate that I was able to have interacted with him and have been mentored by him.

Before I end my brief acknowledgement, I would like to thank my fellow Dr Raghavendra for having helped in creating the schematic drawings and the cover page. I would also like to thank my secretarial staff Sandeep and Roop Narayan, who went beyond their call of duty and would go to Prof Tandon's residence to pick up the papers or drop them back to him.

My humble prostrations to my teacher, like we say in our scriptures "Gurur Brahma, Gurur Vishnu, Gurur Devo Maheshwaraha, Guru Saakshaat Para Brahma Tasmai Shri Guruve Namaha", meaning "The Guru is Brahma, the Guru is Vishnu, the Guru is Maheshwara (Shiva). The Guru is the Supreme God himself. I bow to that Guru.

P Sarat Chandra

Associate Editor

## ABOUT THE BOOK

The book is a treatise compiled from the most significant publications and texts on speech and language concerning the human brain. It starts with the historical perspective and goes to the most recent concepts. It has been written in a simple manner that most would be able to understand or grasp it. Hence, we would say that this book is not just for a neurosurgeon or a neurologist but also for any enthusiastic reader who wants to explore the mysteries of speech and language.

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# PROLOGUE

> "Language is beginning to submit to that uniquely satisfying kind of understanding that we call science, but the news has been kept a secret."
>
> Steven Pinker 1994.[1]

For centuries, scholars have discussed the mechanisms and seats of the psychic phenomena that comprise the mind. However, it was not till the eighteenth century that there was proof of cerebral localisation of these functions (Walker AE 1957,[2] Tandon and Chandra 2022[3]). Though not directly related to the subject of this monograph, i.e., the role of the human brain in the perception and production of speech and language, if real progress is to be made on the subject, a multidisciplinary effort would be necessary to advance knowledge further. This would require coming together of scholars from different disciplines-neuroscientists, psychologists, clinicians, imaging specialists, linguists, anthropologists, philosophers, and computer scientists-to further the frontiers of knowledge on the subject under review.

Only three to four decades ago, a new science was born – now called "Cognitive Science"-combining the tools of behaviour scientists, anthropologists and philosophers, computer science, linguistics, and neurobiology. To this were added the tools and techniques of non-invasive neuroimaging-CT, PET, fMRI, Magneto-encephalography, electrophysiology, event-related potentials and connectomics to enable exploring the working of human intelligence.

In recent years, scientists like Chomsky[4-9] and Pinker,[1,10] Penfield W,[11-16] Geschwind,[17-22] Critchley EM,[23-32] Nadeau SE,[33-38] Kuhl PK,[39-48] Dehaene-Lambertz *et al*.[49-57] have been the persons most responsible for the modern revolution for exploring the structure & function of the brain, the neural substrate of language and human cognition.

It is not surprising that while language has been a subject of detailed study and analysis for many millennia, it has only recently become a subject of a multidisciplinary knowledge system. It is now recognised that language is more complex than just an anatomic brain region. It involves thought, perception, emotions, speech, and writing (Lea WA 1980).[58]

Speech, on the other hand, is the vehicle of language and involves the act of producing specific sounds using the articulating system controlled by the primary motor cortex.

Language is a complex, specialised skill which develops in a child spontaneously without conscious efforts or formal instruction and is deployed without awareness of its underlying logic (Pinker S 1994).[59] Scott SK and Johnsrude IS (2003)[60] considered speech conceptually distinct from language, which, with its syntactic and generative aspects and highly elaborated semantic structure, is likely to be unique to human language.

Terms like "phonemes", "phonology", "graphemes", and "lexicography" constitute the essential elements of a language. It has its own rules, conventions, and grammar, which enables human communities to understand each other's communication-vocal, written or signs.

Language is learnt while an infant is born with vocalisation, which is influenced initially by the mother and later by social influences. Considerable language learning is already taking place in the first year of life in phonology, prosody and word segmentation (Barker BA and Newman RS 2004,[61] Dehaene-Lambertz G 2011[52]).

Surprisingly, millennia-old literature provides unequivocal evidence that scholars of that era were not only aware of these elements of speech and language but contributed to advancing knowledge on these subjects. This included Greek, Roman, Egyptian and Indian scholars (Tandon and Chandra 2022[3]).

However, being familiar with ancient Indian literature, an opportunity is taken to provide extracts from a scholarly write-up of the subject by Bose, Sen and Subbarayappa in their book "A Concise History of Science in India" (1975)[62] [See Annexure 1].

In his book "The Descent of Man", Darwin C [63] concluded that language ability is an "instinctive tendency to acquire an art". This view supports Pinker's (1994) recent book, "The Language Instinct: How the Mind Creates Language" (Penguin Books, Penguin Random House U. K (2003).[10]

The students of science, mathematics, biology, medicine, and neurosciences (basic and clinical), like the author of this monograph, who routinely dealt with issues related to speech and language, are mostly ignorant of the knowledge of linguistics. The reverse of this is also true that linguists who, while analysing language, overlook the scientific relevance of their knowledge.

"Loss of ability to use language was recognised long before Broca described it as being due to localised brain lesion. And almost all forms of dysphasia had been described before 1800 Critchley M(1930).[26]

Notwithstanding the general acceptance of Broca's description of what came to be called Broca's aphasia, Mohr JP *et al*. (1978)[64] had convincingly demonstrated that lesion confined to the third frontal gyrus was not sufficient to produce Broca's aphasia and that this type of aphasia usually occurs with extensive lesions (this was confirmed by Tonkonogy and Goodglass H 1981;[65] Patterson K *et al*., 1984[66]).

Nadeau SE (1988)[37] affirmed, "The destruction of the third frontal gyrus does not produce lasting linguistic impairment that is readily available at the bedside. He adds, "We still do not know what the linguistic function, if any, it does". Alexander MP and Hillis AE[67] speak of Broca's area aphasias as not a single syndrome.

Attempts have been made in this book to bring together knowledge from diverse disciplines to provide a comprehensive view of this unique human trait. Recent advances in cognitive neurosciences and recent neuroimaging techniques have been a rich source of advancing our knowledge, confirming and challenging the existing knowledge, and pointing to new directions for future studies.

This book is meant for much larger groups than a few limited specialists, psychologists, neurologists, neurosurgeons or cognitive scientists on one hand and linguists, behaviour scientists and other explorers of the human brain.

**Annexure 1: Some extracts from ancient India Literature on language**

Ancient Indian Literature, Pre-Vedic, Vedic, Buddhist, etc, were subject to deep analysis from 1500-2000 BC. It provides enough evidence that speech and language were the subjects of deep intellectual exercise, but the neural basis was not even considered.

## Chronology of the Vedic Literature

It provides a deep insight into the chronology of the Vedic Literature, and considering that it must be pre-Buddhist, Max Muller suggested the period between 600 and 200 BC for the development of the *Sutras* (work synchronous with the origin of and spread of Buddhism), the period between 800-600 BC for the development of the prose style of the *Brahmanas*, *Aranyakas* and Upanishads. The period 1000 to 800 BC for the compilation of *Samhitas* of which the poetry or the mantras probably originated in the period between 1200 and 1000 BC.[68] In his view, the oldest of the Vedas, the Rigveda, could not have been composed earlier than 1200 BC. Von Schroeder L [69] suggested a much earlier date, 1500 or even 2000 BC, for Rigveda, while Jacobi HG and Tilak BG,[70,71] on astronomic grounds, tried to date the Vedic literature in the third millennium BC.

## The *Vedangas*, the *Sutras* and the Origin of Sanskrit Scientific Literature

*Vedangas* deal separately with six special branches of knowledge, viz phonetics (*Siksa*), ritual (*Kalpa*), grammar (*Vyakarana*), etymology (*Nirukta*), metrics (*Chandah*), and astronomy (*jyotisa*). These branches of studies arose within the Vedic schools as a necessary condition for mastery of the Vedas. Being the work of human specialists, these are called *Vedangas* or auxiliary sciences of Vedas.

### *Sutras*

Discussing sutra style or highly condensed aphoretic style, Winternitz M (1959 cited also in 1981)[72] pointed out that "*From the point of precision and brevity, there is probably nothing like these Sutras of the Indians in the entire literature of the world. This style of writing was adopted by various philosophical schools, the grammarians, phonetics and the writers of the Arthasastra, the Kamasastra, the natya sastra and so on.*"

While Panini's date is itself debatable most scholars generally agree that Panini lived in the 4th century BC (C 350 BC, according to Keith AB (1976).[73]

A treatise called *Astadhyayi* is divided into eight chapters. He gave formal production rules and definitions to describe Sanskrit grammar. According to Cardona G (1976), Panini's[74] grammar has been evaluated from various points of view.

### Grammar

Although a long line of grammarians must have existed before Panini (C 350 BC), the *Astadhyayi* possibly rendered the efforts of his predecessors obsolete. Written in 4,000 short *Sutras,* the works deal with technical terms, nouns in compositions and case relations, rules for adding suffixes to the roots, and so on, always from the point of view of the language and using it correctly in composition.

### Acknowledgment

Most of the information in Annexure 1 has been extracted from reference 63, Bose et al., published by the Indian National Science Academy. This publication provides very rich information on the subject.

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# Speech, Language and Human Brain: Revisited

## Historical Review

The association of right hemiplegia and speech disturbance had been reported by Goethe JW (1796, cited in his article, 1795)[1] and also cited by Goodglass and Quadfasel FA1954[2] and later by Bluhme (1970).[3] However, the idea of localizing speech in the left hemisphere was first presented at a lecture in 1836 by Marc Dax (cited by Buckingham HW, 2006).[4] However, the real credit for the first publication on this subject goes to Pierre Paul Broca (1824-1880).[6] Based on a single case carefully followed clinically at Pitie-Salpetrier hospital, Paris and studied at autopsy, he presented in 1861, before the Society Anthropologie (which he had founded in 1859), a lesion in the left frontal lobe of his patient who suffered from *aphemie* (later named aphasia by Trousseaus in the same year). It was published in 1865 in the journal of the Anthropology society.[6] The area was later called "Broca's Convolution" by Schiller F(1999),[7] (Tandon and Chandra 2022).[5] Broca further defined this area to the posterior part of the third frontal convolution, especially on the left, which was essential for the faculty of speech. Wernicke C[8,9] (1848-1904) described sensory aphasia (word deafness) in 1874, due to a lesion in the superior temporal gyrus on the left side. His first book on aphasia was also published in the same year. Thus in later half of 19th century and earlier years of 20th century study of speech disorders and their cerebral location involved a large number of neurologists, psychologists and neuroanatomists. Instead of the initial classification of these disorders (dysphasia/aphasia) into motor and sensory the variety of these disorders (syndromes) multiplied and attempts were made to find their cerebral location based on careful clinical studies, syndromes like word blindness, word deafness, agraphia, transcortical sensory, transcortical motor dysphasia, paraphasia, dyslexia, pure alexia, agraphia, conduction aphasia, acalculia, disconnection aphasia were added to the speech & language lexicon. As a matter of fact Alexander MP *et al*.[10-13] has recently referred to eight classical aphasia syndromes. Most of these were no doubt located in the left hemisphere, *though not restricted to the Broca's or Wernicke's areas, and some even to the right hemisphere*. Some of the pioneers in the field, included. Dejerine J,[14] Marie (cited by Brais B, 1992),[15] Charcot[16] (cited by Engelhardt E, 2015), Pitres from France (cited by Lorch and Barrière, 2003),[17] Wernicke C,[18] Lichtheim,[19] Liepmann (cited by Dalfardi B *et al.*),[20] Pick (cited by Roelofs A, 2023),[21] Henschen S,[22-24] Goldstein K[25] and Kleist (cited by Mehigan, 2011)[26] from Germany; Jackson (cited by Head, 19121[27] Gowers (cited by Tyler, 2003),[28] Head H[27,29-31] among others from England made valuable contributions in the field. According to Binder J (2000)[32] as early as 1888, Charcot and his student Marie included the left angular gyrus and the middle temporal gyrus in the region associated with Wernicke's aphasia. Marie also included submarginal gyrus as well. Starr MA (1889),[33] reviewing 50 cases of sensory aphasia published in the literature, 27 of whom had Wernicke's aphasia, reported none of them were found to have lesions restricted to the superior temporal gyrus at autopsy the lesion involved much wider regions involving temporal parietal and occipital convolutions modern neuroimaging has confirmed many of these observation us summarised by Binder JR (2002)[34] in his extensive review "Wernicke's aphasia: A disorder of central language processing".

Wise RJ *et al.* (2001)[35] have further analysed, the distribution of separate neural subsystems within Wernicke's area.

In 1880 Jackson JH [36] from National Hospital for Paralysed and Epileptics, Queen Square, London, localized both propositional and emotional speech to the left cerebral hemisphere (which he called "the leading hemisphere") and of emotional speech alone in the right hemisphere.

Based on a detailed survey of the literature, Goodglass and Quadfasel FA (1954)[2] opined, "that there is cerebral dominance as a property of one hemisphere. The dominant hemisphere now was assumed to control the preferred hand, often the preferred eye and foot and for damaged, to produce aphasia". Handedness, was now used as evidence of dominance of the hemisphere opposite to the preferred hand.

Brain WR (1945)[37] in a review on "Speech and Handedness" mentioned that, "The cerebral hemisphere in which are situated the neural pathways of speech has become known as the dominant or major hemisphere, it is the left hemisphere in the right-handed person". He goes on to question himself "Are the speech centres ever situated in the right hemisphere in a right-handed person? Answering in affirmative "of course", he quotes Gardner's report of a case in this connection (Gardner WJ 1941[38]) [See later].

Before, discussing the details of hemispheric dominance, handedness and transferability of speech to the other side, since it has acquired an awe-inspiring position in clinical practice (to be elaborated later), it is worth looking at the dictionary meaning of the word. It means " having power and influence over others". Its synonyms reinforce this concept further: presiding, ruling, governing, controlling, commanding, ascendant, supreme, authoritative, superior, prepotent, assertive, self-assured, forceful. It will be noted that the term "dominant" as used in clinical practice hardly conforms to the word as enumerated in the dictionary. Yet the fear of producing loss of speech following surgery on the presumed dominant hemisphere made it generally, out of bounds for the neurosurgeons as is obvious from some of the text books of neurosurgery.

Pástzor (1990),[39] while advocating surgery for brain tumours stated," we do not operate when the central region, the area of Broca and Wernicke centre or deep midline structures are infiltrated". Similarly, Garfield (1986)[40] in chapter on malignant intracranial tumors commented that, "Traditionally, severe and distressing focal deficit such as aphasia has been regarded as a contra-indication to attempt tumour removal".

A "Pubmed Search" in 1984 for publications on surgery for dominant hemisphere supratentorial gliomas for previous six years failed to reveal any sizeable series. The same year at the International Symposium on Biology of Brain Tumours, at London, we presented our experience with radical surgical decompression on 500 patients with supratentorial gliomas (nearly equal numbers involving dominant and non-dominant hemisphere.(47.4% right hemisphere, 44.7% left hemisphere; Tandon *et al.* 1986[41]). This unequivocally established that not only, any distressing aphasia did not result inevitably from surgery on the dominant hemisphere, the preoperative dysphasia improved in 50%. cases. A further follow up of 100 such patients who survived for one year or more revealed among 58 of them who had signs of speech deficit preoperatively, 65% improved, it remained the same in 1 and deteriorated in 11. Even among those whose speech deteriorated none developed aphasia: (Tandon *et al.* 1993). As late as 2012, Easwaran[42] in a Textbook of Neurosurgery commented, "The risk of neurological worsening is foremost in the mind of the neurosurgeons attempting radical excision of gliomas. The fear is profound in dealing with lesions of the dominant hemisphere."

It may be mentioned that somewhat similar results were also reported, in few other publications based on limited number of patients. Zollinger (1935)[43] on the basis of a single case report stated that even hemispherectomy of the dominant hemisphere for gliomas did not abolish, but severely restricted, speech. Salcman (1985)[44] claimed that, "It is now possible to safely remove large glial tumours from virtually any hemispheric location without significant impairment of the patient". Similar opinion was expressed by Hillier (1954),[45] Brasser (1964),[46] Smith and Burklund (1966)[47] and Burklund and Smith (1977).[48] The purpose of the above description is not to minimise the role of the left cerebral hemisphere in speech and language function but to point out its limits.

Since 1861, observations by Broca on speech localization in the left cerebral hemisphere, for almost 100 years our knowledge of the cerebral localization of speech was limited to knowledge gained from correlation of clinical observations with postmortem findings. In the past couple of decades advances in diverse fields like linguistics, psychometry, cognitive science on one hand and fMRI, PET Scan, EEG, Evoked Potentials have made it possible to explore in details in vivo the correlation of various aspects of speech and language and precise brain regions involved, in healthy subjects from birth to adulthood.

Démonet *et al.* (1992)[49] on the basis of anatomic study of phonological and lexico semantic processing in normal subjects, using PET Scan indicated the different location of the two major components of auditory comprehension of language. Phonological processing was associated with activation in the left, superior temporal gyrus (mainly Wernicke's area). and to a lesser extent, in Broca's area and in the right Superior temporal regions. Lexicosemantic processing was associated with activity in the left middle and inferior temporal gyri the left inferior parietal region and the left superior prefrontal region, in addition to the left superior temporal region.

Analysis of lesion sites in aphasic patients have suggested that, in right-handed subjects, phonological processing is mediated by neural structures close to the left sylvian fissure such as Wernicke's area, the insular cortex or the Supra-marginal gyrus. By contrast, lexicosemantic process seems to involve more widely distributed regions of association cortex in the left hemisphere (Geschwind 1968[50]) such as angular gyrus, the posterior and inferior parts of the temporal lobe or even the dorsolateral prefrontal area. However, Wise *et al.* (1991) Suggested that a considerable overlap may exist between the activated areas when the tasks are theoretically different (e.g. words versus non-words).

The authors observed that, " although clinical evidence as well as our findings indicate that the major part of language processing is left-sided, it may be that the right superior temporal areas, which are anatomically connected to the homologous areas on the left are co-activated when cognitive processes because more complex.

Binder (2000)[32] in an editorial on "The new neuroanatomy of speech perception" concluded, that functional neuroimaging Studies indicate participation of multiple areas, including the middle and inferior temporal gyri, fusiform gyrus, angular gyrus and the frontal lobe during auditory word recognition.

Using PET imaging to identify, separable neural subsystems within the human cortex demonstrated that the left superior temporal suleus responds to the presence of phonetic information, but its anterior part only responds to if the Stimulus is also intelligible.(Wise *et al.* 2001[35]).

The term intelligible covers several properties of language, including word-form recognition, Syntax and semantics, No one acoustic cue determines the intelligibility of speech.

They proposed that intelligible speech is associated with an anterolateral stream of neural information from the primary auditory cortex. The left superior temporal gyrus ventrolateral to the primary auditory cortex was activated equally by speech, rotated Speech and noise-recorded speech-Anterior and ventral to this, the superior tempore sulcus was activated by intelligible speed only. In the human being the monosynaptic connections from the primary auditory Cortex Cove directed towards the lateral and anterior auditory association cortex. It has been observed that lesions constrained to this region result in result in conduction aphasia in which the patient can comprehend speech but not repeat words (Hickok 2000).[51]

For the speech and rotated speech. Stimuli, there was increased activities. In the right ventrolateral Superior temporal gyrus anterior by the primary auditory cortex.

"By demonstrating distinct neural Subsystems in the auditory processing of speech in the anterior-posterior axis of the temporal lobe, our results are also relevant to understanding the consequences of the location and extent of the left temporal lobe strokes on the recovery of Comprehension. Although the anatomical boundary of Wernicke's area have become too broad to be meaningful, most neurologists and neurophysiologists locate. The core Wernicke's area in the superior temporal cortex posterior to the place of that primary auditory cortex (Galaburda *et al.* 1978[52]). Furthermore, access to the word meaning is considered to be a function of the Wornickes area.

In an editorial in Brain in 2000, Binder,[32] began his write-up by a thought provoking, not generally known, Statement, "Our understanding of speech recognition processes has gradually advanced over the past 50 years, from a state of almost total ignorance to one of well-informed. confusion: And "Until recently, the Conventional neuroanatomical model of speech perception had changed little from the one proposed by Wernicke in 1874"

In the past decade, scientists using functional neuroimaging have repeatedly observed a superior temporal region in both hemispheres that activates more strongly to speech than to non-speech sounds like tones and noise (Demonte 2019,[53] Zatorre *et al.* 1992,[54] Binder *et al.* 1996,[55] 2000,[56] 2017[57] Scott *et al.* 2000,[58] Shannon *et al.* 1995[59]).

Finding from these (neuroimaging) studies indicate participation of multiple areas, including middle and inferior temporal gyri, fusiform gyrus, angular gyrus and frontal lobe during auditory word recognition. The superior temporal system specialized for Speech Sound Recognition is but an early stage in a processing stream that ultimately project to all component of the distributed system (Wise *et al.* 2001).[35]

Bogen and Bogen (1976)[60] stated that "In absence of clear definitions about either its functions or its anatomical boundaries, Wernicke's area' has become a meaningless concept". The authors analysed four functional. neuroimaging (PET) studies to identify anatomically separable, functional Subsystems in the left superior temporal cortex posterior to the primary auditory cortex, (classically considered to be the Wernicke's area). They referred to three distinct architectonic zones in the left temporal and parietal lobes, where the output from both heard and written words forms (lexical) systems converge (Mesulam 1998[61]). The authors found that the three PET studies demonstrated a conjunction of activity the posterior left superior temporal gyrus in response to hearing single words and during cued word retrieval. They postulated that this local system transiently, represents the temporally ordered sequence of sounds that comprise a heard (external) or retrieval (internal) word and that it acts as an interface between the perception and long-term mental representations of familiar words. A fourth PET study demonstrated an adjacent local system at the medical left temporal *et al.* junction that acts as an interface between posterior temporal cortex and motor cortex for speech.

These two anatomically and functionally separable regions are candidates for systems that must exist to allow us to perceive and rehearse novel words until they are acquired as retrievable lexical memories.

It is now recognized that speech perception and production is a complex psychophysical process which consists of a number of neuroanatomical subsystems as distributed processing networks.

Thus, clinically speech disorder may manifest as any isolated disturbance of any single component or a combination of these as a syndrome depending upon the size and site of the lesion.[32,55-57] Clinico pathological correlation may provide misleading information about the precise location of the lesion.

In recent years studies on normal healthy subject using PET (Chugani *et al.*,1987;[62] Dehaene-Lambertz *et al.*, 2002;[63] 2006;[64] Gaillard *et al.* 2000;[65] Pujol *et al.* 1999;[66] Pfefferbaum *et al.*, 1994,[67] Imada *et al.*, 2006[68]) fMRI, magnetoencephalography (MEG), event related potentials, near infrared spectroscopy (Bortfeld *et al.*, 2007[69]) have been used in infants as young as few months old to study neural mechanisms involved in learning language have focussed on the neural basis of processing speech sounds. A large number of such studies, brain activation states measured during presentation of speech sounds in contrast to no sounds consistently and robustly activated the superior temporal gyrus bilaterally (Binder 2000,[32] Howard *et al.* 1992,[70] Hirano *et al.* 1997[71]). Thus, besides the classical motor (Broca's) and sensory (Wernicke's) aphasia a variety of speech disorders were clinically identified. This included disconnection aphasia, transcortical motor aphasia, transcortical sensory aphasia, conduction aphasia, optic aphasia, dynamic aphasia, amusia, pure-alexia, deep-alexia, alexia without agraphia, agraphia, acalculia, and word-deafness.

Alexander and Hillis (2008)[72] described eight classical aphasic syndromes. Speech itself was assessed in respect to phoneme, graphene, prosody, paragraphic graphene to semantic features. (Blumstein *et al.* 1980,[73] Blesser 1972,[74] Damasio and Damasio 1980,[75] Binder 2002[34]).

It became obvious that it is not just the site and extent of the cortical lesion which determines the characteristics of speech disorder syndrome, but the also the extent of interconnecting network. Binder (2003)[56] Critchley (1953),[76] Robinson *et al.* (2001),[77] Gitelman (2003),[78] Kahn and Whitaker (1991),[79] Kaczmarek (1984);[80] Nadeau (1988),[81] Nadeau and Crosson (1995),[82] Costlett (2003).[83]

Pick was the first to put Wernicke's ideas of aphasia on a sound pathoanatomic basis and his studies on the subject and on apraxia and agrammatism were published in his book, "Die agrammatischen Sprachstorungen: Studien zur psychologischen Grundlegung der Aphasielehre, Berlin, Springer 1913.[84]

Interestingly, Pierre Marie (1857-1940, cited by Webb-Johnson, 1953)[85] attacked Broca in his paper, "The third left frontal convolution has no special role in the function of language?

## Recent Studies on Development of Speech Areas in Humans

For nearly one hundred years after the localization of speech to the left hemisphere by Broca (1861) and Wernicke (1874), except for a large number of clinic-pathological studies, dissecting the various speech syndromes and their cerebral localization (mentioned above), there was little known about the development of neuroanatomical substrate during foetal life, neonatal period and infancy and its comparison to adults. Kuhl (2011)[86] acknowledged, "We are still just breaking ground with regards to the neural mechanisms that underlie language development and its critical period. In the last decade, brain and behavioural studies indicate a very complex set of interacting brain systems in the initial acquisition of language early in infancy, many of which reflect adult

language processing (Dehaene-Lambertz *et al.* 2006).[64] Little was known about when and how functional lateralization (to the left hemisphere) developed in human brain. Geschwind and Levitsky (1968),[87] reported left-right asymmetries in the temporal speech region in 100 adult brains obtained at post-mortem. They found marked anatomical asymmetries between the planum temporale of the two sides, it being larger on the left in 65 percent of brains and only in 11 percent on the right. The planum temporale contains auditory association cortex which extends to the lateral surface of the posterior portion of the first (superior) temporal gyrus-the area well-known to be the site of lesion of Wernicke's sensory aphasia.

On the basis of their observation of electric response obtained from this area, Penfield and Roberts (1959)[88] considered it to be a part of the speech function.

Wada *et al*. (1975)[89] studied cortical speech zones in 100 adults and 100 infant brains. They observed that morphological asymmetry of the frontal opercular (Broca's area) and temporal planum (Wernicke's area) becomes measurable at the 29$^{th}$ week of gestation. They found evidence of subsequent differential development of the planum in favour of the left, with the left planum larger than the right. While both the frontal operculum and the left planum were always present, the right planum ranged in size from absent (10%) to larger than left (about 10%.). They considered these findings to suggest that a higher percentage of persons may have right sided cerebral speech dominance or bilateral. cerebral representation of speech far more than has been assumed previously. The presence of measurable morphological asymmetry as early as 29$^{th}$ week of gestation in cortical areas known to be involved in the processing of speech and language function suggests that the human brain possesses a predetermined morphological and functional capacity for the development of lateralized hemispheric function for speech (dominance!).

Branch *et al*. (1964)[90] using intracarotid sodium amytal test (Wada's Test) in 123 subjects for lateralization of cerebral speech dominance concluded that speech functions are localized in the left hemisphere in most adults regardless of their handedness.

These findings were confirmed by Witelson and Pallie (1973)[91] who measured the superior surface of the temporal lobe (planum temporale) of a group of 14 neonatal and 16 adults human brain specimens. The left sided planum area was statistically significantly larger in the neonates as in the adults. This indicates that the asymmetry implies that the infant is born with a pre-programmed biological capacity to process speech sounds, and may be an important factor in determining the typical pattern of left hemisphere speech lateralization found in most adults.

These anatomical observations find support from behavioural studies by Eimas *et al.* 1971,[92] Trehub and Rabinovitch 1972.[93] Speech perception was found in an 1 year and 4 month old infant. They were able to make fine distinction approximating the manner in which adults perceive same sound, thus confirming the biological basis of this function, Chi *et al.* (1977)[94] in a large series of human brains from infants of 10 to 44 gestational week found left-right asymmetries of the transverse temporal gyri, Sylvian fissures, planum temporale, these being longer. the left side, Surprisingly, in general the right cerebral hemisphere shows gyral complexity earlier than the left (?significance) On the right side the superior frontal and superior temporal gyri, were first visualized approximately one to two weeks earlier than the left side. This has not been confirmed by others, nor is its functional significance discussed.

As a matter of fact, DeCasper and Fifer (1980)[95] claimed that human responsiveness to sound begins in the third trimester of life and by birth reaches sophisticated levels, especially with respect to speech. More recently Dehaene-Lambertz *et al*. (2002)[63] utilizing functional neuroimaging (fMRI) in 3-month-old infants found left side brain regions similar to those of adults, including the superior temporal and angular gyri were already active in infants. Additional activation in right prefrontal cortex was seen in awake infants processing normal speech. These studies were carried further to explore functional organization of perisylvian activation during presentation of sentences to 3 months old preverbal infants. It was observed that perisylvian areas are activated by speech early in life with a well-defined temporal structure. The fact that Broca's area is active in infants before the babbling stage implies that the activity in this region is not the consequence of sophisticated motor learning but on the contrary, that this region may drive, through interaction with the perceptional system, the learning of the complex motor sequence required for future speech production (Dehaene-Lambertz *et al*. 2002, 2006, Dehaene-Lambertz 2011).[63,64,96]

Dehaene-Lambertz (2011)[96] summarising their group's studies extending over a decade concluded, "The methods of brain and cognitive sciences have reached a stage such that we can, now objectively monitor the developmental trajectory of the child's brain. Non-invasive brain imaging methods can now be used together with behavioural measurements to examine the development of infant cerebral and mental organization and its growth. The results reveal both a highly structured early organization of brain networks for language, with hemispheric specialization, and it's very fast maturation in the first months of life"

They observed, "Brain maturation continues in adolescence and early adulthood with remarkable changes in the dynamic interaction of distributed brain regions. The initially rather diffuse networks became more segregated and focused. The genetically layout of the connection architecture provides a universal neural platforms, which will be later shaped by specific cultural experiences (Dehaene-Lambertz and Baillet 1998,[97] Dehaene-Lambertz *et al.* 2002,[63] Dehaene-Lambertz 2011,[96] Meyer *et al*. 2012).[98]

According to Johnson (2001)[99] the functional specialization of regions of the cerebral cortex arises through intrinsic genetic and molecular mechanisms and that experience merely has a role in the final fine turning "However there is evidence to suggest that some aspects of human functional development involve a prolonged process of specialization that is shaped by postnatal experience (Neville *et al*. 2015).[100]

Anatomical asymmetry in language area is clearly present at an age when bilateral representation and hemisphere

transferability of speech are still present (Basser 1962,[101] Rasmussen 1964[102]). It is suggested that this anatomical asymmetry precedes any learning effect. Exploring functional anatomy of cognitive development.

Gaillard (2000)[103] using fMRI to evaluate verbal fluency in children (mean age 10.7 years) and adults (mean age 28.7 Years) confirmed the predominant neural networks underlying language functions lie in the left hemisphere in nearly all normal adults. However, a few normal right-handed individuals had right hemisphere dominate for language, either as a normal variant or because disease and neuronal injury early in life led to shifting of language from left to the right hemisphere. Some degree of bilateral activation (e.g., in the right hemisphere analogs of Wernicke's and Broca's areas) were seen in fMRI studies. Pujol *et al.* (1999)[66] in a similar fMRI study had also reported 4% of right-handed had greater right than left activation.

Human language learning has sometimes been viewed as being modularized (Fodor 1983),[104] but there are substantial arguments against this interpretation (Fitch 2010).[105] Although there are areas of human brain important to speech production and comprehension, the speech control system is not as localized and highly structured as expected for a Fodorian module. Speech production and comprehension also do not appear to be computationally autonomous. Language learning is positively associated with other cognitive abilities.

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# What is Speech? What is Language? What is Their Neural Substrate?

> "Having a language, of course, is part of what it means to be human."
>
> Steven Pinker 1994.[1]

The reader of this book will be surprised to learn that the questions mentioned above arose in the author's mind only after he had reviewed a substantial amount of literature on speech and language, mainly contributed by biomedical scientists, and most of the chapters of the monograph were already written. The reason was that most clinicians and bio-medical scientists used the two terms as synonyms. Having practiced and taught clinical neurosciences-neurology and neurosurgery-for half a century, the author hardly systematically evaluated language in speech disorders, even those with various aphasias. Various examination systems failed to assess the details of linguistic nuances. However, in recent years, with advances in neurolinguistics on one hand and non-invasive neuroimaging in healthy volunteers and patients with different types of brain damage, it became apparent that though these terms were often used as synonymous, there are subtle differences between speech and language and so are the differences in their neural substrate. However, it was still generally felt that keeping their differences in mind, there is so much commonality between the two that it is reasonable to discuss them. Notwithstanding, this general practice prompted the author to study the subject in detail for scientific accuracy and possible future knowledge.

Initially, a search in Chamber's Everyday Dictionary proved to be of little help. Thus, it defined *Speech* as what is spoken and *Language as the* power of speaking: manner of speaking, oration discourse. It also mentions language as human speech: a variety of speech or body of words and idioms, especially that of a people, made of expression, or any manner of expressing thought (including sign language). This dictionary definition of speech and language didn't clearly distinguish between the two for a person brought up using unambiguous definitions of the subjects of investigation. This led the author to seek a linguist's help and further explore the literature from this point of view. What follows is the outcome of this effort. It was felt that including a separate chapter on the subject would be helpful for clinical and basic neuroscientists and may be for neuropsychologists, also.

It is worth pointing out that except for song birds who manifest some form of speech, only humans are blessed with the capability of speech and language. Even the highest non-human primates have vocalization but not speech. It is well-known that much lower animals have some means of communication with each other but have no speech; they may even vocalize for this purpose, and undoubtedly, they have no language. Even plants were shown to communicate with each other (Tandon 2019).[2,3] The newborn human infant does not have speech or language, which evolves from vocalization to verbalization during development.

**Some scientifically applicable definitions of speech and language are collected from various sources, including Wikipedia and literature on linguistics:**

Speech is a form of verbal communication used by humans to convey thoughts, ideas, and information to others. It involves producing specific sounds using the articulatory system controlled by the primary motor cortex. Its building blocks are known as phonemes, which form the basis of spoken language.

Speech and language, though often considered synonymous, are different elements of communication. Speech is the vehicle of language. Language is a complex and well-defined communication system used by a specific community of human beings to express their thoughts, ideas and emotions. It has rules, conventions, and grammar, which enable them to understand each other's communication-vocal, written or sign. Unsurprisingly, the English spoken by British, Americans, and Indians is not entirely identical, yet specific groups easily understand it.

Factors such as accent, intonation, pitch, rhythm, and articulation vary considerably between individual groups, regions and cultures, providing the unique qualities of the personal and regional dialects. Because of this, when a friend rings up after several years, we can still recognize him based on the characteristics of his voice. However, very little is known about the neural basis of these characteristics. While these characteristics are of great significance for speech pathologists and speech therapists, these have not been of special interest to neurologists.

The development of speech depends a great deal on the auditory function, and deaf-mutism is a classic example of this (Belin *et al.*, 2000).[4] Infants born with hearing disorders due to cochlear defects progressively develop speech impairment if cochlear implants are not provided early enough. Neurologically speaking, speech consists of two functional elements, e.g., production and reception, with their separate neural substrate.

Scott SK *et al*. (2003)[5] considered speech conceptually distinct from language, which, with its syntactic and generative aspects and highly elaborated semantic structure, is likely to be unique to humans. Language encompasses several key components: phonetics and phonology, morphology, syntax, semantics, and pragmatics.

Phonetics and Phonology: deal with the sounds of language. Phonetics is concerned with the physical properties of sounds, while phonology is concerned with how sounds function within a particular language.

Morphology deals with the structure and formation of words. It analyses the most minor meaningful units, called morphemes and studies how they combine to create words (word structure).

The above two are the matter of study by linguists, while the following are already interesting for neurologists.

**Syntax**: is concerned with how words are combined to form sentences (Sentence Structure).

**Semantics**: deals with the meaning of words, phrases and sentences. It encompasses the study of the relationship between words, such as synonyms, antonyms and associations. (meaning) Language can vary in regions and cultures, and communication can result in dialects. Language is the mental faculty that we use to communicate-It involves associating sounds and symbols with meaningful concepts and enables us to describe our external environment and abstract thoughts. The effective use of language requires the interaction of memory with sensory inputs and motor output systems. The principal types of memory necessary for language are phonological (the sounds of words), orthographic (the spelling of words) and semantic (our knowledge of the world).

Sensory input to these memories can be via auditory processing (for spoken words, environmental sounds and music, Berlin *et al.*, 2000[4]), visual processing for written words, objects, faces and signs) or tactile processing (braille). The motor output enables the expression of concepts via articulation, writing, signing or drawing. It can either be self-generated (in response to internally generated thought) or stimulus-driven (e.g. in response to written or heard words) (Price CJ 2000).[6]

Language is generally used vocally in writing, such as alphabets, logographic systems, or syllabaries. Sign language has already been developed for routine use in communicating with the hearing impaired, and it is routinely used in some countries for news telecasts.

Social inputs greatly influence language development in its early stages. An extreme example is an infant lifted away by a wolf and reared by the wolf family when it was discovered several years later that wolf sounds were the only speech/ language he used.

Language disorders affect the understanding (sensory) or expression (motor) aspects of language, as observed in patients with aphasia or speech apraxia and developmental language disorders (DLD). These have been studied ever since the initial description by Broca and Wernicke. Clinicopathological studies have led to recognising several syndromes characterized by only a part of the original syndromes or a combination of the two. However, these studies failed to reveal the neural basis of speech and language development beginning in the fetus in utero passing through various stages till full maturity in adolescence.

During the last couple of decades, the coming together of investigators specializing in neurolinguistics, experimental neuropsychologists, behaviour scientists, neuroscientists on one hand and clinicians, neurophysiologists and neuroimaging experts on the other has resulted in clinical-neuro anatomical correlations unimagined earlier. An attempt has been made in this book to highlight this new knowledge compared to what was known.

The annexure provides brief notes on some papers that illustrate recent studies on neuroanatomical correlates of the various constituents of speech and language. Vigneau M *et al*. 2006[7] reported phonologic, semantics and sentence processing areas in a meta-analysis of left hemisphere language areas.

## Annexure 1

**Definitions** of relevant terms used by linguists, neurologists and cognitive scientists.

**Phonemes** are the range of vocal sounds we use for speaking.

**Phonological Process** are those involved in the perception or production of phonemes.

**Graphemes** are the visual symbols that depict one or more phonemes.

**Orthography** refers to grapheme combinations that constitute the spelling of the words.

**Lexical Processing** refers to that concerned with whole words.

**The auditory input lexicon** stores the "auditory images of words".

**The orthographic input lexicon** stores the "Visual images of words.

**The orthographic output lexicon** stores the "motor images for articulating words".

**The orthographic output lexicon** stores the "motor images for writing words".

**Semantic memory** is our knowledge of the word.

**Lexico semantics** concerns the conceptual meaning of words.

**Sublexical process** concerns those related to the parts of words.

## Annexure 2

To quote some examples of multidisciplinary studies (most of these are described in some detail in different chapters).

1. Dehaene-Lambertz G, Dehaene S, Hertz-Pannier L. Functional neuroimaging of speech perception in infants using fMRI (Science 298, 2013-2015, 2002).[8]
   This is one of the earliest papers to establish that human infants begin to acquire their native language in the first months of life.
2. Dehaene-Lambertz G: Pannier, LH, Dubois J: Functional organization of perisylvian activation during presentation of sentences to preverbal infants (PNAS 103, 14240-14245, 2000).[9]
   The authors studied ten full-term infants between 11 and 17 weeks after birth. They found that perisylvian areas

are activated by speech early in life with a well-defined temporal structure and capacity for memorizing sentences. Though details of precise aspects of this organization could not be ascertained, they observed that the infant behavioural repertoire in the language domain includes categorical perception of phonemes, recognition and long-term storing of intonation contours and episodic verbal memory.

3. Goldman-Rakic PS: Development of cortical circuitry and cognitive function. Child Dev 58, 601-602, 1987.[10]
   The acquisition and consolidation of new skills during childhood and adolescence occur with a synaptic density and efficiency change.
4. Galliard WD, Hertz-Pannier, L, Mott SH *et al*.: Functional anatomy of cognitive development: fMRI of verbal fluency in children and adults (Neurology 54, 180-185, 2000).[11]
   Here, fMRI studies were conducted to identify age-dependent activation patterns of verbal fluency in children and adults. Both activated similar regions of the brain, predominantly the left inferior frontal cortex (Broca's area) and left middle frontal gyrus (dorsolateral pre-frontal cortex). The children had, on average, a 60% greater extent of activation than adults. The children also had significantly more right hemisphere and inferior frontal gyrus activation than adults. The activation pattern for fluency appears to be established by middle childhood.
5. Scott, SK, Johnsrude, Ingrid S: The neuroanatomical and functional organization of speech perception (Trends Neurosci 26, 100-107, 2003).[5]
   Functional neuroimaging studies prove that human speech perception might be based on multiple hierarchical processing pathways. Different representations could be preferentially treated in these different streams (such as acoustic-phonetic features and articulatory gestures). The authors provide detailed descriptions in this regard. Regarding the classical neuroanatomy of speech, the authors' study expands the role of Broca's and Wernicke's areas as output and input systems and differentiating sub-systems within these.
6. Hickok G, Poeppel D: Towards a functional neuroanatomy of speech perception (Trend Cogn Sci 4, 131-138, 2000).[12]
   The authors elaborate on the neural substrate for speech perception and provide the basis for the auditory-motor interface. The authors provide a single cortical network model supporting speech perception and related language function.
7. Wise, RJ, Scott SK, Blank SC, *et al*. Separate neural subsystems within the Wernicke's area. Brain 124, 83-95, (2001).[13]
   This is a PET study. The results are compatible with a hypothesis that the posterior superior temporal cortex specialized for the process involved in mimicry of sound, including repetition, the specific role of the posterior left superior temporal sulcus being to represent phonetic sequences transiently. These processes are central to acquiring long-term lexical memories of novel words.
8. Petersen SE, Fox PT, Posner MI *et al*. Positron emission tomographic study of processing single words (Journal of Cognitive Neuroscience, 1(2) 153-170, 1988).[14]
   Language can be studied at many levels and interests philosophers, linguists, and anthropologists. Cognitive psychological and neurological investigations often focus on a subset of the diverse whole, language, and the processing of individual words. This aspect of language, lexical processing, involves a network of several levels of internal coding that can be isolated by experiment (*and studied elegantly with recent neuroimaging techniques* (Italic added by the present author).
9. Price CJ: Functional anatomy of word comprehension and production (Trends in Cogn Sci 2, 281-288, 1998).[15]
   A review article based on data from functional neuroimaging studies in normal subjects and patients with brain damage indicates that the left inferior temporal and left posterior-inferior parietal cortices are required for accessing semantic knowledge; the left posterior basal temporal lobe and the left frontal operculum are necessary for translating semantic into phonological output and the left anterior-inferior parietal cortex is required for translating orthography to phonology.
10. Fiez JA, Petersen SE: Neuroimaging studies of word reading (PNAS 95, 914-921, 1998).[16]
    Study results converge to reveal a set of areas active during word reading, including left lateralized regions in the occipital and occipitotemporal cortex, the left frontal operculum, bilateral regions within the cerebellum, primary motor cortex, the superior and middle temporal cortex, and the medial regions in the supplementary motor area and anterior cingulum. The review highlights the importance of spelling-to-sound in transforming orthographie (word form) to phonological (word sound) representation.
11. Price CJ, Wise RJS, Warburton EA, *et al*. Hearing and saying: The functional neuroanatomy of auditory word processing (Brain 119, 919-931, 1996).[17]
    A very detailed PET study, several of its findings regarding the perception of another's voice were confirmed in the paper (10) above
12. Demonet J-F, Chollet F, Ramsay S, *et al*.: The phonological and semantic processing anatomy in normal subjects (Brain 115, 1753-1768, 1992).[18]
13. Demonet J-F, Price C, Wise R, Frackowiak RST: Differential activation of the right and left posterior sylvian regions by semantic and phonological tasks: A positron-emission tomography study in normal human subjects (Neurosci Lett 182, 25-28, 1994).[19]
    Summaries of these two papers (number 12 & 13) by a very distinguished group of neuroscientists utilizing PET studies show that by comparison to a reference non-verbal tasks (tones), a phonological (phonemes) task gave rise to rCBF increases in the mid-part of the left superior temporal gyrus and the posterior part of the left inferior frontal gyrus. In contrast, a lexical-semantic (words) task is activated over and above these areas, including inferior temporal, inferior parietal, and superior prefrontal cortices.(For more detailed information, refer to these papers.)
14. Binder JR, Frost JA, Hammeke TA, *et al*.: Function of the left planum temporale in auditory and linguistic processing. (Brain 119, 1239-1247, 1996).[20]
    The author's fMRI study consisted of 12 normal right-handed subjects during passive and active hearing. The authors reported a clear differentiation between auditory and linguistic aspects of speech. The planum temporale is likely to be involved in early auditory processing, while specifically linguistic functions are distributed in the left hemisphere. As language is a function that integrates

sensory information across modalities, language zones might be expected to have multimodal or even a modal neurophysiological characteristic and to be positioned where processing streams arising from multimodalities converge (Geschwind N 1965,[21,22] Damasio 1988[23]). The present study has confirmed this.

15. Belin P, Zatorre RJ, Lafaille P *et al*. Voice-selective areas in the human auditory cortex (Nature 403, 309-313, 2000).[4]
    The human voice contains a wealth of information on the speaker's identity and emotional state in its acoustic structure, which we perceive with remarkable ease and accuracy. However, little is known about its neural basis. The authors using fMRI imaging in human volunteers show that voice-selective regions can be found bilaterally along the upper bank of the superior temporal sulcus (STS).
16. Scott SK, Blank C, Wise RJS: Identification of a pathway for intelligible speech in the left temporal lobe (Brain 123, 2400-2406, 2000).[24]
    The authors claim, using PET imaging, that the left superior temporal sulcus (STS) responds to the presence of phonetic information. Still, its anterior part only responds if the stimulus is also intelligible. Speech is an immensely complex stimulus from which acoustic-phonetic features must be processed before they become comprehensible. "Intelligibility" covers several properties of language, including word form, recognitive syntax and semantics.
17. Rumsey JM, Horwitz B, Donohue BC, *et al*. Phonological and orthographic components of word recognition: A PET-rCBF study (Rumsey JM et al Brain 120, 739-759, 1997).[25]
    Phonological processing is subserved by a left-lateralized large-scale network of regions involved in spoken language, which include the posterior superior temporal gyrus, the angular/supramarginal gyri and the inferior frontal/insular region. Aspects of phonological processing in pronunciation, namely recording for lexical access, involve the superior temporal gyri, particularly the left. Phonological decision-making, which involves subvocal rehearsal and articulatory programming (components of verbal working memory), is predominantly subserved by the left-sided region near Broca's area, including BA44 and the insula. Phonological processing is slower than orthographic processing, which some investigators hypothesize is not modular but involves visual coding of linguistic patterns or invariances. Both phonological and orthographic processing appear to rely on a common language network, consistent with connectionist models of reading. Phonological processing is not a unitary process but refers to various methods, including perception and memory processes, rather than being subserved by a single localized mechanism in the brain. These processes deferentially engage multiple regions within a large-scale language network. The present finding and others suggest that these regions include the inferior frontal cortex (BA44), the superior temporal gyri (BA 22/42), the angular/supramarginal gyri (BA39/40) and the insula, particularly on the left. Orthographic processing is a relatively poorly defined construct.
18. Zatorre RJ, Meyer E, Gjedde A, Evans AC. PET studies of phonetic processing of speech: Review, replication and reanalysis (Zatorre RJ *et al*. Cereb Cortex 6, 21-30, 1996).[26]
    "It is reasonable to assume that the new process in the superior temporal gyri is initially responsible for perceptional analysis of the complex incoming speech stream. Neurophysiological studies of the auditory cortex reveal the presence of neuronal populations sensitive to acoustic features present in speech sounds, such as frequency modulation or onset time. It is, therefore, likely that the CBF activation in the left and right anterior superior temporal area observed during passive speech (Petersen *et al*. 1988,[14] Wise *et al*. 1991,[27] Zatorre *et al*. 1992,[28] Binder *et al*. 1994,[29] Jarvis, 2004[30]) reflected the operations of such neural systems.
    The posterior region of the left superior temporal plane, roughly coextensive with classically defined Wernicke's area, likely plays a unique role in speech processing. Since this region is not activated by simple tones or noise stimuli (Zatorre *et al*. 1992[28]) or by auditory tonal discrimination tasks (Démonet *et al*. 1992,[18] 1994,[19] Zatorre *et al*. 1994[31]) but is consistently activated by speech stimuli (Petersen *et al*. 1988,[14] Wise *et al*. 1991,[27] Zatorre *et al*. 1992,[28] Binder *et al*. 1994,[29] Rumsey *et al*., 1997,[25] Scott SK *et al*., 2000[24]). Furthermore, Price *et al*. (1996[32]) found that CBF increases linearly with the word presentation rate in most right and left superior temporal regions but is constant in the left superior temporal area. This finding suggests that perceptional analysis occurs bilaterally in the superior temporal gyri since such analysis would depend on the amount of acoustic input per unit of time. In contrast, the left superior temporal region presumably performs a more abstract level of analysis. The processing carried out within the left posterior temporal area is not fully understood but probably involves the study of speech sounds leading to comprehension and may operate at syllable or whole word level.
    Cortical stimulation studies are also relevant: Ojemann and Mateer (1979)[33] noted that disruption in phonetic tasks was often observed at sites that also led to orofacial movement. This association suggests that the articulatory motor process and phonetic process share an overlapping neural representation, a conclusion that aligns well with our model.
    Khul PK (2011)[34-36]: As late as 2011, Palricia Kuhl, from the Centre for Mind, Brain and Learning, University of Washington, Seattle, remarked, "We are still just breaking ground regarding the neural mechanism that underlies language development and its critical period. In the last decade, brain and behavioural studies indicated a complex set of interacting brain systems in the initial acquisition of language early in infancy, many of which reflect adult language processing.

## Summary

PET studies were used to identify brain areas related to lexical (single-word) processing. A few discrete regions were active during several task conditions, including modal-specific (auditory or visual) areas activated by passive word input, primary motion and premotor areas during speech output. Yet, during tasks that make semantic or attentional demands brain damage, it became apparent that though often used synonymously, there is a subtle difference between speech and language. There are differences in their neural substrate. However, it was still generally felt that keeping their differences in mind, there is so much commonalty between the two that it is reasonable to discuss these together. Notwithstanding this

general practice, it prompted the present author to study the subject in some detail for the sake of scientific accuracy.

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# Birdsong: Comparisons with Human Speech and Language

## Speech and Language vis-à-vis Birdsong

A unique aspect of humans is their ability to use speech and languages to communicate with others of their own species. Whereas speech refers to the manner in which sounds and words are produced, which includes articulation, the voice and fluency or the rhythm of speech, language can be defined as the words used in speech and how they are used to share ideas and convey information. Language includes what words mean, how to make new words, how to put together words and to be able to use appropriate sets of words depending on the context (https://www.asha.org/public/speech/development/speech-and-language/). Although speech and language are unique to humans, many aspects of this form of communication are very similar to vocalizations produced by songbirds. Birdsongs are composed of patterned vocalizations consisting of individual units called notes or syllables of various types (such as frequency-modulated or harmonic stacks), separated by intersyllable intervals and sung in a specific order. A specific sequence of syllables/notes which is repeated multiple times is called a song or a motif and several such motifs form a bout (cf. Zann RA, 1996).[1] Furthermore, just as language-use is context-dependent, songbirds use their vocalizations to attract mates or to defend their territories (Catchpole CK and Slater PJ, (1957).[2] In addition, birds such as zebra finches, wherein only males sing and produce female-directed songs to attract mates, also produce 'undirected' songs which are sung in isolation, and may be for maintenance or practice (Morris, 1954;[3] Zan RA, 1996[1]).

## Vocal Learning and Sensitive Periods

The sensitive period is a restricted period during development when the brain is highly susceptible to modifications, since new synapses can be formed at this time based on changes in the sensory environment (Hensch TK, 2004[4]). However, some degree of plasticity remains until adulthood, so that experience-dependent modulations are possible in the adult brain as well. One of the most striking similarities between speech and birdsong is that both are learned or acquired from parents or tutors during a sensitive period early in development. In humans, the development of speech or more specifically, speech recognition starts in intrauterine life, since fetuses are capable of perceiving sound as early as 22-24 gestation weeks (GW). It is therefore not surprising that by birth (40GW), neonates can respond specifically to sound stimuli including their mothers' voices (DeCasper AJ and Fifer WP 1980;[5,6] Moon C *et al*., 1993;[7] Lang S *et al*., 2021[8]) and languages (Fifer and Moon, 1994), melodies (Hepper PG, 1988[9]) and sound such as that of aircrafts, if their mothers were living in the vicinity of airports (Ando Y and Hattori H, 1970[10]) was tested by using EEG, ECG, habituation tests and ultrasounds (DeCasper AJ and Spence MJ, 1986;[11] Partanen E *et al*., 2011,[12] Granier-Deferre C *et al*., 2011,[13] James *et al*., 2002[14]). Furthermore, Best CC and Roberts GW (2003[15]) have demonstrated that 6-8month-old infants are sensitive to categorical perceptions of native and non-native speech sounds (contrasts between various consonants in different languages. However, they lose the ability to discriminate between non-native speech sounds by 10-12 months, at which time they only retain this ability for their native language. Yet another interesting study (Mampe B *et al*., 2009[16]) has demonstrated that as a result of prenatal exposure to single native languages (French or German), infants from each group produce different melody contours while crying, which are similar to the prosody of spoken French or German. The human auditory cortex is known to be highly plastic until ~7 years of age, after which the ability to learn a second language decreases (Sharma *et al*., 2002; Kuhl PK, 2010[17] for review). Additionally, cochlear implants performed on congenitally hearing-impaired children during the sensitive period (18 postnatal months) provide better cortical activation, language acquisition and speech production compared to those at older ages (Dunn CC *et al*., 2014;[18] Sharma *et al*., 2002, Kral A and Sharma A, 2012;[19,20] Ponton and Eggermont, 2002[21]).

Besides humans, a small group of mammals (including bats, whales and dolphins) are capable of acquiring meaningful vocalizations by imitating their parents and/or peers and other conspecifics (Jarvis ED, 2004[22]). Besides these species, parrots, hummingbirds and songbirds have also evolved as vocal learners and learn to produce their vocalizations during an early sensitive period from their parents/tutors (Immelmann 1969;[23] Marler P 1970;[24] Thorpe WH 1958;[25] for review, Brainard MS and Doupe AJ, 2002[26]). Whereas some species of songbirds are classified as open-ended learners, since their songs change every year (for example, canaries and nightingales, Kipper S *et al*., 2004;[27] Jackel D 2022;[28] Bartsch C *et al*., 2015[29]), species such as zebra finches produce only one fairly stereotyped song at the end of the sensitive period, which is typically learned from their fathers (Immelmann, 1969;[23] Marler P and Peters S 1982;[30] Böhner J, 1990;[31] Zann R 1990;[32] Slater P *et al*., 1993[33]).

Although it was earlier thought that songbirds start learning their vocalizations at ~20 days after hatching, recent studies have demonstrated that the structure of begging calls (used to attract the parents' attention during feeding) of superb fairy wrens are similar to the incubation calls produced by their mothers (Colombelli-Negrel D *et al*. 2014, 2017[34,35]). These findings have been confirmed by the increase in heart

rate which has been observed in the embryos of these birds in response to playbacks of the mother's songs (Colombelli-Negrel D and Kleindorfer S 2017[35]). However, a large part of vocal learning in birds takes place later during development. In the commonly studied zebra finch, juvenile birds initially memorize their tutors' songs during an initial 'sensory' phase (20d – 35d post-hatch) and form a 'template' or memory of this song in their brains, they refine their songs by practice and auditory-motor integration in the 'sensorimotor' phase (35d-65d; Slater *et al*., 1993;[33] Gobes SM *et al*., 2019[36] for review). The sensory and sensorimotor phases may overlap or may be separate in different species of songbirds (Arnold AP 1975;[37] Böhner, 1990;[31] Doupe AJ and Kuhl PK, 1999;[38] Brainard MS and Doupe AJ, 2002[26] for review).

## Sensory Inputs are Important for Song Learning

### Auditory input

The acquisition and production of a normal song in birds depends on different types of sensory inputs during the sensitive period for song learning. Removing any of these important inputs within this period dramatically disrupts song in adult birds, a feature common to many other sensory systems such as the developing visual or somatosensory system in mammals. One of the most important inputs for a juvenile bird to be able to produce a normal song during adulthood is to hear its father or a tutor's song during the sensitive period. Experiments to test the importance of normal auditory inputs have been of two kinds: to remove auditory input and prevent auditory feedback by deafening birds or to remove only auditory input (by isolating juvenile birds). Deafening birds by ablating both cochleae at the onset of song learning (20d) causes a major disruption in the songs of these birds at adulthood (Konishi M, 1965[39]). Compared to the songs of normal adults, the songs of deafened birds have highly variable notes, very little structure, low repetition of individual notes and long inter-note intervals (Price PH,1979[40]). Price (1979) also demonstrated that there was no difference in the extent of disruptions in the songs of zebra finches deafened between 16-43 days or 63-84 days post-hatch during the sensitive period for song learning. Since the 16-43-day interval occurred before the template was formed while the 63-84-day interval followed the formation of the template, it was concluded that auditory feedback was as important as auditory input for song learning.

Auditory input to juvenile birds can also be disrupted by placing them in isolation so that they are not exposed to a tutor's song during the sensitive period. In this case, birds still have access to auditory feedback unlike birds which have been deafened. Individually isolated juvenile birds also showed very large disruptions in the song they produced during adulthood with fewer distinct notes, higher variability in notes as well as inter-note intervals as compared to controls (Price PH,1979[40]). Furthermore, Eales (1985) isolated 35d-old juvenile male zebra finches from their parents. These birds had still not completely formed a normal template and therefore produced an abnormal song when they reached adulthood. Eales found that the sexually mature isolates (at 180 days of age) were still able to form good copies of their tutors' songs. This finding indicates that the sensitive period for song learning is not rigid but may be lengthened if birds do not have access to a normal song during the sensitive period for song learning.

Interestingly, Deshpande M *et al*.[41] (2014) have demonstrated that only 75 seconds of exposure to the tutor song which was played to juvenile male zebra finches for two hours leads to song learning. This study has also shown that the changes that were induced by single session of exposure to the tutor's song between 35d and 45d post-hatch could be correlated with the songs that the tutored birds produced as adults.

### Auditory and visual cues: Social interactions

Earlier studies have demonstrated that songbirds are capable of vocal learning by using taped conspecific vocalizations, which allows greater control over factors such as syllable structure and timing the adult song (Soha and Marler 2000; Soha JA and Marler P 2001[42]). However, such manipulations disrupt the natural environment and socialization that are needed for juvenile birds to acquire songs. Interestingly, Baptista LF and Petrinovich L (1984; 1986)[43,44] have demonstrated that swamp sparrows which were exposed to tapes of tutor songs during their sensorimotor period learned the songs of live tutors presented to them after this period, suggesting an extension of their sensitive period for song learning (cf. Eales, 1985). These findings suggest that besides normal auditory input, a normal social milieu and social interactions with the tutor are necessary for song learning. As a caveat, recent studies (Nordby JC *et al*., 2001[45]) have demonstrated that song sparrows (*Melospiza georgiana*) which have been exposed to live tutors during their sensitive period can still learn from a different tutor as late as 150d after initial crystallization of their songs.

Besides auditory input, visual cues are also extremely important for the formation of a normal song. When birds are placed in isolation, with normal auditory but no visual cues, their songs are almost as disrupted as birds deprived of auditory cues (Price, 1979[40]). In addition to visual cues, social interactions are also very important for the acquisition and production of normal song. Different groups of investigators (Immelman, 1969,[23] Price, 1979[40]) have found that juvenile zebra finch males can be cross fostered to males of other species. When juvenile zebra finches are exposed to Bengalese or Strawberry finch males during their sensitive period, they form a good copy of their foster father's songs. Interestingly, juvenile zebra finches raised by a heterospecific foster father and later exposed to their natural fathers will still imitate their foster father's song (Immelman, 1969[23]). These findings are similar to those from studies on humans showing that babies do not learn languages from audio or audio/video recordings but can learn efficiently from active tutors (Kuhl PK *et al*. 2003[46]). Another study has also demonstrated that positive responses provided by mothers in response to babbling by infants produced more syllable-like sounds compared to infants whose mothers' responses did not synchronize with their babbling (Goldstein MH *et al*., 2003[47]). This was also observed in zebra finches, wherein there was an improvement in the quality of vocalizations produced by young zebra finches which elicited a response from their mothers (Carouso-Peck S and Goldstein MH, 2019[48]). In addition, normal auditory input is also required in adult birds for the maintenance of a normal song (Konishi M, 1965;[39] Immelman, 1969;[23] Eales, 1985,1987;[49,50] Marler P and Sherman V, 1983;[51] Nordeen and Nordeen KW, 1993,[52] Iyengar S and Bottjer SW, 2002[53]), which is similar to requirement of normal auditory input and feedback for the maintenance of normal speech in adult humans.

It has also been found that if juvenile zebra finches are given the opportunity to copy songs of different tutors, they seem to prefer the song of the tutor who behaves most aggressively towards them. This study also demonstrated that when groups of juvenile male zebra finches which had been isolated from their tutors and raised in a group of similarly aged birds, their song were very similar to each other (Volman SF and Khanna H, 1995;[54] Tchernichovski and Nottebohm, 1998[55]). Thus, it is possible that social interactions with siblings in zebra finches are also necessary for the refinement of song after the sensorimotor phase begins.

## Stages of Vocal Learning

Kuhl PK and Meltzoff AN (1996)[56] have proposed that vocal learning in humans can be divided into five stages. The first, occurring between 0 and 2 months involves reflexive phonation and includes sneezing, coughing and crying. Infants predominantly produce 'quasivocalic' sound or cooing sounds which resemble vowels between 1-4 months during the second stage of vocal learning. This is followed by a third stage, that is, 'expansion', during which 3-8month-old infants can clearly produce vowels, besides being able to yell, scream and whisper. In the fourth or 'canonical babbling' stage, 5-10month-old infants produce series of consonants and vowels, finally giving way to 'meaningful speech' (fifth stage) which includes occasional bouts of babbling at 10-18 months. In a similar manner, after an initial period of only producing begging calls, juvenile birds start to produce soft, rambling vocalizations called 'subsongs', akin to babbling in human infants during the initial part of the sensorimotor period (Doupe and Kuhl, 1999[38]). This is followed by the production of 'plastic' song, which is still malleable and involves auditory-motor integration to match self-generated syllables with those present in the template. At the end of the sensorimotor period, adult birds are able to produce a 'stereotyped' or a fixed song, which does not change significantly in closed-ended learners, such as zebra finches but continues to be changeable in open-ended learners such as canaries (Brainard MS and Doupe AJ, 2002;[26] for review).

## Neural Circuits Underlying Hearing, Vocalization and Vocal Learning

### Auditory pathways

Parrots, songbirds and hummingbirds possess dedicated neural circuits which are important for hearing, vocalization and vocal learning. There are a number of similarities between the auditory pathways of mammals, birds and reptiles (Vates GE *et al*., 1996;[57] Carr CE and Code RA, 2000;[58] Webster DB and Fay RR, (2013)[59]). In humans, hair cells in the cochlea send auditory input to sensory neurons whose axons collectively form the cochlear nerve. The cochlear nerve sends auditory inputs to brainstem auditory nuclei, including the superior olivary nucleus and the nucleus of the lateral lemniscus. Auditory information from these brainstem nuclei is further transmitted to inferior colliculus which projects to the medial geniculate nucleus (MGN) of the thalamus. The MGN in turn, gives rise to thalamocortical projections, innervating the primary and auditory association areas for further processing [for review, see Iyengar S, 2012;[60] Figure 1].

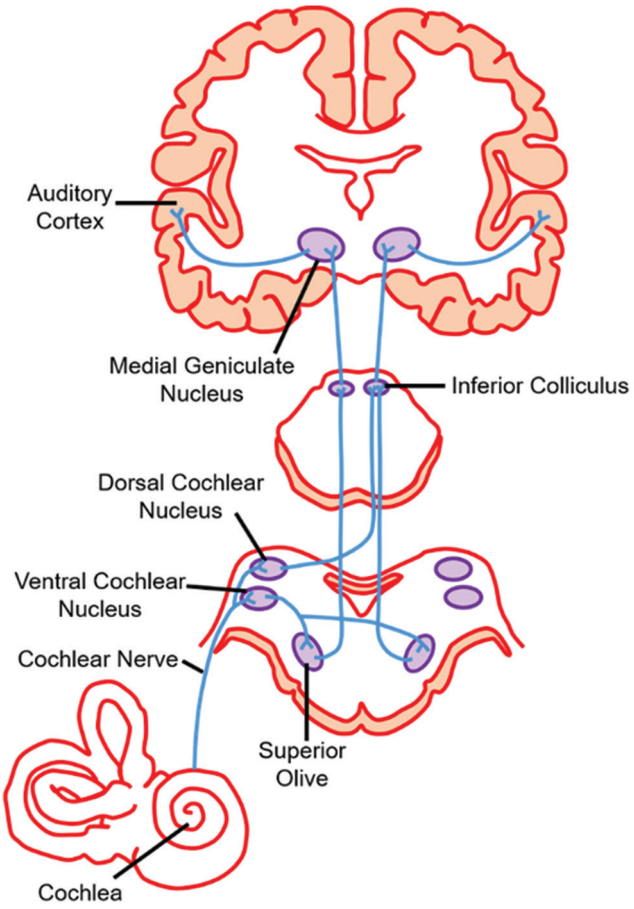


Figure 1

Parrots, songbirds and hummingbirds also possess dedicated neural circuits which are important for hearing, vocalization and vocal learning. The cochlear nerve in these avian species sends auditory information to brainstem lemniscal nuclei which then project to the nucleus mesencephalicus lateralis, pars dorsalis (MLd) which is homologous to the mammalian inferior colliculus. The MLd projects to nucleus ovoidalis, a homologue of MGN, which further projects to Field L2 or the avian auditory cortex. Interestingly, Wild JM *et al*. (1993)[61] and Karten HJ (1991)[62] have demonstrated that avian pallial regions (L1 and L3) are similar to the supragranular layers of the mammalian primary auditory cortex. Furthermore, Jarvis ED (2004)[22] has proposed that the caudal part of the medial nidopallium (NCM) and caudal part of the medial mesopallium (CMM) in birds are also similar to supragranular layers of the primary auditory cortex in mammals, based on reciprocal connections between these regions and their inputs from Field L2 [see Figure 2 for a comparison of avian and mammalian neural circuits important for vocal learning and hearing adapted from Jarvis ED, 2004[22]].

### Neural circuits for vocalization and vocal learning

In humans, the circuitry for speech perception and comprehension is explained with the help of the older Wernicke-Lichtheim-Geschwind model and a recent dual-stream model. The Wernicke-Lichtheim-Geschwind model proposed two language centres, a motor word centre and an auditory word centre. The motor word centre is located in the

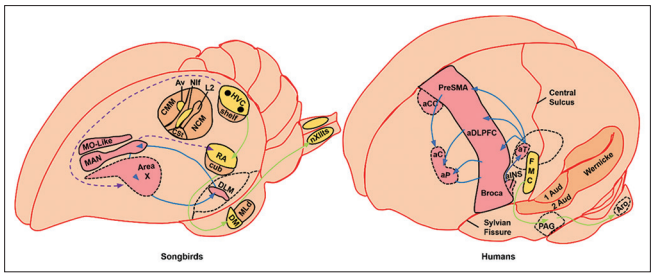


Figure 2

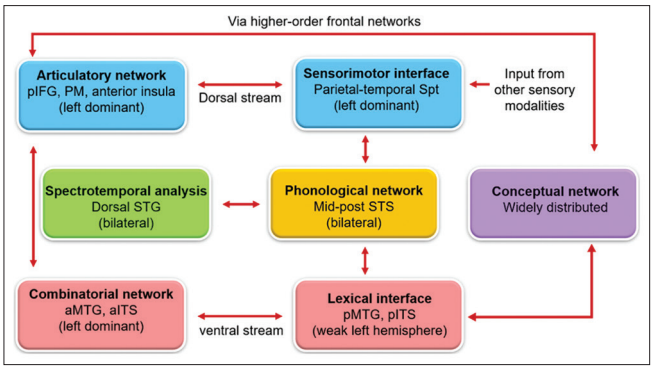


Figure 3

left posterior inferior frontal gyrus and the auditory word centre is located in the left posterior superior temporal gyrus (STG) and they communicate with each other via cortico-cortical connections. This model also proposes a conceptual knowledge network that connects with these word centres. According to this model, the auditory word centre is activated during speech comprehension, which further activates conceptual networks to enable comprehension. Similarly, for expressing thoughts in the form of speech, the motor centre is activated by conceptual networks and the auditory word centre. Thus, the auditory centre is required for both comprehension and speech. According to this model, damage to the motor centre leads to non-fluent speech (Broca's aphasia), damage to the auditory word centre leads to impaired comprehension (Wernicke's aphasia), damage to connections between these two areas results in fluent paraphasic speech with spared comprehension (conduction aphasia) and damage to neural circuits linking the language system to conceptual networks leads to transcortical aphasias. Recent research, however, has demonstrated that the language network stretches beyond the classical Broca's and Wernicke's areas and involves multiple white matter tracts and subcortical circuits (Hickok G and Poeppel D, 2007[63]).

More recently, the 'Hickok-Poeppel dual-stream model' of speech processing has modified the classical Wernicke-Lichtheim-Geschwind model for speech and comprehension to include other parts of the brain. This model has proposed that as in the case of vision (Hickok G and Poeppel D, 2007[63]), neural circuits important for speech are organized in the form of 'dorsal' and 'ventral' streams. The dorsal stream is associated with speech production and computes a mapping between the phonological representations of speech in the superior temporal gyrus (STG) and/the superior temporal sulcus (STS) and motor-based speech codes in the frontal lobe. This mapping is mediated by a cortical auditory-motor transformation zone in the posterior Sylvian region at the temporal-parietal boundary. In contrast, the ventral stream is important for the spectro-temporal analysis of speech sounds bilaterally in the primary auditory cortex. The ventral stream is also important for phonological analysis bilaterally in the STG and for computing the mapping between phonological and distributed conceptual representations in the left (dominant) middle temporal gyrus. This model therefore includes two broad streams, a dorsal stream which maps sensory or phonological representations onto articulatory motor representations, and a ventral pathway which maps sensory or phonological representations onto lexical conceptual representations. The flowchart below represents the Hickok-Poeppel dual-stream model [Figure 3; adapted from Hickok G and Poeppel D, 2007;[63] see Chapters on "From Cortex to Connectome" and "Aphasias"].

Besides the auditory pathways, songbirds possess specialized, interconnected neural circuits for vocalization, vocal learning and context-dependent singing. The pathway important for vocalization is also termed the Vocal Motor Pathway (VMP) and consists of projections from the nucleus interfacialis (NIf) to HVC (abbreviation used as a proper name; Reiner A *et al*., 2004[64]), a pallial nucleus (McCasland JS, 1987[65]). A subset of neurons in HVC (also pallial) project to a motor cortical nucleus, the robust nucleus of the arcopallium (RA; Nottebohm F *et al*.,[52,66] 1993, 1982), which in turn, projects to motor neurons in the tracheosyringeal part of the hypoglossal nerve nucleus (nXIIts). This pool of motor neurons projects to and activates muscles of the vocal organ, which is called the syrinx in songbird [Vicario DS, 1994;[67] Figure 4]. Jarvis ED (2004[22]) has suggested that RA is analogous to Layer 5 of the mammalian motor cortex since it projects to nXIIts, which is similar to projections of the pyramidal tract. Furthermore, this study has also proposed that HVC→RA projecting neurons are similar to those in Layers 2 and 3 of the mammalian motor cortex.

In addition to the VMP, an interconnected pathway in the anterior forebrain (called the anterior forebrain pathway or AFP) is specialized for song learning and contextual singing in adulthood in songbirds such as zebra finches. Interestingly, the organization of this pathway is similar to corticothalamic-basal ganglia loops which are present in mammals and are important for motor learning, cognition, executive functions and emotions [Johnson F *et al*. 1995;[68] Vates GE and Nottebohm F, 1995;[69] Iyengar S *et al*., 1999;[70] Luo M *et al*., 2001;[71] Figure 4]. A subset of neurons in HVC (distinct from that which project to RA) send projections to Area X, a prominent nucleus in the avian basal ganglia which is specialized for vocal learning (Nottebohm F *et al*., 1976, 1982[66,72]). Unlike the mammalian basal ganglia which is composed of distinct striatal and pallidal components, Area X consists of a mixture of medium spiny neurons which are similar to those present in the mammalian striatum and larger neurons which are pallidal (Reiner *et al*., 2004a, b;[64] Carrillo GD and Doupe AJ, 2004;[73] Farries MA and Perkel DJ, 2002;[74] Farries MD *et al*., 2005[75]). However, overall connectivity is similar in mammals and birds, in that striatal neurons project to pallidal neurons and both sets are inhibitory or GABAergic. The pallidal neurons project to the nucleus dorsolateralis anterior, pars medialis (DLM) of the thalamus, which projects to the lateral magnocellular nucleus

**Table 1: Comparisons between avian and mammalian brain regions**

| Avian brain regions | Mammalian brain equivalent |
|---|---|
| Pallium | Cortex |
| Nucleus MLd | Inferior colliculus |
| Nucleus ovoidalis, thalamus | Medial geniculate nucleus of the thalamus |
| Field L2 | Primary auditory cortex |
| Caudal part of the NCM | Supragranular layers of the primary auditory cortex |
| CMM | Supragranular layers of the primary auditory cortex |
| HVC (a pallial nucleus) | Motor cortex |
| Nif | Premotor cortex |
| RA | Motor cortex |
| Area X | Basal ganglia |
| DLM | VA and VL nuclei of the thalamus |
| LMAN | Premotor cortex |

MLd: Mesencephalicus lateralis, pars dorsalis, NCM: Medial nidopallium, Nif: Nucleus interfacialis, RA: Robust nucleus of the arcopallium, DLM: Dorsolateral anterior nucleus, pars medialis, LMAN: Lateral magnocellular nucleus of the anterior nidopallium, VA: Ventral anterior, VL: Ventral lateral, CMM: Caudal part of the medial mesopallium

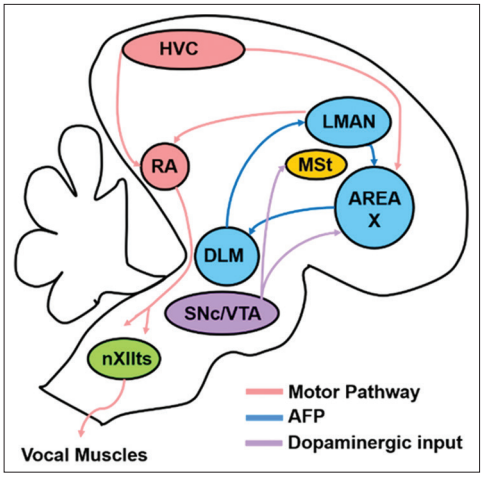


**Figure 4**

of the anterior nidopallium (LMAN), a cortical nucleus in the anterior forebrain (Carrillo GD and Doupe AJ, 2004;[73] Reiner A *et al*., 2004b[76]) which is analogous to components of the premotor cortex in humans. Furthermore, LMAN projects to both Area X and RA, forming loops within the song control system (Nixdorf-Bergweiler BE *et al*., 1995;[77] Vates GE and Nottebohm F, 1995[69]). The LMAN projection neurons may be homologous to Layers 3 and the upper part of Layer 5 of the premotor cortex in mammals, since in both cases, these neurons project to medium spiny neurons in the basal ganglia (Area X in songbirds and the striatum in mammals) as well as to RA, which is homologous to the motor cortex (Jarvis, 2004[22]). Pallidal neurons in Area X project to DLM, which is similar to projections from the mammalian globus pallidus to the VA (ventral anterior) and ventral lateral (VL) thalamic nuclei. Furthermore, both of these thalamic nuclei project to Layer 3 of the premotor cortex which has been considered homologous to LMAN (Jacobson S and Trojanowski JQ, 1975;[78] Alexander GE *et al*., 1986;[79] Alexander GE and Crutcher MD, 1990;[80] see Table 1 for comparisons between avian and mammalian brains; Jarvis, 2004[22]).

## Conclusions

Comparisons between neural circuits underlying speech and language in humans and those important for song learning and vocalization in songbirds are remarkably similar. Furthermore, the auditory system in both species is also similarly organized. As a result, a vast body of literature has used songbirds as a model system to study the effects of neuromodulators, neurotransmitters and hormones on vocalization and vocal learning, which cannot be tested in humans (cf. Doupe AJ and Kuhl PK, 1999;[38] Brainard MS and Doupe AJ, 2002[26]). Recently, songbirds have also been used to study the genetic underpinnings of speech and language since genes important for speech in humans (such as FOXP2; Vargha-Khadem F *et al*., 2005[81]) are also present in songbirds and knockdowns in these genes lead to the same kinds of deficits that are present in humans with FOXP2 mutations (Haesler S *et al*., 2007[82]). Taken together, these results demonstrate that besides neural circuits, the molecular underpinnings of speech and language can now be studied in zebra finches and extrapolated to humans.

### Abbreviations

AFP – Anterior Forebrain Pathway
ATP2C2 – ATPase Secretory Pathway Ca2+ Transporting 2
CASPR2 – Contactin-Associated Protein-like 2
chABC – Chondroitinase ABC
CMIP – c-MAF Inducing Protein
CMM – Medial Mesopallium
d – Days
DLM – Dorsolateralis Anterior Pars Medialis
DVD – Developmental Verbal Dyspraxia
ECG – Electrocardiography
ECM – Extracellular Matrix
EEG – Electroencephalography
FD – Female-Directed songs
*FOXP2* – Forkhead Box P2
GABA – Gamma-AminoButyric Acid
GAD67 – Glutamic Acid Decarboxylase 67
GW – Gestation Weeks
HVC (abbreviation used as a common name)
L1, L2, L3, L4, L5, L6 – Cortical Layers 1, 2, 3, 4, 5, and 6
LMAN – Lateral Magnocellular Nucleus of the Anterior Nidopallium
LoC – Locus Coeruleus
MGN – Medial Geniculate Nucleus
MLd – Mesencephalicus Lateralis Pars Dorsalis
MSt – Medial Striatum
NCM – Medial Nidopallium
NE – Norepinephrine
NIf – Nucleus Interfacialis
NMDA – N-methyl-D-aspartate
nXIIts – Tracheosyringeal part of the Hypoglossal Nucleus
PNNs – Perineuronal Nets
RA – Robust nucleus of the Arcopallium
RNAi – RNA interference
STG – Superior Temporal Gyrus
STS – Superior Temporal Sulcus

UD – Undirected songs
VA – Ventral Anterior Thalamic Nuclei
VL – Ventral Lateral Thalamic Nuclei
VMP – Vocal Motor Pathway
VTA-SNc – Ventral Tegmental Area – Substantia Nigra Complex

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# Neuroanatomical Correlates of Speech and Language Disorders

## A: Speech and Language Disorders: Neuroanatomical Correlates

Since antiquity, philosophers, scientists and medical men have been searching for the functions of the human brain and their location. Most of them in earlier years were searching for a seat for soul/spirit/mind/consciousness. Alemaeon (around 5th century BC) from Crotona in Southern Italy is credited for introducing human anatomy as a science. His most notable contribution was connecting the sense organs and the brain. He concluded that the brain was the organ of the mind. It was around this time that Greek philosophers, scientists, and physicians like Hippocrates (460-370 BC), Plato (428-348 BC), Aristotle (384-322BC), and Galen (129-210 AD) showed a gradual evolution from belief in super-natural and demoniac causes of mental illness to a more rational explanation attributing these to the brain. However, even as late as the 16th and early 17th centuries, Rene Descartes (1596-1650) localised the soul in the pineal gland. Interestingly, Franz Joseph Gall (1758-1828) taught that the shape and irregularities of the skull were projections of the underlying brain and, consequently, of a person's mental characteristics but without any scientific basis of the same. However, this "promoted" the concept that the brain is a composite of discrete but interrelated functions anatomically confined to specific areas (Please refer for above to Tandon P and Chandra PS, 2022).[1]

As far as recorded knowledge goes, the earliest instances of rational medical expertise in India are found in the *Rig Veda* and *Atharvaveda,* both from the 2nd millennium BC. However, even the most famous. Medical luminaries of ancient India Susruta (800-700 BCE)* and Charaka (400-200 BCE)* were primarily concerned about the location and function of the soul, mind and Self *(Atman)*.

(*these figures are approximations, rather than verified. For A brief note on "Ancient-Neuroscience see Evolution of Neurosciences by PN Tandon and P. Sarat Chandra, Neurol India Special Issue: Vol 70. Suppl 1, 2022 [available on www.neurologyindia.com[1]]).

However, credit goes to Pierre Paul Broca (1824-1880)[2,3] of Paris, who, based on a single case he had carefully followed clinically, developed the concept of the relationship between specific symptoms and circumscribed lesions in the brain. It was in 1861 that he demonstrated this case before the Society Anthropologie (which he had founded in 1859). This lesion in the left frontal lobe of his patient who suffered from *aphemie* (renamed aphasia by Trousseau in the same year). Ferrier D, 1878[4] later called this area Broca's convolution.

From his and Broca's subsequent observations, he concluded that the integrity of the posterior part of the third frontal convolution was indispensable to articulate speech and, therefore, termed the region the *circumvolution du language* (Later Ferrier referred to as Broca's Convolution). Thus, Broca brought to a head the smouldering idea promulgated some decades before in Paris by Franz Joseph Gall (1757-1828, cited by Eling P[5]) that frontal lobe lesions may cause speech disorders.

Broca's concept[6-9] was not accepted generally and was opposed by Trousseau and Huglings Jackson. However, his idea typically got approbation in 1868 at the British Association for Advancement of Science. Following this epic event held in Norwich, it got further support from Wernicke C in 1874.[10,11] A host of contemporary neurologists in Paris and London contributed to this field: Charcot (1825-1893, cited by Jaccard C, 2024[12]), Pierre Marie (1853-1940, Pearce JM, 2004[13]), Jackson JH (1835-1911),[14,15] Wernicke C (1848-1904),[10,11] Franz Joseph Gall (1757-1828, cited by Polgár L 1976),[16] Sir David Ferrier (1843-1928, cited by Sandrone S and Zanin E, 2014)[17] (For a brief review of early developments in the field, see Chapter 1).

A detailed clinical study carried out by Mazzocchi F and Vignolo LA (1979)[18] on 90 right-handed patients with present or past evidence of dysphasia following a stroke, given a standard language battery and submitted to a CT scan examination for whom a correlation was carried out on the location and extent of lesion seen on the scan. It revealed most of the findings to be compatible with the traditional views, e.g., anterior lesions resulted in non-fluent aphasias with good comprehension (Broca's type aphasia), and a posterior lesion resulted in fluent aphasia (e.g. Wernicke's type aphasia). However, they observed several unexpected findings, e.g. pure anterior lesions resulting in global aphasia or purely deep lesions not involving the cortex in Broca's aphasia.

Recent advances in neurolinguistics, experimental neuropsychology, cognitive neuroscience, and non-invasive neuroimaging have contributed significantly to elaborating on the cerebral localization of speech and language. Detailed clinical studies of speech defects revealed that the original concept of motor (Broca's) and sensory (Wernicke's)[19] dysphasia consisted of a number of subgroups, and their studies using recently developed neuroimaging studies revealed a much better neuroanatomical localization than the earlier clinicopathological correlation based on autopsy studies. In this chapter, an attempt is made to summarise the recent advances in the field. This would include not only the classical speech disorders but also disorders of speech, writing, reading and calculation [Table 1].

Over the years, multiple extensions and subdivisions of speech and language clinical syndromes have existed. Thus, Bogen JE and Bogen GM (1976)[20] commented, "There are almost as many classifications of aphasia as there are aphasiologists".

Wernicke himself questioned, "Is there more than one kind of aphasia?". However, for most clinicians, there were two classical aphasias, the Broca's and Wernicke, no doubt this was far from the truth. Interestingly, Lichtheim L, as early as 1885,[21] pointed out at least seven different dysphasic syndromes. According to Alexander MP (2003),[22] at least eight different aphasic syndromes exist. Based on different studies, the cortical localization of these syndromes is summarized in Table 2.

**Table 1: Summarises the various types of aphasic syndromes. Sources from 1 to 7 have been also referenced from Kahn and Whitaker, 1991[23]**

| Authors | Source | Site of lesions |
|---|---|---|
| 1. Lewandowski[24] | They reported one of the first cases of an acquired calculation disorder before Henschen (1920) | Left occipital hematoma: Difficulty with addition and subtraction as well as regrouping two and three-place numbers |
| 2. Henschen[25,26] | Based on approximately 110 abstracts and sources | Pointed out that exclusive lesions of the left hemisphere, which resulted in aphasia, figure blindness, figure agraphia and acalculia, only infrequently produced a number of deafness. He also thought that the right hemisphere participated in calculation in a compensatory manner |
| 3. Berger[27] | 18 cases, only 3 cases studied in detail | Case 1: Lesion of a left hemispheric glioma reaching into posterior temporal and occipital lobes with both angular gyrus and supramarginal gyrus intact<br>Case 2: Glioma left paracentral lobule invading funicular gyrus, corpus callosum and left occipital lobe<br>Case 3: With total loss of division and multiplication, had a left thalamic glial sarcoma |
| 4. Head[28] | In his book Aphasia Kindred Disorders | Made only a brief reference to calculation. He considered each variety of aphasia associated with a distinct form of arithmetic disorder |
| 5. Cohen (1926, cited by Eling and Whitaker[29]) and Head (1961, cited by Jacyna[30], see also[28,31-33]) | | According to Cohen (1961), Head (1926) indicated that in severe "nominal aphasia" which resulted from a lesion primarily in the angular gyrus of the dominant hemisphere, profound confusion about the meaning of number occurred. Acalculia typically followed. Lesions in the parieto-occipital region and occasionally the frontal lobe. He stated that most published cases had lesions in the occipital lobe |
| 6. Hecaen and Houillier[34] | Two papers | Both papers noted that most acalculia cases exhibited bilateral lesions primarily in the parieto-occipital region and argued that in all instances, parietal lobes constituted the central zone for calculation processes. They also proposed that arithmetic would only be found in case of diffuse cortical damage |
| 7. Luria[35] | Book on "Higher Cortical Function in Man" | Proposed that the parieto-occipital region and its role in linguistic operations are equally critical for arithmetic |

**Table 2: Cortical localization of various aphasia syndromes**

| Authors | Source | Site of lesion |
|---|---|---|
| 1. Whitaker and Ivers[36] | A single case of a deep left hemisphere infarct | Anterior and posterior lentiform nucleus, superior caudate nucleus and adjacent white matter |
| 2. Corbett *et al.*[37] | Single case study of subcortical infarct of the left hemisphere | The head of the caudate, the nucleus anterior superior putamen, and the anterior limb of the internal capsule extending superiorly into the periventricular white matter |
| 3. Kahn and Whitaker[23] | An extensive review of cases of acalculia since Henschen | Neither a localized region nor a specific hemisphere uniquely underlies the disorder |
| 4. Lucchelli and De Renzi (1993[38] | Case report | Primary dyscalculia after a medial frontal lesion of the left hemisphere |
| 5. Fosotti[39] | Clinical study | Patients with frontal lesions have difficulty translating arithmetical word problems into an internal representation. They did not find significant differences in performance with left, right or bilateral frontal lesions |
| 6. Allison *et al.*[40] | Intracranial ERP recording | Area in the fusiform and inferior temporal gyri were responsive to numerals |
| 7. Dehaene and Cohen (2007)[41] | fMRI studies | The left inferior parietal cortex is the most frequent cortical site of damage causing anarithmetia |
| 8. Polk and Farah[42] | Using FMRI in six subjects | Left side occipitotemporal area that responded more to letters than numerical |
| 9. Dehaene *et al.*[43] | fMRI studies | Multiple mental representations are used for different tasks, even within the small arithmetic domain. Exact arithmetic emphasizes language-specific representations and relies on a left inferior frontal circuit for generating associations between words |
| 10 Gitelman[44] | Chapter in a book: A very detailed review | Several figures depict brain areas involved in various numerical functions over virtually every brain region. Table 7.7 [Figures 7.6, 7.7 and 7.8] are illustrative of lesions |

FMRI: Functional magnetic resonance imaging

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## B: Cerebellum: Speech and Language

> "To regard the cerebellum as only serving motor function is too narrow a concept".
>
> Gordon 1996.[1]

During the past 2 decades, the collaboration across disciplines and the methodologies and conceptual advances of contemporary neuroscience have substantially modified the traditional view of the cerebellum as a mere coordinator of autonomic and somatic motor functions.

Growing insights into the neuroanatomy of the cerebellum and its interconnections, evidence from functional neuroimaging and neurophysiological research, and advancements in clinical and experimental neuropsychology have established the view that the cerebellum participates in a much more comprehensive range of functions than conventionally accepted. This increase in insight has brought to the fore that the cerebellum modulates the cognitive functioning of at least those parts of the brain to which it is reciprocally connected (Marien *et al*. 2001).[2] The authors discuss the modulatory role of the cerebellum in various non-motor language processes such as lexical retrieval, syntax, and language dynamics (For a detailed historical evolution of this concept, see the original paper).

Gordon N (1996)[1] elaborated on how the cerebellum can affect speech and language in several ways. The most commonly recognized is dysarthria, when motor movements are deprived of regulatory control, which is one of the main functions of the cerebellum. Less well-known is cerebellar mutism (Bolk L 1906,[3] Snider RS 1950,[4] Moore MT 1969,[5] D'Avanzo R *et al*. 1993,[6] Courchesne E *et al*. 1995,[7] van Dongen HR *et al*. 1994[8]), which most often occurs after the removal of a cerebellar tumor. The most controversial aspect of cerebellar function is the contribution it may make to language production. Several studies have revealed that the cerebellum can be involved in cognition and language (Snider RS 1950,[4] Fabbro F *et al*. 2000,[9] Mathiak K *et al*. 2002,[10] Ackermann H and Daum I 1995,[11] Leggio MG *et al*. 2000,[12] Ackermann H *et al*. 1999,[13] Stoodley CJ and Schmahmann JD 2009a,[14] Leiner HC and Leniner AL 1993,[15] Ito 1993[16]).

Further studies attributed this to the crossed cerebro-cerebellar fiber pathways (Fiez JA *et al*. 1996)[17] or the Cortico-ponto-cerebellar pathway (Brodal and Bjaalie 1997).[18] Thus, linguistic impairment is known to arise following right cerebellar hemisphere damage (Fabbro F *et al*. 2000,[19] Marien P *et al*. 2001[2]).

Language-related activation during phonological, semantic, and word generation paradigms has generally been observed in the right posterolateral cerebellum, with more involvement of the anterior lobe when articulation is a factor (Stoodley CJ and Schmahmann JD 2009a, b).[14,20]

Marien P *et al*. 2001[2] in their review of experimental and clinical data, highlighted the modulatory role of the cerebellum in various non-motor language processes such as lexical retrieval, syntax, and language dynamics. In agreement with the findings indicating topographical organization of the cerebellar structures involved in language pathology, they advanced the concept of a "lateralized linguistic cerebellum."

(Table 2 summarizes cerebellar involvement in speech and language, quoting a series of their papers highlighting the role of the right cerebellar hemisphere.)

Ackermann H and Daum I (1995),[11] while admitting that the neocerebellum has a significant reciprocal fiber connection with the associate areas of the cerebral cortex and that functional imaging studies have revealed activation of cerebellar regions during cognitive tasks, questioned the role of the cerebellum in language function.

However, Ackermann H (2008),[21] based on psycholinguistic and neuro-biological perspectives, recognized that. "Besides other structures, the cerebellum pertains to the brain network engaged in spoken language production. Data from different sources point to a dual role of this organ within the verbal domain: (i) the cerebellum appears to subserve the online sequencing of syllables into fast, smooth, and rhythmically organized larger utterances of overt speech, and (ii) furthermore the cerebellum seems to participate in the temporal organization of internal speech, that is a prearticular verbal code (Internal Speech)."

Several fMRI studies found focal cerebellar activation during the production of internal (Covert/Silent) speech (Callan DE *et al*. 2006[22]). There is enough evidence for the superior cerebellum's contribution to pre-articulatory speech motor control processes.

A meta-analysis of neuroimaging studies of the functional topography of the human cerebellum (including 11 studies of language) supported the hypothesis of Bolk L (1906)[3] that there is topography of motor function within the cerebellum. Contemporary clinical studies suggest that whereas the cerebellar anterior lobe is principally engaged in motor control, the cerebellar vermis is involved in affective processing, and the posterior cerebellum contributes to complex cognitive operations (Leiner HC *et al*. 1993,[15] Grafman J *et al*. 1992[23]). Furthermore, consistent with the crossed cerebro-cerebellar pathways, linguistic impairment can arise following right cerebellar hemisphere damage (Fiez JA *et al*. 1996,[17] Brodal p and Bjaalie JG 1997[18]).

### Language Processing

Language-related activation during phonological, semantic, and word generation paradigms has generally been observed in the right posterolateral cerebellum with more involvement of the anterior lobe when articulation is a factor (Stoodley CJ and Schmahmann JD, 2009a, b[14,20]).

Cerebellar patients showed significantly slower response times when naming common nouns, which correlated with their degree of motor impairment. Patients were considerably impaired on phonemic and semantic fluency measures compared to the controls.

The authors suggest that the integrity of cerebellar prefrontal loops might underlie poorer performance on measures of executive function in cerebellar patients.

The findings of (Stoodley CJ and Schmahmann JD, 2009a)[14] are confirmed by clinical and imagery studies of (Stoodley CJ and Schmahmann JD, 2009b).[20]

Notwithstanding some early contrary views, it is now well established, based on clinical, neuropsychological, and the latest non-invasive neuroimaging studies on healthy human volunteers and patients affected with cerebellar lesions, that the cerebellum plays a vital role in both overt and covert aspects of speech. The cerebellum's well-defined functional topographic regions are responsible for various aspects of speech and language functions. The cerebellum performs these functions through its fiber connections with the cortical areas responsible for the perception and production of speech and language (detailed in earlier chapters).

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## C: From Cortex to Connectome: The Latest Advances in the Neuroanatomy of Speech and Language

The neurobiological model for speech and language established by Broca-Wernicke and Lichtheim in the 19th century (as mentioned in prior sections) has long been the accepted idea regarding the brain's basis for speech and language. Geschwind N expanded on this concept in 1965[1,2] and 1970.[3] New insights from modern non-invasive neuroimaging methods have changed how we view the neuroanatomy of speech and language. Earlier ideas focused on a few specific cortical regions responsible for speech and language. Wernicke also added that there is a fiber connection between Broca's and Wernicke's areas.

Not long after, the subcallosal fasciculus was described by Muratoff W in 1895[4] in dogs and by Dejerine in 1895 in humans (as cited by Duffau H *et al.* 2002).[5] Yet, this information did not generate much interest. It is odd that it was not included in standard Anatomy or Neurology textbooks during the academic tenure of the current authors. In contrast, it is now recognized that speech and language rely on several anatomical connections that enable functional interactions among cortical areas. The pathways identified through connectomic studies form the connectomes that support the distributed cortical and subcortical systems responsible for speech and language in the human brain.

Beginning with gross anatomical dissection in the late 1800s, research on monkeys, as discussed by Seltzer B and Pandya DN (1984)[6] and Makris N and Pandya DN (2009),[7] has been significantly advanced by contemporary neuroimaging and neurophysiological methods.

Starting with CT scans of patients who have brain damage (Alexander MP *et al.* 1987)[8] and fMRI studies (Binder JR *et al.* 1996),[9] there have also been studies involving electrical

stimulation during surgery on awake, cooperative humans (Berger MS and Ojemann GA 1992),[10] as well as works by Dufau H *et al.* (2002,[5] 2008,[11] 2014[12]). Techniques such as Diffusion Tensor Imaging (Alexander *et al.* 2007;[13] Glasser MF and Rilling JK 2008;[14] Makris N and Pandya DN 2009,[7] Wassermann D *et al.* 2013,[15] Bernal B and Altman N 2010[16]) have continued to expand our understanding of the fiber tracts involved in speech and language neural substrates in humans.

Various papers from the early 2000s underscored the importance of connectomics related to speech and language (Witwer BP *et al.* 2002;[17] Clark EV 2004;[18] Jellison BJ *et al.* 2004[19]). Dick AS *et al.* (2014)[20] offered a thorough discussion of these pathways in their article, "The Language Connectome: New Pathways, New Concepts." They propose that the most effective model for language processing involves two interactive "streams"—a dorsal and a ventral stream—supported by long association fiber pathways like the superior longitudinal fasciculus, arcuate fasciculus, uncinate fasciculus, inferior longitudinal fasciculus, and inferior fronto-occipital fasciculus, along with two lesser-known pathways: the middle longitudinal fasciculus and the extreme capsule. Recently, the frontal aslant tract has been added to this framework. This two-stream model is further supported by Brauer J *et al.* (2013),[21] Griffiths JD *et al.* (2013),[22] Cloutman LL(2013),[23] and Wilson SM *et al.* (2011).[24]

The dorsal stream comprises of the superior longitudinal fasciculus (SLF) and the arcuate fasciculus (AF). Even though critical for understanding the neurobiology of language, the exact path, origin, and end of the superior longitudinal fasciculus remain unclear (Dick AS *et al.* 2014).[20] The SLF AF fiber bundles extend longitudinally within each cerebral hemisphere, linking the frontal cortex with posterior regions such as the temporal and inferior parietal cortex (Ivanova MV *et al.* 2016).[25] The AF connects frontal gyri to the medial and lateral parts of the temporal lobe through the extreme capsule adjacent to the insula (Duffau H *et al.* 2002).[5] Histological studies of the macaque by Schmahmann JD and Pandya D (2009) [26] found that SLF/AF can be divided into four subcomponents, a point also elaborated by Makris N and Pandya DN (2009).[7]

In humans, diffusion tensor imaging (DTI) and blunt fiber dissection methods by Glasser MF and Rilling JK (2008), [14]Makris N and Pandya DN (2009), [7]Catani M *et al.* (2007), [27]Brauer J *et al.* (2013), [21]and Dick AS *et al.* (2014) [20]have proposed several alternative models for dorsal stream connectivity (see Figure 1 of this well- illustrated paper). **(Figure 1 & 2)**

Earlier research proposed that the SLF/AF transmits information between Wernicke's area and Broca's area (Geschwind 1970),[3] though this has since been contested. New evidence points to the significance of this pathway in processing phonology and syntax during speech production and understanding. Damage to the AF in the dominant hemisphere can lead to various aphasia symptoms. The SLF is also linked to repetition capabilities (Brier S 2008[28]). Stimulation of the AF resulted in phonemic paraphasia and anomia similar to symptoms seen in conduction aphasia syndrome (Duffau H *et al.* 2008).[11] A nearby bundle that runs lateral to the classic AF, connecting Broca's area with the inferior parietal lobule (referred to as the Fronto-parietal phonology loop), has been shown to induce speech apraxia when stimulated (Duffau H *et al.* 2008).[11]

Earlier, the arcuate fasciculus (AF) was seen as a key component of the ventral stream. However, Ivanova MV *et al.* (2016)[25] stated, "Only within.. In the past 10 years, studies using neuroimaging and electrical stimulation have shown the importance of ventral white matter tracts in language processing. Notable contributions include work by Catani M *et al.* (2005),[29] Dronkers NF *et al.* (2017,[30] and Dick AS *et al.* (2014).[20] The French team at Salpetriere hospital, led by Duffau and colleagues, published various papers starting around 2000, focusing on electrical stimulation of these fiber tracts during surgery for low-grade gliomas. These studies have led to the understanding that, in addition to the arcuate fasciculus (AF), the ventral stream includes fibers such as the inferior longitudinal fasciculus (ILF), inferior frontal-occipital fasciculus (IFOF), uncinate fasciculus (UF), middle longitudinal fasciculus (MLF), and extreme capsule (EmC).

The ILF and IFOF are long intrahemisphere association fibers extending through the temporal lobe. The ILF connects the occipital lobe to the anterior areas of the middle and inferior temporal gyri, the temporal pole, and limbic structures. The IFOF runs medial to the ILF and connects the inferior medial occipital lobe to the inferior orbitofrontal cortex (Catani M and Thiebaut de Schotten M 2008).[31]

Semantic paraphasic errors were observed when the IFOF was electrically stimulated during surgery, as discussed by Bello L *et al.* (2008),[32] whereas similar effects were not noted with the ILF. Additional research indicates a stronger role for the IFOF in language and semantic processing, while the ILF may relate more to grammatical accuracy, indicating that longer utterances connect to the IFOF (Grossman M *et al.*, 2013).[33] In humans, the IFOF intersects with the extreme capsule, supporting its role in language comprehension.

The functional significance of the uncinate fasciculus (UF) in language processing is still uncertain. It is thought to be involved in lexical retrieval, semantic association, and aspects of naming.

The middle longitudinal fasciculus (MdLF) links the inferior parietal lobe with the anterior superior temporal gyrus and potentially the temporal pole (Makris N *et al.*, 2014, 2013),[34,35] but its specific role in language function remains undocumented.

Furthermore, the periventricular white matter near the lateral ventricle, located beneath the lower motor/sensory cortex area for the mouth, was thoroughly described by Naeser MA *et al.* (1989),[36] noting that lesions here could disrupt pathways essential for motor execution and sensory feedback. Duffau H *et al.* (2008)[11] found that stimulating this area led to anarthria or dysarthria.

The subcallosal fasciculus is the white matter surrounding the lateral angle of the frontal horn, containing pathways that connect fibers from the cingulate gyrus and supplementary motor area to the caudate nucleus.

It is clear from the above that much valuable information has been gathered in recent decades regarding the role of white matter fiber tracts in speech and language functions. This knowledge is not merely of academic interest; it has practical applications during surgeries on the dominant hemisphere. Preoperative identification of fiber tracts through diffusion tensor imaging (DTI) (Alexander

AL *et al*. 2007,[13] Glasser MF and Rilling JK 2008,[14] Makris N *et al*. 2009,[7] Bernal B and Altman N 2010,[16] Makris N *et al*. 2013[35]) facilitates surgical planning. Additionally, intraoperative electrical stimulation for conscious patients has made the safe removal of tumors or excision of epileptic foci possible, as demonstrated by Berger MS and Ojemann GA (1992),[10] Duffau H *et al*. (2002, 2008),[5,11] and Dick AS *et al*. (2014).[20]

This account also underscores the current gaps in our understanding of the neuroanatomy and specific functions of these tracts, highlighting ongoing debates in this field. The text has pointed out important information. Many new insights are anticipated soon.

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## D: Cerebral Dominance and Speech: A Historical Perspective

The recognition of the predominant importance of the left cerebral hemisphere for speech is attributed to Marc Dax (1836, cited by Roe D and Finger S 1996).[1] Still, it was generally accepted only after reports of the clinicopathological studies of Broca P (1865)[2,3] and Wernicke C (1874).[4] Around the same time, Hughlings Jackson (1880, cited by Head Henry in 1920)[5] localized propositional and emotional speech in the left cerebral hemisphere. He called it "the leading hemisphere".

Brain WR (1945)[6] pointed out, "The cerebral hemisphere, which is situated in the neural pathways of speech, has become known as the dominant or major hemisphere; it is the left hemisphere in right-handed persons. Questioning himself, he stated "Are the speech centres ever situated in the right hemisphere in right-handed persons". He emphatically said, "Of course," and in support quoted a report by Gardner (1941, cited by Brain WR, 1945,[6] also cited Gardner RC and Lambert WE, 1965).[7]

Ettlinger G *et al*. (1956)[8] extended the criteria for cerebral dominance to include (i) Severe and relatively persistent dysphasia, (ii) dysgraphia, (iii) bilateral dyspraxia, and (iv) the presence of specific parietal lobe signs in particular Gertsman Syndrome resulting from its damage. Lesions of the left cerebral hemisphere, which generally resulted in dysphasia/aphasia and right-sided loss of motor, sensory, and visual field defects, thus reinforced its "dominant" character and relegated the right cerebral hemisphere to a "minor "status.

Interestingly, none of the criteria of dominance described above would satisfy the dictionary meaning of the word "dominance". According to Google, the word dominant implies "having power and influence over others," its synonyms are presiding, ruling, governing, controlling, commanding, ascendant, supreme, authoritative, superior, proponent, assertive, self-assured, and forceful.

Based on extensive evidence available today, one could say that the left cerebral hemisphere is "superior" to the right concerning its function regarding speech and language and, in that limited sense, retains its "status" as the dominant hemisphere.

### Language Laterality and Handedness

According to Goodglass H and Quadfasel FA (1954),[9] the idea of the linkage of handedness and speech was first formulated by Bouillaud in May 1865, when he spoke of "*gaucherie cerebral*," and by Baillarger in May 1865 (cited by Leblanc R in 1865, re-published in Neurosurg Focus in 2019)[10] and Broca in June 1865.[2] (For more historical facts, see Goodglass H and Quadfasel FA 1954).[9] Thus, the occurrence of aphasia with left hemisphere lesions in the great majority of patients, most of whom are right-handed, led to the assumption of dominance of the left cerebral hemisphere. It prompted the converse suggestion that left-handedness would lead to right-hemisphere dominance. However, a series of studies established that even in left-handed individuals, representation of speech in the right hemisphere was rare (Milner M 1952,[11] Milner & Rasmussen, 1954; cited by Rasmussen T & Milner B in 1977,[12] Penfield W 1954,[13] Ettlinger G *et al*. 1956,[8] Subirana A 1958,[14] (1964) [15] Penfield W and Roberts L 1959,[16] Zangwill OL 1960,[17] Wada J and Rasmussen T 1960[18]). A detailed review on "Cerebral Dominance in Sinistrals" has been provided by Ettlinger G *et al*. (1956),[8] who pointed out that, as a matter of fact, "Left-braindedness" might well appear to be the more prevalent form of cerebral organization in left-handed individuals. This finding is supported by the work of Goodglass H and Quadfasel FA (1954)[9] and Zangwill OL (1960).[17] Humphrey ME and Zangwill OL (1952),[19] who studied dysphasia in left-handed patients with unilateral brain lesions (5 right and 5 left hemispheres), supported the hypothesis that cerebral dominance either does not occur in so-called left-handed persons or, if it does, it is less developed than in right-handed person. In addition, he found calculation was permanently impaired in 4 out of 5 patients with right hemisphere lesions.

Ettlinger G *et al*. (1956)[8] concluded that "In contrast, some degree of cerebral ambilaterality may exist in a certain proportion of cases, unilateral representation of speech *usually left but occasionally right* is the most prevalent form of cerebral organization in sinistrals".

Goodglass H and Quadfasel FA (1954),[9] following a detailed study of 320 cases of aphasia (including 123 left-handed with aphasia in 50), concluded, "laterality for language is not identical with laterality for handedness. Handedness does not determine braindedness".

## Clinical Determination of Cerebral Dominance

The risk of speech impairment following surgery on the "dominant" hemisphere led to the search for the criteria for determining it preoperatively. The first criteria were the patient's handedness and, in the broader sense, the family history of handedness. It soon became apparent that while the left hemisphere was, as a rule, dominant for speech in right-handed persons, the reverse was not invariably true [Brain WR (1945),[6] Goodglass H and Quadfasel FA (1954),[9] Ettlinger G *et al*. (1956),[8] Penfield W and Roberts L (1959),[16] Rasmussen T (1964)[20]]. The introduction of the intracarotid sodium amytal test by Wada (1949,cited be Loring DW and Meador KJ, 2019)[21] and later used in a more significant number of patients investigated for surgery for epilepsy by Milner, Rasmussen, and colleagues revealed that while 90 percent of right-handed individuals had speech lateralized to the left hemisphere, only 49 percent of left-handed persons the speech was lateralized to the right and 43 percent to the left hemisphere [Wada (1949, cited by Loring DW and Meador KJ, 2019),[21] Wada & Rasmussen T (1960),[18] Hecaen H and Sanguet J (1971),[22] Searleman A (1977),[23] Branch C *et al*. (1964),[24] Rasmussen T and Milner M (1977),[12]]. Similar findings were reported by Penfield W (1954),[13] Ojemann G *et al*. (1989)[25] using electrical stimulation of the brain to localize speech during surgery for epilepsy.

## Speech and Right Cerebral Hemisphere

According to Basser LS 1962[26] "The dominance of one cerebral hemisphere, *usually the left, in relation to speech, may*

*not be innate and is certainly not immutable".* In support of this statement, Basser quotes many earlier reports on cases of right cerebral laterality for speech associated with right-handedness, including the following. He quotes his own 2 cases (Case no. 29, 30) whose right cerebral laterality for speech was primary from the onset. The evidence in all these cases was concerned with dysphasia resulting from a lesion of the right cerebral hemisphere.

It is interesting to note that even the earlier pioneers (Broca, 1865;[2] GowersW, 1887[27] and John JH A, 1876[28]) believed that the right hemisphere played a significant role in language functioning. Broca (1865)[2,3] defined the speech area to the posterior part of the third frontal convolution, especially on the left, which was essential for speech. Broca advocated that the right hemisphere could even produce speech, particularly automatic speech such as expletives and well-known phrases. Searleman A (1977)[23] has presented detailed reviews of the large amount of scattered evidence concerning the linguistic capabilities of the right hemisphere".

Coltheart M (1983)[29] proposed that the right hemisphere is limited literate in the typical right-handed left-hemisphere dominant individual. This right hemisphere reading system's primary skill is comprehending printed words, especially concrete imaginative words. A later paper argues that the reading performance of deep dyslexic patients derives from the right hemisphere reading system (Coltheart M 2000).[30]

Some specific aspects of speech, like arithmetical operations or music, have been attributed to the right cerebral hemisphere. (Henschen S 1919,[31] 1924[32]), Kahn HJ and Whitaker HA (1991),[33] Critchley M (1953),[34] Dehaene S *et al.* (1999).[35] Some of the authors attributed bilateral distribution of language function in left-handed persons (Goodglass and Quadfasel 1954,[9] Subirana (1958[14]), Hecaen and Sanget (1971).[22] The subject of the bilateral representation of speech has been discussed in detail by Subirana A (1952),[36] Humphrey and Zangwill (1952),[19] and Goodglass H and Quadfasel FA (1954).[9]

Boatman D *et al*. (1999)[37] reported that correct hemisphere speech perception was revealed by amobarbital injection and electrical interference. These and many other studies, thus, reinforced the impression that in most human beings, the left cerebral hemisphere is "dominant" for speech. They *also point out that the right hemisphere is not always completely devoid of speech function*. Gavalas RJ and Sperry RW(1969,[38] also cited in the Nobel lecture, 1981[39]), based on their split-brain studies in humans, commented, "*Right hemisphere was not mute, but indeed a conscious system in its own right, perceiving, thinking, remembering, reasoning, willing and emoting all at a characteristic human level*……."

Further on, they observed, "The left hemisphere was more geared towards language and an analytic thought and calculation, the right hemisphere was more important for understanding spatial pattern and complex sounds like music". *If true, the right hemisphere must play some role in speech-language functions.* Levy J *et al*. (1971)[40] and Gazzaniga MS (1983)[41] further elaborated on the role of the "minor" or right hemisphere in expressive language following brain bisection.

According to Prather JF *et al*. (2017),[42] language is associated with a network of specialized areas spanning frontal, temporal, and parietal regions, and there is a stark asymmetry in the contribution of the left and right hemispheres. For example, the areas in the left hemisphere are associated with focal syntax, temporal properties of speech, and the brief transitions present in sound-composing speech. In contrast, corresponding regions in the right hemisphere are more closely associated with prosody, spectral properties of speech, and emotional valence of the sounds used in vocal communication.

Most information regarding linguistic capabilities of the right cerebral hemisphere in right-handed persons has been obtained from patients undergoing hemispherectomy for infantile hemiplegia, developmental defects of the brain (cortical dysplasia), and hemimegenencephaly. Surge-Weber Syndrome, Rasmussen Syndrome, intractable epilepsy, and brain tumors. Clinico-pathological studies of aphasics and dyslexic and, more recently, psychophysiological and non-invasive neuro-imaging studies using fMRI, PET Scan, MEG, SPECT, and near-infrared spectroscopy have added valuable information.

## Speech Following Hemispherectomy

It was common knowledge that the right hemisphere, being "non-dominant" injury, disease, or surgical damage, does not produce dysphasia compared to the left hemisphere. Hence, surgeons avoided operations on the left hemisphere. Dandy (1928, cited by Torres I *et al.*, 2021)[43] was the first to perform a hemispherectomy for a brain tumour, soon followed by Gardner's (1941) right hemispherectomy for brain tumors (1928, cited by Bahuleyan B *et al.*, 2013).[44] McKenzie (1938, cited by Bahuleyan *et al.*, 2013)[44] reported a patient who had right hemispherectomy done for epilepsy. "There was no speech defect while they had or developed left hemiplegia". Roberts (1951, cited by Penfield and Roberts, 1959),[16] analysing operations for epilepsy by Penfield, observed that aphasia followed operations on the right hemisphere only twice in 258 right-handed patients and three times in 23 left-handed patients. Ettlinger *et al*. (1955)[8] reported a 35-year-old patient developing dysphasia following a right temporal lobectomy in a right-handed man, indicating that this patient's right cerebral hemisphere was "dominant". They mentioned that "we have been able to trace references to 15 cases in which aphasia has been found in association with right-sided lesions in right-handed patients". These cases have been summarised in a table by them.

On the other hand, Zollinger (1935, cited by Bahuleyan B *et al.*, 2013)[44] reported a case of left cerebral hemispherectomy for an infiltrating tumor in a 43-year-old right-handed woman who had right hemiparesis and dysphasia pre-operatively. "The most interesting observation during the postoperative period was the ability of the patient to speak". She was utterly aphasic when she was discharged from the hospital. However, she improved for two weeks, "conversing with relatives, she could talk intelligently ". The interesting question was "Could she have a bilateral representation of speech?".

Krynauw (1950, cited by Bahuleyan B *et al.*, 2013)[44] aroused new interest in this field by performing hemispherectomy on 12 patients suffering from infantile hemiplegia, showing reasonable control of seizures, improvement in motor power, and marked

improvement in personality, behaviour, and mentality. He pointed out that "with regards to the language function. young children who have learned to speak and then develop infantile hemiplegia may lose this function. In other cases, speech may be little disturbed". More details about the outcome of dominant hemispherectomy for brain damage in early childhood, including those with infantile hemiplegia, are described below.

## Hemispherectomy for Infantile Hemiplegia: Early Childhood Hemisphere Injury, Developmental Defects (Sturge-Weber) Rasmussen Encephalitis, Intractable Epilepsy

Following the report by Krynauw RA (1950)[45] that removal of the left hemisphere for infantile hemiplegia resulted in no speech disturbance, a series of studies on left (dominant) hemispherectomy for a variety of lesions in childhood confirmed this valuable observation [Cairns and Davidson (1951, cited by Griffith H and David M, 1966),[46] McKissock W (1953),[47] Gardner *et al.* (1955, cited by Smith A, 1966),[48] Goldstein R and Goodman (1961),[49] McFie J (1961),[50] Smith A (1966),[48] Carmichael (1966, cited by Carson BS *et al.*, 1996),[51] Rasmussen T (1978-1988, cited by Carson *et al.*, 1996),[51] Tinuper (1988, cited by Carson BS *et al.*, 1996),[51] Strauss & Verity (1983, cited by Carson BS *et al.*, 1996),[51] Davies *et al.* (1993, Carson BS *et al.*, 1996),[51] Patterson K *et al.* (1989),[52] White HH (1961),[53] Griffith (1967, cited by Carson BS *et al.*, 1996),[51] Wilson PJ (1970)[55]].

In an extensive review on cerebral hemispherectomy (CH) in the treatment of Infantile Hemiplegia (IH), White HH in 1961[53] reported that a total of 267 cases of CH for IH had been recorded in the world literature. Of these, 150 documented in sufficient detail were analysed in Table II) and the remaining 117 were arranged in tabular form. The paper provides a wealth of information on IH and its management, including a detailed list of references. The critical messages that emerge from this study are as follows:

Aphasia does not result from cerebral hemispherectomy/ hemispheric surgery (carried out for infantile hemiplegia), regardless of which hemisphere is removed.

The below points may be noted with interest.

- Functional sites, like motility and sensibility, for speech, praxis, and gnostic modalities, are reallocated to other healthy brain areas in the development of infantile hemiplegia
- This transference of function can be spontaneously accomplished only before cerebral dominance is established.
- The age of establishment of cerebral dominance and the degree to which this dominance occurs are, without doubt, extremely variable among individuals.

Disorders such as those accompanying parietal lobe disease are acquired in adulthood (constructional apraxia, Gerstmann's syndrome, disorder of body-image, disorders of spatial and symbolic thought) are not seen following CH for hemiplegia of childhood.

Basser (1962),[26] following a critical study of one hundred and two cases of hemiplegia of early onset (48 with left hemisphere lesions and 54 right hemisphere lesions), 35 of whom (17 on the left and 18 on the right) observed that speech was developed and maintained in the intact hemisphere and in this respect the left and right hemispheres were equipotential. His overarching conclusion on his study is very relevant to the subject-under review here: *"Although it is one of the most constant and remarkable phenomena of human cerebral physiology, the dominance of one cerebral hemisphere usually the left, in relation to speech, may not be innate and is certainly not immutable, as is shown by the development of speech in cases of massive unilateral cerebral hemisphere lesions occurring early life."*

Commenting on the normal development of cerebral laterality for speech, the author reported, *"The relative frequency of speech disturbance following right hemisphere lesions in right-handed children, as compared to adults, suggest that often both hemispheres participate in the development of speech before lateralization takes place to the left hemisphere* (Quotes his cases 31, 32, 33 to illustrate this). Carmichael EA (1966)[54] reported similar findings, i.e., no loss of speech function irrespective of the right or left-sided hemispherectomy in his group of patients.

Levy J (1969),[55] who studied the speech of patients whose neocortical commissures had been surgically divided for the treatment of epilepsy, commented, *"There is evidence that the minor hemisphere possessed some minimal ability to express language, but it is difficult to observe because of competition from the major hemisphere for the control of the motor mechanism for the production of language."*

Wilson PJ (1970)[56] analysed 50 cases of hemispherectomy (30 right, 20 left) carried out by McKissock primarily for intractable epilepsy in children suffering from infantile hemiplegia. All but 2 youngest patients had either acquired speech before surgery or were in the process of doing so. Forty-two patients (84%) retained unimpaired speech function irrespective of whether the right or left hemisphere was removed. The two children who had not acquired speech before the operation (Right and Left hemispherectomy) at the ages 1½ and 2½ years, respectively, subsequently developed perfectly normal speech. Dysphasia or aphasia occurred postoperatively in 6 patients, all but one following left hemispherectomy; in half, the loss of speech was permanent.

More recently, Vining E (1999),[57] reported the outcome of 58 children after hemispherectomy carried out at the Johns Hopkins Hospital between 1968 and 1996 (27 for Rasmussen syndrome, 24 for Cortical dysplasia, hemimegalencephalies, and 7 for Sturge-Weber Syndrome and other congenital vascular problems). Though the authors do not expressly, report on the effect of the operation on speech, they indicate their general satisfaction as also of the parents with the outcome and that all the surviving "children were at school". They discussed the reason for improving motor function after hemispherectomy and "why language recovers after removal of the dysfunctional left hemisphere".

## Speech Following Hemispherectomy for Brain Tumour

Gardner WJ *et al.* (1955),[58] comparing the residual function following hemispherectomy for tumour and infantile

hemiplegia, found their case no. 7, a left-handed 10-year-old girl undergoing left hemispherectomy for a glioma," could speak though with some hesitation and exhibited some degree of anomia and alexia. One year later the speech was more spontaneous".

Hillier WF (1954)[59] reported a 14-year-old right-handed boy who underwent a left hemispherectomy for malignant glioma 27 months after the surgery had quite accurate comprehension of spoken words. He was capable of reading individual letters but could not recognize words. He had some anomia and motor aphasia, which were rapidly improving.

McFie J (1961)[50] pointed out that on psychological testing of patients with unilateral hemisphere damage, the pattern of deficit was different in children and adults. "The more significant defect in the digital symbol test with right hemisphere lesions (in children) was not found in adults, and the considerable mean deficit on the vocabulary test with lesions in either hemisphere was altogether different from the findings in adults, who showed no mean deficit on this test with lesions in any location. Before lateralization of speech is completed (largely by 5 years), either hemisphere can support language (Brain 1945,[6] Zaidel E 1976,[60] Searleman A 1997[23]).

Smith A and Burklund CW (1966)[61] reported a 47-year-old male submitted to left hemispherectomy for a malignant glioma who preoperatively had moderate expressive dysphasia. Immediately following the operation, the patient "developed severe receptive and expressive aphasia". On the 6th post-operative day, he could speak a few words in response to a question, but not spontaneously. His speech continued to improve till 181 post-operative days (Details of the psychometric examinations at different intervals are tabulated). Although speech was severely impaired, speech and verbal comprehension were present (and not abolished), and *"Since speaking, writing, understanding language showed continuous improvement" after left (dominant) hemispherectomy, "the right hemisphere apparently contributed to all these functions although in varying proportion"* (italic added by present author). The receptive language functions were initially less impaired and showed more significant recovery than expressive language. The author quotes similar findings by Zollinger R (1935)[62] and Crockett and Estridge (1951, cited by French LA *et al*., 1955[63]). Gott PS (1973),[64] who investigated linguistic and related cognitive abilities two years after a dominant left hemispherectomy for a brain tumour in a 12-year-old girl, concluded that" language mechanisms in the cerebral hemisphere were not just at a low level of development of functions found in the dominant hemisphere but were modified as a result of interference by the pre-existing spatio-temporal system." She remarked that "the right hemisphere readily exhibits personality characteristics such as humor, boredom, love, and frustration to be substantially the same as before surgery" and *"the right hemisphere seems capable of making association and in particular musical mode of expression. Auditory comprehension of speech comprehension of verbal speech was one of the least impaired language functions.* "When she awoke from surgery, she was conscious and could sing and speak single words, for instance. her sister's name. She could count correctly to 30 or 40 could perform simple addition". Obviously, after a total left (dominant) hemispherectomy, though dysphasic, she retained several aspects of linguistic functions which are obviously attributable to the right cerebral hemisphere.

Jerre Levy-Agresti (1968, cited by Levy J, 1969)[65] pointed out that there is evidence that the minor hemisphere possesses some minimal ability to express language. Still, it is difficult to observe because of competition from the major hemisphere to control the motor mechanisms for language production. Psychological testing after forebrain commissurotomy for therapeutic reasons clarified the nature of functional differences in the two hemispheres. The data indicate that the mute, minor hemisphere specializes in Gestalt perception, primarily synthesizing information input. The speaking major hemisphere, in contrast, seems to operate in a more logical, analytic, computer-like fashion. Its language is inadequate for rapid complex synthesis achieved by the minor hemisphere (Comment: Does this account for RT hemisphere being better concerning music and arithmetic?).

Searleman (1977),[23] in a detailed review of right hemisphere linguistic capabilities, concluded, *"In recent years' evidence has accumulated that the right hemisphere also possesses linguistic skills".*

Patterson K *et al*. (1989)[52] reported a 13-year-old right-handed girl who underwent a left hemispherectomy for epilepsy. Preoperatively, she had expressive dysphasia and right hemiparesis. Postoperatively, her "spontaneous. speech was very limited both in content and grammatical form, perhaps more anomic than classical agrammatic, *"but the reading pattern was by no means one of undifferentiated impairment."*

St James Roberts I (1981),[66] after an extensive review of hemispherectomy literature, reported "considerable variability of language function in left hemispherectomy cases independent of the age of onset", obviously attributing language function in the right hemisphere.

Several psychologists and cognitive scientists including Moscovitch M *et al.* (1981),[67] Moscovitch M (1981),[68] Paterson K and Basner D (1984),[69] Max Coltheart (1980, 1983 and 1985),[29,30] Patterson k *et al*. (1989),[52] Gazzaniga MS (1983),[41] Zaidel E (1976),[60] Zaedel E and Schweiger A (1985)[70] hotly debated the role of right hemisphere in reading. While no unanimity could be arrived at since the opinion was based on psychological investigations of a few patients of the split-brain, deep or pure alexia (alexia without agraphia), left/right hemispherectomy such distinguished investigators found it fit to discuss the role the right hemisphere played in linguistic activity indicates that under certain circumstances, the subject is worth serious consideration.

Rabinowich B and Moscovitch M (1984)[71] and Coltheart M (1980, 1983)[29] proposed that in the typical right-handed, left-hemisphere dominant individual, the right hemisphere is, with limitation, literate. This right hemisphere's primary skill is comprehending printed words, especially concrete/imageable words. He argued that the reading performance of deep dyslexic patients derives from the right hemisphere reading system. In a series of papers, Costlett discussed the right hemisphere's role in reading "Pure alexia", optic alexia, and evidence from transcranial magnetic stimulation (Costlett HB and Saffran EM 1989,[72] 1992,[73] Costlett HB and Monsul N

1994[74]). Coltheart M (2000)[30] again described right hemisphere reading in deep dyslexia patients. Kahn HJ and Whitaker HA (1991)[33] and Gitelman D (2003)[75] discussed the same in patients developing acalculia following cerebral lesions. Coltheart M (1985)[29] opined "that no satisfactory evidence exists that the right hemisphere of the normal brain plays any role in normal reading or is even capable of doing so. There is no doubt that certain reading tasks can be performed by the right hemisphere of the commissurotomy patients studied exclusively by Zaidel". An intriguing, generally inconclusive" debate regarding the role of the right hemisphere at least in respect to reading, followed (Zaidel E 1976,[60] 1977,[76] Zaidel E and Schweiger A 1985,[70] Gazzaniga MS 1983,[41] Levy J *et al*. 1971,[40] Levy J 1983,[77] Patterson K and Besner D 1984,[69] Coltheart M 2000,[30] Chiron C *et al*. 1997[78]). Dennis and colleagues, who studied three patients in detail, two with left and one with right hemispherectomy for intractable seizures, found that "all three patients could speak and read and had verbal IQ in the normal range. Nonetheless, the two left hemidecorticates were inferior to the right hemidecorticate on various linguistic tasks that required complex syntactic or inferential abilities.

## Laterality of Speech

Levy J (1969)[65] tried to provide further information on the possible basis for the evolution of lateral specialization of the human brain.

Wada JA *et al*. (1975)[79] stated, "Little is known about when and how functional lateralization (of speech) develops in the human brain," even though the neuroanatomical studies had indicated that the structural basis for it was already seen at the 29 gestational weeks".

A recent paper by Chiron C *et al*. (1997),[78] who utilized cerebral functional imaging using SPECT, makes a very unusual statement, "These findings support the hypothesis that in man the right hemisphere develops its function earlier than left."And, "Between 1-3 years of age, the blood flow shows a right hemisphere predominance mainly due to the activity in the posterior associative area. Asymmetry shifts to the left after 3 years". While its significance regarding speech and language acquisition is unclear, the paper states, "The right brain hemisphere is dominant in human infants". There has been no independent confirmation of these observations.

Boatman D *et al*. (1999)[37] investigated language recovery after left hemispherectomy in right-handed children (age 7-14 years) with late-onset seizures; the children had at least 5 years of normal language development before the onset of seizures. Within 4 to 16 days after surgery, patients showed improved phoneme discrimination compared with their performance just before surgery; other language functions remained severely impaired until at least 6 months after surgery. By 1 year after surgery, receptive functions were comparable with or surpassed patient pre-surgery performance. The language function observed after the left hemispherectomy was attributable to the right hemisphere.

Devlin AM *et al*. (2003)[80] studied clinical outcomes of hemispherectomy for epilepsy in childhood and adolescence (33 children aged at surgery 0.33 to 17 years). They observed seizure-free or rare seizures postoperatively in >75 percent of cases, and hemiplegia remained unchanged. While no details are provided regarding language function, there was no significant cognitive deterioration or loss of speech function following right-sided or left-sided hemispherectomy. The authors quote similar findings from Wilson PJ (1970),[56] Peacock WJ *et al*. (1996),[81] and Vining EP *et al*. (1997).[82]

Summarising, an extensive review of the literature establishes that at birth, speech is represented in both cerebral hemispheres probably equally. However, its anatomic substrate from the 29-30 weeks gestational period already exists predominantly in the left perisylvian area. It progressively gets literalized to the left hemisphere; the exact age by which it gets lateralized is not firmly established. Most studies suggest the age of 5 years, but several reports have found this period to extend to 10-12 years. The neural network involved in speech perception and expression continues expanding until adolescence.

Recent advances in neuroimaging techniques have demonstrated the participation of areas beyond the classical Broca's and Wernicke's areas to anterior cingulate, a large part of the temporal cortex, insular cortex, left medical extrastriate cortex, supplementary motor area, and cerebellum (Fiez JA *et al*. 1996[83]).

Developmental hemispheric disorders like Sturge-Weber Syndrome, hemimeganencephaly, cortical dysplasia, and diseases like infantile hemiplegia and Rasmussen syndrome, etc. result in hemiplegia, Rasmussen syndrome. results in an early transfer of laterality. Hence, hemispherectomy of either hemisphere does not result in speech disorder.

A few individuals, even right-handed ones, have speech representation in the right hemisphere, while others retain bilaterality. No explanation is available for it. Much evidence indicates that the right hemisphere is not devoid of at least some aspects of speech function in normal individuals. The paper highlights some of these. Future studies will elucidate their clinical significance.

## Conclusion

It is now generally recognized that speech is lateralized chiefly to the left hemisphere, not only in right-handed individuals but in a percentage of even left-handed ones. However, there is enough evidence to show that the non-dominant right hemisphere is involved in many language operations. Recent non-invasive neuroimage studies and earlier clinical and brain studies indicate that the non-dominant hemisphere understands many words, especially concrete nouns, and suggest that it is involved in other aspects of language processing. The best examples for these operations are those about processing the discourse level of language, interpreting how literal languages such as metaphors, and appreciating the tone of a discourse, for example, if it is humorous (Caplan D 2017).[84]

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# Functional Development of Speech in Human Brain

"Our understanding of speech recognition processes has gradually advanced over the past 50 years from almost total ignorance to well-informed confusion……..."
(Kuhl 2011).[1]

For nearly a hundred years after Broca P (1861)[2,3] and Wernicke C (1874)[4,5] localized speech in the left cerebral hemisphere, based on their clinico–pathological observations on just a few cases, it came to be accepted as a gospel truth. No doubt, most well-known neurologists around this era, including Dejerine J,[6] Marie and Charcot from France (cited by Engelhardt E and Gomes MD, 2015);[7] Lichtheim L,[8] Liepmann (cited by Rothi *et al.*, 2004),[9] Pick (cited by Hodges J, 2017),[10] Henschen S,[11] Goldstein K,[12] and Kleist K [13] from Germany; Jackson JH,[14,15] Gowers W,[16] Head H,[17-19] Wilson PJ,[20] and Nielson KA *et al*.[21] from England, further elaborated and confirmed these observations. However, these studies did not provide significant information about the ontogenic development, timing, precise anatomic structure, distribution, and functional organization of the neural substrate of speech.

The initial neuroanatomic studies got a further impetus in the later part of the 20th century and the current era by the development of newer disciplines-neuropsychology, cognitive science, linguistics on one hand and non-invasive neurophysiological investigations (EEG, Event-Related Potentials, magnetoencephalography, and neuroimaging techniques (fMRI, PET, SPECT, Near-Infrared Spectroscopy).

However, as late as 2011, Kuhl PK [22] commented that we are still just breaking ground regarding the neural mechanism that underlies language development and its critical period. In the last decade, brain and behavioural studies indicated a complex set of interacting brain systems in the initial acquisition of language early in infancy, many of which reflect adult language processing (Kuhl PK, 2003,[22-26] 2011,[22] Dehaene Lambertz G *et al.* 2002,[27] 2006,[28] Dehaene Lambertz G 2011,[29] Dehaene S *et al.* 2010,[30] Newman RS and Jusczyk PW 1996,[31] Barker BA and Newman RS 2004[32]).

Neuro-behavioral studies in infants indicated that considerable language learning is already taking place in the first year of life in phonology, prosody, and word segmentation. Event-related potential studies suggest that the temporal lobes contain neural circuits for phoneme description, which become attuned to the mother language during the first year of life (Reinholtz J and Pollman S 2005,[33] Barker BA and Newman RS 2004[32]). It is claimed that after birth, behavioural studies indicate that infants already recognize the mother's voice (DeCasper AJ and Fifer WP 1980[34]). This has been confirmed by Dehaene-Lambertz G (2010),[30] who, using fMRI, observed several differences in the activation measured when infants were listening to their mother's voice compared to the voice of other women. It was observed that the unknown voice activated the mother's voice-activated prefrontal cortex and the orbitofrontal cortex. Sebastian-Galles N (2006)[35] observed that infants show facilitation for native language phonetic perception between 6 and 12 months, while they can learn from first-time natural exposure to a foreign language at 9 months. The present review deals with these advances in our understanding of the neural substrate of human speech.

## Neuroanatomical Studies

Levitsky W and Geschwind N (1968)[36] were the first to provide an anatomical explanation of the left-sided location of Wernicke's speech area. A study of 100 adult human brains obtained at autopsy found marked anatomical asymmetries between the planum temporale (upper surfaces) of the right and left temporal lobes by naked eye measurement. It was more prominent on the left side in 65% of the brain and only in 11% on the right. They concluded that planum temporale contains the auditory association cortex on the left, which constitutes the classical Wernicke's area.

Mullan S and Penfield W (1959),[37] based on electrical stimulation of the brain in conscious patients during epilepsy surgery, had already reported this area to be of significant importance in language function. Interestingly, in 1962, Vernon Mountcastle (cited in Mountcastle, 1998),[38] while discussing the cerebral *domain*, assumed "that perhaps some subtle structural asymmetry might exist related to the functional differentiation" of the two hemispheres concerning speech.

Wada JA *et al.* (1975)[39] confirmed the cerebral asymmetry in cortical speech zones in 100 adults and 100 infant brains. They found this asymmetry in the planum temporale and the frontal operculum (Broca's area). They found that these areas become measurable at the 29th week of gestation. There was evidence of subsequent differential development of the planum in favour of the left, with the left planum larger than the right. In addition, they found that while both the frontal operculum and left planum were always present, the right planum ranged in size from absent (10%) to more prominent than the left (about 10%). The findings suggest that more persons may have a right-sided dominance or bilateral representation of speech than previously assumed. It may be pointed out that cases of right cerebral laterality for speech associated with right-handedness had been recorded by (Collier, 1899; Souques, 1910; Mendel, 1912; Claude and Schaeffer, 1921; Wilson, 1926; all cited by Basser LS 1962).[40]

They commented, "Little is known about when and how functional lateralization develops in the human brain. Zangwill OL (1960),[41] based on a review of hemispherectomy

in early childhood, stated: All that can be said with any competence is that cerebral dominance appears to evolve *pari passu* with the development of speech and to be fully established well before the advent of maturity. Witelson SF and Rabinovitch MS (1972)[42] demonstrated auditory linguistic sensitivity in early infancy. Anatomical measurement of the language mediating area of the left and right planum temporal in 14 neonatal and 16 adult human brains by Witelson SF and Pallie W (1973)[43] revealed the left-sided area to be significantly larger in neonates than in adults. They suggested that neonatal asymmetry indicates that the infant has a pre-programmed biological capacity to process speech sounds. Interestingly, the anatomical asymmetry in language area is present at an age when bilateral representation and hemispheric transferability of speech are still present (Basser LS 1962,[40] Rasmussen T 1964[44]).

DeCasper AJ and Fifer WP (1980),[34] in a study of newborns, observed that human responsiveness to sound begins in the third trimester of pregnancy and by birth reaches sophisticated levels (i) especially concerning speech, (ii) early auditory competency probably subserves a variety of development functions such as language acquisition and mother-infant bonding.

Chi JG *et al*. (1977),[45] in a study of gross photographs of 507 brains and serial section of 20 brains from infants of 10 to 44 weeks gestational age, confirmed the left-right asymmetry of the transverse temporal gyrus, Sylvian fissures and planum temporale. The left Sylvian fissure was longer than the right and helped to provide more space for the płanum temporale.

Discussing the Anatomical Basis of Language (in Man) Geschwind N [46-48] highlights the significance of the association cortex (as opposed to primary projection areas eg auditory/visual areas). In man as opposed to lower mammals. In this connection, he quotes Flechsig (1901).[47,48] He introduces the significance of the human inferior parietal lobule, which includes the angular and supramarginal gyrus, to a rough approximation of areas 39, 40 of Broadmann. According to Critchley M (1953),[49] even in higher apes, these areas are present only in rudimentary form. This region matures cytoarchitectonically very late, often in late childhood. According to Bonin and Bailey (1961, cited by Chusid JG, 1964),[50] "The part of the brain which increases in man most strikingly is not the frontal lobe but the parietal and temporal lobes----------and it is here that we should look for the substrate of certain functions which are supposed to be characteristic of man." This includes speech. He proposed that "Language development is probably heavily dependent on the emergence of the parietal association area since at least in what is perhaps its most straightforward aspect (object naming) language depends on associations between other modalities and audition. Early language experience, at least, most likely depends heavily on the forming of somesthetic-auditory and visual-auditory association, as well as auditory-auditory association and he quotes Critchley's(1953)[49] comment, "that it is tempting to associate growth of the posterior parietal region with the development of speech".

Geschwind N[46-48] states, "It is also not unlikely that the development of cerebral dominance is related to greater development of this new parietal association area". He adds that "this dominance depends on enhanced activity of the left speech area. The most important part of this area is the middle and posterior portion of the superior temporal gyrus, which are part of the auditory association area and form the classical Wernicke's area. Connexions from other sensory modalities, at least vision and somesthetic sensations, are assumed to come to this speech zone through the angular gyrus region.

## Role of Splenium and Corpus Callosum

Geschwind N (1965)[47,48] describes cases of pure alexia without agraphia, a clue to the involvement of corpus callosum. In contrast, some patients with alexia without agraphia could spell and comprehend simple spelled words. This patient had normal spontaneous speech. However, he was unable to Spell even the simplest words. Similarly, although he understood complex spoken sentences, he could not understand even three-or four-letter words when spelled to him.

This disturbance of spelling gives us a handy clue as to the function of the part of the angular gyrus involved in "visual word memory". It is the region which turns written language into spoken language and vice versa. Dejerine J (1892)[6] described one of the earliest cases of alexia without agraphia. Soon after, several such cases were described with detailed autopsy findings. In several such cases, the lesion involved the left occipital cortex and the splenium of the corpus callosum. The splenium lesion disconnects the right visual region from the angular gyrus.

Geschwind N (1965)[46-48] quotes a patient of Foix and Hillemand (1925), whose autopsy found an infarct of the left visual field cortex without involvement of the splenium, had no alexia in life. The author provides several other references in support of the role of splenium in this syndrome. The author describes various pathways possibly subserving this function. The author suggests the possible function of the angular gyrus as a visual memory centre for words by acting as an area for forming and storing cross-modal associations involving more than words. Speculating the basis of the failure of reading acquisition in cases of so-called congenital dyslexia. The author proposed it to be due to delayed development of the angular gyrus region, probably bilaterally.

Discussing a variety of syndromes like "Pure word deafness", "Tactile aphasia", "Pure Word Blindness, etc., the author attributes these to result from disconnection (rather than damage to the cortex).

Around the same time, Eimas PD *et al*. (1971)[51] started to study speech perception in infants. They found that infants as young as 1 month of age are responsive to speech sounds and able to discriminate and perceive speech sounds along the voicing continuum in a manner in which adults perceive the same sound. This implies that it must be part of the biological makeup of the organism.

Johnson MH (2001)[52] proposed that functional specialization of different regions of the human cerebral cortex arises through intrinsic genetic and molecular mechanisms. That experience merely has a role in the final 'fine-tuning'. The interaction

of genetic and experiential effects on human neurocognitive development was reported by Neville H *et al*. (2011).[53]

## Neuroimaging Studies

The introduction of non-invasive neuroimaging techniques, fMRI, PET, SPECT, and Near-Infra-Red Spectroscopy in the last decades of the 20th century opened new vistas to explore the physiological (functional) anatomy of the human brain. In 1987, Chugani HT *et al*.[54] published a positron emission tomography (PET) study of the functional development of the human brain. Around the same time, Martin E *et al*. (1988)[55] reported an MR study of the development stages of the human brain. Other studies on functional localization of speech, language, and other cognitive functions soon followed (Cabeza R and Nyberg L 2000).[56,57]

Prather JF *et al*. (2017)[58] state that separate but continually interacting neural networks underlie vocal production, sensory-motor learning, auditory perception, and memory. These networks are responsible for interaction between the Wernicke's and Broea's areas. These networks serve much more complex roles than just production or perception of speech. They are involved in the syntactic and semantic aspects of language and interact through several connecting pathways (Friedericia AD 2002, 2011).[59,60] Human newborns show increased activity in the superior temporal lobe but not in the inferior frontal cortex in response to human speech (Imada T *et al*. 2006).[61] An fMRI study in a 3-month-old infant showed activation of Wernicke's and Broca's areas in response to hearing speech (Dehaene-Lambertz G *et al*. 2006[27]). Their findings suggest that Wernicke's area is an integral part of the neural substrate for speech perception in neonates and that Broca's area becomes active later when infants start babbling.

Gaillard WD *et al*. (2000)[62,63] investigated the functional anatomy of cognitive development of verbal fluency in 10 normal children and 10 normal adults using fMRI. They found the predominant neural network underlying language function to lie in the left hemisphere in nearly all normal adults, irrespective of their handedness. In a verbal fluency test, normal children and adolescents had cortical activation patterns similar to those of adults. However, a few normal adults had a mixed pattern showing right-sided inferior frontal gyrus dominance or bilateral representation.

In a detailed study of cerebral localization of language in 50 left-handed and 50 right-handed normal adults, Pujol J *et al*. (1999)[64] found 96 percent of right-handed subjects showed fMRI changes lateralized to the left hemisphere, whereas 4% had bilateral activation patterns. In contrast, left hemisphere lateralization occurred in 76% of left-handers, bilateral activation occurred in 14% and right hemisphere lateralization was found in the remaining 10%.

Dehaene–Lambertz G (2000),[27,30] and her colleagues carried out a series of fMRI studies on cerebral localization of various aspects of speech and language in neonates and young infants.

Some of their significant findings are summarized below:-

- An adult-like structure of fMRI response was observed in 3-month-old infants listening to their mother's language along the superior temporal region.(Dehaene-Lambertz G *et al*. 2002).[27]
- The cerebral specialization for speech stimuli in infants was different.(Dehaene-Lambertz G 2000).[65]
- The fact that Broca's area was active in infants before the babbling stage implies that activity in the region is not the consequence of sophisticated motor learning but, on the contrary, that this region may drive, through interaction with the perceptual system, the teaching of the complex motor sequences required for future speech production.
- Their observations suggest the existence of a complex hierarchical organization of the human brain in the first months of life, which may play a crucial role in language acquisition in our species (Dehaene-Lambertz G *et al*. 2006).[28]
- Human infants acquire their native language in the first months of life.
- Their findings showed that the infant cortex is already structured into several functional regions, as adults listening to speech activated a large subset of the temporal lobe with a significant left hemisphere dominance.
- Their studies indicate an early functional asymmetry in the capacity to process speech in the two hemispheres.
- They discussed the two mechanisms of language acquisition, one being its genetically determined mechanism and the other being that the infant's brain is initially immature and plastic. Exposure to speech inputs progressively shapes its organization through the domain-general mechanism of learning and plasticity (Dehaene–Lambertz G 2006).[66]
- Eimas PD *et al*. (1971)[51] have suggested that aspects of speech perception may be biologically programmed at an unexpectedly early age.
- This group studied several other aspects of speech and language in infants (Dehaene S *et al*., 1998,[67] 1999;[68] Dehaene-Lambertz G, 2011[29]).

On the receptive side, the onset of hearing occurs in utero. Based on sound, they evoked responses from 263 foetuses. Birnholz JC and Benacerraf BR (1983)[69] found the onset of hearing to occur between 26 and 28 weeks of gestation. However, the sensitivity to tones in 20-23 dB remains poor till 2 years of age. Auditory skills improve slowly till early adolescence (Werner LA, 2019).[70]

In contrast, vocal production begins shortly after birth, and infants produce speech syllables containing consonant bowel pairs, e.g. 'mama' and 'da-da' at about 6 months, called canonical babbling (Werner LA, 2019).[70] Battro AM *et al*. (2011)[71] summarised their observations on non-invasive imaging methods and behavioural measurements. These revealed both highly structured early organization of brain networks for language with hemispheric specialization and its fast maturation in the first months of life. However, they observed that brain maturation continued in adolescence and early adulthood. Initially, diffuse networks become more segregated and focused. Recent evidence has indicated that neural pathways, dendritic trees, synaptic pruning and even gene expression are modified in millions of neurons as a function of learning experience. Infants initially hear all phonetic differences and have a universal phonetic capacity at birth (Eimas PD *et al*. 1971).[51] Between 6 and 12 months of age, native discrimination declines, and native language speech perception

significantly increases (Kuhl PK *et al*. 2006,[72] Kuhl 2010,[25] Knudson RC 2015[73]). Several studies have brought out the significance of the mother's voice in the development of speech acquisition (Barker and Newman 2004,[32] Dehaene-Lambertz *et al*. 2002,[27] Dehaene-Lambertz G 2011[29]).

Newman RS and Jusczyk PW (1996)[31] demonstrated that infants can attend to a female voice while a male voice speaks simultaneously in the background. Barker BA and Newman RS (2004)[32] further established that infants could segregate female voices, but only when one (usually the mother) was well-known to them. Learning is undoubtedly involved at every stage of development, from the production and perception of vowels to the syntax of a sentence. English-speaking infants typically display word comprehension by 8-10 months, with median comprehension increasing from 54 words at 11 months to 160 words at 16 months. In contrast, median word production is minimal before 12 months and rises to a median of 40 words by 16 months. Thus, speech production lags behind comprehension.

Vocal production begins shortly after birth, and infants produce speech syllables containing consonant-vowel pairs (e.g., mama or da) at about 6 months, called canonical babbling. Most normal-hearing infants displayed canonical babbling before 11 months, whereas congenitally deaf infants did not begin to babble until a much later age.

Language is often viewed as an isolated "modular activity that is separated from other, more general human systems. Goldstein MH *et al*. (2003)[74] and several others suggest that language emerges in infants by relying on a broader set of perceptual, cognitive and social skills.[75,76]

Recent neuroimaging studies repeatedly observed that a superior temporal region in *both hemispheres* activates speech more strongly than no-speech sounds like tones and noise (Demonte V 1992,[77] Zatorre RJ *et al*. 2002,[78] Binder JR *et al*. 1996,[79] 2000[80]). What was initially surprising was the location of this new "speech centre. Whereas the conventional neuroanatomic model of language processing emphasized the importance of the posterior part of the superior temporal gyrus, the specific activation lay Antero lateral to the primary auditory cortex and anterior to the midpoint of the gyrus. By contrast, the planum temporale, an area on the posterior superior temporal gyrus that has long been considered a speech centre, did not prefer speech sounds (Binder JR *et al*. 1996[79]). This view has been supported by Scott SK *et al*. (2000).[81] The lack of withstanding reference to the role of the right hemisphere in the speech process mentioned by several authors here remains debatable. (See chapter Cerebral Dominance).

Hickok G (2014)[82] referred to the persistent loss of consensus on the neurology of speech perception. The authors argued that at least two distinct pathways participate in speech perception in a dependent manner, and they are more strongly localized to the left hemisphere. The first is a ventral pathway, which probably involves the cortex near the tempero-parietal occipital function. This pathway is essential for interfacing sound-based representation of speech with widely distributed conceptual relationships and, therefore, is engaged in tasks that require access to the mental lexicon. The second is a dorsal pathway. This pathway appears to play a significant role in tasks that require explicit access to specific lexical speech segments.

## Speech Perception

Binder JR *et al*. (1996),[79] summarising a large number of studies, pointed out that the anatomy and physiology of speech perception have been studied with a variety of methods, including cytoarchitectural mapping of the auditory cortex (Galaburda A and Sanides F 1980),[75,76] behavioural-anatomical correlation in the brain injured (Goldstein 1973[83]), evoked potentials to auditory stimuli (Celsia GG 1976,[84] Hari R 1991[85]), mapping of rional blood flow and metabolism changes during the audition (Mazziota JC *et al*. 1981[86]), PET Scan and fMRI (Binder JR *et al*. 1994[87]) Incorporating the findings of these diverse investigations suggested a significant role for perisylvian temporal cortex in the analysis of auditory speech stimuli (Goldstein MN 1974,[88] Hari R 1991,[85] Wise R *et al*. 1991,[89] Petersen SE *et al*. 1989,[90] Zatorre RJ *et al*. 1992[91]).

Both the right and the left temporal regions appeared to participate to some degree (Hari R 1991,[85] Mazziotta JC *et al*. 1981,[86] Wise R *et al*. 1991[89]). Using fMRI studies in response to their subjects passively listening to speech and non-speech stimuli consistently observed superior temporal gyrus activation in all subjects. A significant finding of this study was that speech stimuli activate a spatially more extensive region of the temporal lobe than does unmodulated noise and different speech stimuli (words, non-words).

Recent advanced neurolinguistic and neuropsychological studies have elaborated on various unique features, other than phonetics, that characterize human speech. At the same time, neuropsychological and neuroimaging techniques have helped to identify the neural highlights that the human voice contains in its acoustic structures, a wealth of information on the speaker's identity and emotional state, which we perceive with remarkable ease and accuracy. However, little is known about its neural basis in studying voice-selective areas in the human auditory cortex; using fMRI, they observed strong evidence that the human brain contains regions that are sensitive to but strongly selective to the human voice.

## Speech Laterality and Hemispheric Transfer

This was already dealt with in the earlier chapter on Cerebral dominance.

Obrador (1964, cited by Smith A in 1966)[92] described the transfer of language to the 'non-dominant' hemisphere as a rule up to the age of 15 years, while a ten-year-old left-handed girl (Gardner WJ *et al*. 1955)[93] and a 14-year right-handed boy (Hillier WF 1954)[94] were reported living 53 and 27 months respectively with functional speech after left hemispherectomy. Smith A (1966)[92] described a 47-year-old, right-handed male who had preoperatively unequivocal evidence of left–hemisphere dominance 8 months after a left – left-hemispherectomy for glioma and had right hemiparesis but no receptive dysphasia. However, the speech was slow and deliberate; he would find words if given time. His speech continued to improve till 181 days post-hemispherectomy. Smith pointed out that "The findings in this case are strikingly

consistent with observations in the two reported similar cases by Robert Z (1935)[95] and Crockett and Estridge N (1951).[96] However, Boatman D *et al*. (1999)[97] observed that speech lateralisation is primarily completed by 5 years of age. The time course for acquiring receptive and expressive language functions in early development differs, with comprehension preceding production.

Wilson (1970),[20] who reported 50 cases submitted to hemispherectomy for infantile hemiplegia by Mc Kissock, observed that all but 2 youngest patients in this series had either acquired speech before operations or were in the process of doing so. Sixty per cent had the right, and 40 per cent had a left hemispherectomy. Eighty-four per cent retained unimpaired speech functions irrespective of whether the right or left hemisphere had been removed. Post-operative dysphasia or aphasia occurred in 6 (12%), all but one having left hemisphere removed. In half, the loss of speech was permanent (all these had gross mental recordation as well).

It is interesting to note that in hemispherectomy (even of the dominant left hemisphere) in patients of infantile hemiplegia, Rasmussen Syndrome, developmental anomalies did not increase the existing motor, sensory or speech deficits and, in some cases, even improved such deficits. The 'damaged' diseased hemisphere inflicts its adverse effects on the opposite normal side (Krynauw RW 1950,[98] Welch K and Penfield W 1950,[99] Rasmussen and Milner B 1977,[100] Searleman A 1977[101]).

Penfield W (1954)[102] justified the removal of the damaged brain with an aphorism-"No brain is better than a diseased brain" (Penfield W and Jasper H 1954,[103] also cited by de Almeida AN *et al*.).[104] Searleman A (1977),[101] in a review of right hemisphere linguistic capabilities, reiterated that following left hemispherectomy for infantile hemiplegia, the right hemisphere almost invariably assumes the speech and language functions that would typically have been reserved for the left. While some investigators (Basser 1962,[40] Carlson J *et al*. 1968,[105] Smith A and Burklund CW 1966,[106] Wilson 1970[20] believed this transfer to be complete, others argued that this transfer of linguistic skills is incomplete (Annett M 1973,[107] Gardner 1955,[96] McFie J 1961,[108] Gazzaniga MS and Sperry RW, 1967[109]) reported similar findings in patients following surgical disconnection of the two cerebral hemispheres.

Cutler HM and Goldstein R [110] show that social feedback makes a difference in the quantity and quality of young infants' utterances. They observed that "mother's responsiveness to their infant's vocalization may be an essential component contributing to infants' language learning. The unique way adults talk to babies, called motherese, may assist language learning. Following a series of studies, Kuhl PK *et al*. (2003)[23] concluded, "Considering all of the data, there is a very tight coupling between language and social cognition.

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# Correlates of Conventional Clinico-Pathological Syndromes

## A: Broca's Aphasia

### Historical Review

Broca, the acknowledged pioneer firmly establishing the localization of speech to a small area of the cerebral cortex, started a virtual revolution in the field of functional localization in the human brain. Hence, historical details that are much more significant than usual have been included in this section.

In April 1861, Pierre Paul Broca,[1,2] a surgeon at Biceter Hospital (actually a nursing home) near Paris, was advised to see a patient named Laborgne,[3] who was admitted there in 1840 with a speech disorder who had developed a gangrenous lesion involving the right leg. Before performing surgery. Broca (an anthropologist, not a neurologist) got interested in the patient's speech disorder.

Having little knowledge of speech disorders, Broca requested that his friend Arbutin to evaluate the patient's speech. The Latter recorded that the patient had lost articulated speech except to say "tan tan". Within a couple of days following surgery for the gangrenous limb, the patient expired on 17 April 1861. Broca performed an autopsy the next day and, after a preliminary study of his brain, preserved it in the Dupuytren Museum without slicing it. Soon after, Broca had another patient, Lelong. He had a speech disorder and could speak only five words: 'oui' (yes), non (no), tois (misspelled for trois; three), toujours (always) and Lelo (a mispronunciation of his own name, Dronkers NF *et al.* 2007).[4]

On March 21, 1861, Broca addressed the Societe Anthropologie on cerebral localization of speech (He was the founder and Secretary of the Society). Later that year, he published the detailed autopsy findings of both the patients (Leborgne and Lelong), in Bulletin de la Societe d' Anthropologie (Broca 1861, a, b) and Bulletin de la Societe d' Anatomique de Paris (1861 c & d),[3,4] (Lee DA 1981).[5] By the spring of 1863, Broca had 8 such cases of speech disorders. All exhibited lesions of the left hemisphere (Finger S 1988, 2004).[6,7] A series of papers on the subject were published by Broca in the following years (Broca 1863, 1865, cited by Schiller F in 1992).[8] It is not surprising that within a few years, the subject of cerebral localization of speech and language attracted the attention of many eminent physicians. Remarkably, almost identical clinical observation of the relationship between aphasia and right hemiplegia was not recognized generally until the last half of the 19th century. Hence, the credit for localization of speech to the left hemisphere has been typically attributed to Broca's presentations in 1861. However, this led to an academic debate about attributing the priority to Marc Dax's earlier contribution in 1836, twenty-five years before Broca's observation. This was due to the lack of its formal presentation or publication till its publication by his son Gustav Dax in 1865 (Those interested in this intriguing historical subject: Refer to Benton AL and Joynt RJ 1960,[9] Joynt RJ and Benton AL 1964,[10] Schiller F 1983[11]).

By the late nineteenth century, two different beliefs about the brain's function in language production had formed. The localization theory, supported by Wernicke C 1969,[12] Lichtheim L 1885,[13] Henschen S 1920-1922,[14,15] and others, maintained that specific mental functions were localized in particular brain areas. Several illustrious opponents of the localization theory (Head H 1963,[16] Brais B 1906,[17] Geschwind N 1964,[18] Marx OM 1967[19]) maintained that mental functions were a product of the entire brain working as a unit, and aphasia was attributed to dysfunction of the whole brain rather than a specific part of the brain (Lee DA 1981[5]).

It is remarkable that Broca not only performed the autopsy but deposited the brain of both Laborgne and Lelong in the Dupuytrens Museum without slicing it or even drawing a picture. These brains became the subjects of further studies by his colleague, Pierre Marie, a student of Charot (1906) and recently utilizing CT (Signoret *et al.*. 1993[20]) and MR imaging (Dronker NF *et al.*. 2007).[4]

These studies have not challenged the revolutionary contribution of Broca on the localization of functions in the human brain but have elaborated on the precise extent of brain damage since Broca had not even sliced these brains.

It is, however, surprising that with limited knowledge of the extent of brain damage, Broca confidently proposed that the patient's aphasia was due to the involvement of a restricted area of the pars opercularis and pars triangularis of the inferior frontal gyrus. Recent studies have established this is inaccurate. However, it does not reduce the historical significance of Broca's contribution.

This raises the question whether it the intuition of a genius?

Broca thought these patients understood what was said to them and that their language was intact; hence, he called the disorder "aphemia," referring to the absence of speech (Dronker *et al*. 2007).[4]

### Clinical Features

As mentioned above, Broca's first patient could utter only "tan tan" and the second patient's vocabulary was restricted to five words. Hence, it is not possible to ascertain the precise speech disorder the patients suffered from. Initially, Broca called the speech disorder of these patients "Aphemia" (Aphemic), believing that it was a loss of the "faculte due language articule" (the articulated language) and not "amnesia verbale" (verbal

amnesia). According to him, "The aphemic patient has a profoundly reduced vocabulary and maybe speechless except for some monosyllable, or swear words that do not seem to belong to any language". Broca reported that "the patient's ideas are intact, and he can understand what is said to him and recognize words and phrases which he cannot pronounce or even repeat. On the other hand, the amnesic patients no longer recognize the conventional association established between ideas and words" (Broca l861 a, b quoted by Lee DA 1981[5]). The term "Aphasia" was coined by Trousseau and Ferrier (1864, cited by de Oliveira-Souza R, 2016),[21] who called the third frontal gyrus "Broca's area" (Dronkers NF *et al.* 2007).[4]

***Footnote*:** The debate that finally resulted in the general acceptance of the Trousseau's (1864) term "Aphasia" to Broca's term "Aphaemia" was pervasive. Those interested in its history refer to Ryalls JH (1984).[22]

Clinically, over the years, Broca's aphasia has come to be recognized as a syndrome of disordered language, characterized by effortful speech production, impaired in melodic line and articulation, associated with semantic and phonemic paraphasias, telegraphic or at least shortened phrase length, reduced and abnormal grammatical form and a comprehension deficit most apparent when tested on material dependent upon understanding syntax (Alexander MP *et al*. 1990).[23] In routine clinical parlance, Broca's aphasia is classified as "Motor aphasia." It was generally believed that these patients had no associated comprehension deficit. However, it is now well established that there is also an element of comprehension deficit in Broca's aphasia.

Mohr JP (1978),[24] in a detailed study of Broca's aphasia, posited that recently, the term Broca's aphasia has come to refer to a complex clinical deficit featuring agrammatism, agraphia and even varying degrees of disturbance in comprehension, reflecting special grammatical language functions thought to be mediated by the Broca's motor speech area.

From a linguistic standpoint, it is now apparent that Broca's aphasia is a complex syndrome consisting of several different symptoms, including problems with fluency, articulation, word-finding, repetition, and producing and comprehending complex grammatical structure orally and in writing. A subset of these patients have a more severe disorder. These patients cannot make much of meaningful words or phrases, often restricted to explicit or some jargon (Dronkers NF *et al*. 2007).[4]

Despite several studies following Broca's epic-making description and those contradicting his contention about cerebral localization of speech-language, till recently, most clinicians believed that at least motor aphasia was due to a lesion restricted to anatomical area of the cortex called the Broca's area.

## Neuroanatomy

The autopsy report of Broca's first patient as reported by him, had a large lesion encompassing the left insula, frontal, central, and parietal operculum and even extending into the adjacent inferior parietal region posterior to the Sylvian fissure (Mohr JP *et al*. 1978).[24] Even his second patient had a large lesion in the left frontal lobe, including but not restricted to the third frontal gyrus. Yet, surprisingly, Broca attributed the aphasia patients to the involvement of the frontal operculum, even a smaller area, the gyrus triangularis. Ferrier D (1878)[25] called the third frontal gyrus the Broca's convolution. Interestingly, Broca's colleague Pierre Marie, a student of Charcot, re-examined the brains of Broca's patients and his interpretation of attributing speech defect to the third frontal gyrus in his paper. He concluded that "The third left frontal convolution has no unique role in the function of language." Even Ferrier did not fully agree with Broca's contention. Soon after, Wernicke (1874),[12,26] acknowledging Broca's contribution, described now well-recognized Wernicke's area primarily involved speech perception and being responsible for "sensory aphasia" (See chapter on Wernicke Aphasia).

Wernicke also recognized that a lesion involving a fiber connection between Broca's area and Wernicke's area results in a specific type of aphasia now clinically recognized as ("Conduction Aphasia").

Over the following decades, clinicopathological studies suggested that other brain regions also play a role in speech production, some of which are medial to the area of the brain initially described by Broca on the lateral surface of the frontal lobe. Results also indicated inconsistencies between the area initially identified by Broca and what came to be called Broca's area (Dronkers NF *et al.* 2007).[4]

Recent studies of speech function of healthy volunteers and patients with well-defined focal brain lesions have revealed that Broca's original description of the brain lesion responsible for aphasia, generally accepted as a fact, has several limitations.

CT scans of stroke patients revealed that infarction of the Broca's area produced neither Broca's aphasia nor any persisting aphasia but that Broca's aphasia emerges as a residuum of a large infarct, which initially produces global aphasia. It was pointed out that "Modern description of opercular syndromes contain little reference to aphasia (Bruyn and Gauthier 1990).[27]

Mohr JP *et al*. (1978)[24] convincingly demonstrated that lesions confined to the third frontal gyrus were not sufficient to produce Broca's aphasia and that this type of aphasia usually occurs with extensive lesions that initially produce global aphasia. This was confirmed by Kaplan E and Goodglass H (1981)[28] and Pedersen PM *et al*. (1995).[29] Nadeau SE (1988)[30] affirmed that "destruction of the third frontal convolution does not produce lasting linguistic impairment that is readily evident at the bedside. He adds, "We still do not know what the linguistic function was, if any, it does fulfill."

These observations support the CT scans of Broca's first patient (Signoret *et al*. 1993)[20] and MR imaging (Dronkers *et al*. 2007)[4] of the brains of Broca's two initial patients preserved in Dupuytrens Museum in Paris. The detailed anatomical description and accompanying figures in both these papers illustrate the extensive left hemispheric lesions in both these patients. Summarises of the findings of MR imaging of the brains of both these patients of Broca by Dronkers NF *et al*. (2007)[4] are reproduced below: Leborgne's brain (Broca's first patient) "demonstrates significant damage throughout the left hemisphere, both cortically and subcortically.

It was seen that sagittal and coronal slices through the brain revealed lesions in the left inferior frontal gyrus, deep inferior parietal lobe, and anterior superior temporal lobe. In addition, there is an extensive subcortical involvement, including the claustrum, putamen, globus pallidus, head of the caudate nucleus, and internal and external capsule. The insula is wholly destroyed. The entire length of the superior longitudinal fasciculus is also obliterated along with other fronto-parietal periventricular white matter. The medial sub-callosal fasciculus is also affected. The right hemisphere is unaffected".

MR imaging of Lelong's specimens (Broca's second patient) revealed, "The lesion involves the posterior part of the pars opercularis, while the anterior half of the structure and the entire pars triangularis are completely spared. In addition, small but distinct lesions are present in the superior longitudinal fasciculus close to the insula and lateral to the anterior horn of the left lateral ventricle. Though severely atrophied, the insula is not specifically lesioned in this case, nor are other deep structures, including the medial subcallosal fasciculus. There are also abnormalities in the white matter pathways in the left temporal lobe." (For a detailed description and MR images, refer to Dronkers NF *et al*. 2007).[4]

The above account established that in contrast to the original commonly quoted description of Broca's aphasia, the pathologic anatomy usually involves damage to several areas across a large portion of the left hemisphere, including but never limited to the frontal operculum. This is in keeping with the multi-dimension of the known language disturbance, which constitutes the syndrome of Broca's aphasia.

Mohr JP (1978)[24] had already shown that, interestingly, lesions that are confined to the generally referred to as "Broca's Area" produce only motor and praxis deficit, without specific language involvement, and that to create a full-blown syndrome of Broca's aphasia requires much more extensive lesions.

Alexander MP *et al*. (1990)[23] elaborated on Mohr's version and proposed that Broca's area aphasias subsumes 3 separate profiles that may overlap in any combination: Each profile has its pathologic anatomy. Analysis of the breakdown of various speech and language components in these cases suggests that the operculum, lower motor cortex, and subjacent subcortical and periventricular white matter contain critical parts of different language systems. These systems can be independently impaired. They add, "Several common language syndromes follow damage, including the left frontal operculum. These syndromes reflect the effects of the direction and extent of the lesion in the various language systems". According to them, the classical Broca's aphasia, as described above, follows damage to all three systems plus additional damage to the limbic frontal periventricular pathways of medial subcallosal fasciculus plus white matter pathways. They arrived at an exciting conclusion, "with a lesion so large and a syndrome so complex, it is impossible to dissect the contributions of the various functional systems."

These profiles were based on analysis of neuroimaging studies, which defined much more restricted clinical-anatomical relationship following lesions of the posterior inferior frontal region than those of classical Broca's aphasia (Henderson VW 1985,[31] Naeser MA *et al*. 1989,[32] Mori E *et al*. 1989[33]). One profile is focused on impaired speech and language initiation and occurs with lesions centered on the operculum. A second profile is concentrated in disturbed articulatory function; it follows lesions in the lower motor cortex immediately posterior to the operculum. A third profile includes some elements of classical Broca's aphasia, which follows lesions that contain both the operculum and the lower motor cortex and pathways deep to the Rolandic cortex. It is interesting to note that Alexander MP *et al*. (1990)[23] speak of *Broca's area aphasias* and not it being a single syndrome.

P. S: The reader is referred to Tandon PN. Paul Pierre Broca: Birth Bicentenary. Neurol India 2024 for further information.[2]

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## B: Wernicke's Aphasia

A little more than a decade after Broca's pathbreaking contribution of a well-defined cortical area to be responsible for motor aphasia (Broca 1861),[1-7] Wernicke (1874)[8-10] described what came to be known as "Sensory " aphasia due to a lesion of the left superior temporal gyrus".

In his monograph, Wernicke described the entire region of the first convolution, which encircles around the fossa of Sylvii, in conjunction with the insular cortex as a speech centre. A picture in his textbook on neurology of 1881, shows the area for language comprehension to be almost synonymous with the first temporal gyrus (Bogen JE and Bogen GM 1976[11]).

By 1917, Foix (quoted in Dax G )[12] affirmed that only cerebral territory whose lesion produces Wernicke's aphasia includes gyrus supramarginalis, angularis and the feet of the first two temporals (Bogen JE and Bogen GM 1976).[11] For more historical details, see the paper titled "Wernicke's region, where is it ?" The neuroanatomical basis of sensory aphasia was a central issue of many German-speaking neurologists of the late 19th and early 20th century. This included Lichtheim, Bonhoefer, Pick, Henschen, Goldstein and Kleist. French neurologists Charcot, Marie, Dejerine and others; English neurologists included Bastian, Mills, Bromwell, Head, Wilson and Nielson.

Based on accumulated information, Binder JR (2017)[13] summarized, "Like the other aphasias, Wernicke's aphasia is a syndrome complex composed of several distinct signs. The central characteristic is language comprehension manifested by incorrect or unexpected responses to spoken commands and other language stimuli. In the acute stage, this deficit may be so severe as to be seen too often, appearing to show no reaction to verbal input from others and no interest in comprehension. However, it is universally accepted that patients with this syndrome must demonstrate a comprehension disturbance for auditory verbal (most likely written) inputs, as Wernicke proposed.

Geschwind N (1970),[14] Goodglass H *et al*. (1976),[15] and Alexander A *et al*. (1999)[16] have described the comprehension problem as multimodal.

An essential feature of speech of patients with Wernicke's aphasia is the appearance of paraphasias in spoken and written output. This term refers to a range of output errors, including substitution, addition, duplication/omission and transportation of linguistic units. Paraphasia (semantic or phonemic) may affect letters within words, syllables within words or words within sentences. The speech is fluent and often excessive (logorrhoea). There may be neologistic substitution resulting in "Jargon aphasia". Naming and writing are abnormal, though handwriting is well-formed and legible.

Like Broca's aphasia and Broca's area, it soon became apparent that neither the clinical syndrome nor its neuroanatomical correlation was restricted, as Wernicke described. Soon after Wernicke, Charcot, along with his student Marie, included the supramarginal gyrus (SMG) along with the superior temporal gyrus (STG) as the site of lesion responsible for the Wernicke's aphasia (Marie 1888/1971)".

Starr MA (1889)[17] reviewed 50 cases of sensory aphasia published in the literature with autopsy correlation, 27 of whom had Wernicke's aphasia. It was already recognized that not all sensory aphasias confirm to Wernicke's original description. None of these patients had lesions restricted to the STG. Like the original Broca's patients, the lesion in these cases was wide in extent, involving the tempro-parietal and occipital convolutions (Binder 2003).

An authentic study by Geschwind N (1970) (**Figure 1**)[14]and several other investigators showed that the region most consistently involved in Wernicke's aphasia is the posterior left STG or STG and MTG (middle temporal gyrus). It is not surprising that a recent review concluded that the classic Wernicke's syndrome reflects damage to no less than nine distinct language processing modules (Margolin DI 1991).[18]

Functionally, there has also been debate about the fundamental functional defect in Wernicke's aphasia. While Bogen JE and Bogen GM (1976)[11] attributed Wernicke's aphasia to damage to the brain's "Comprehension Centre", Binder (2000)[19] argued that "comprehension is not a unitary process in the brain but rather a complex cascade of interacting events involving sensory processing, pattern recognition, mapping of sensory patterns to more abstract word recognition and retrieval of semantic and syntactic information".

Thus, Wernicke's aphasia is not simply an impairment of comprehension but includes key components in addition to comprehension disturbances, notably paraphasic and paragraphic outputs. Wernicke attributed this to a centre for "word-sound images centre" (*Wortklangbilder*) that is necessary for both word recognition and production (It may be noted that Broca'a aphasia is no longer considered a pure motor aphasia but has an element of difficulty in comprehension).

## Neuroanatomical Correlates

Wernicke's proposition of the significance of lesions of STG in producing sensory aphasia was already questioned by Henschen (1920-22) based on a review of 109 autopsied cases of the temporal lobe. Foix C 1928[20] and Mohr JP and Hier DB 1992[21] disputed the claim that lesions restricted to the posterior superior temporal gyrus caused Wernicke's aphasia. Benson DF *et al*. 1973,[22] Boller F 1973,[23] Damasio AR *et al*. (1980),[24] and Basso A *et al*. (1985)[25] have reported cases of isolated lesions of left STG with completely normal auditory and written language/speech comprehension. It is evident that, like Broca's aphasia, it is usually a large lesion involving the areas described above that results in Wernicke's aphasia.

It was generally agreed that patients with Wernicke's aphasia have lesions in the lateral temporal and parietal lobe, seldom involving the ventral and medial surface. It typically requires a cortex in and around the posterior Sylvian fissure (Mohr JP and Hier DB 1992).[21] This area is in the territory of the vascular supply of the lower division of the middle cerebral artery. Areas other than the STG, like the middle temporal gyrus, small portions of the inferior temporal gyrus were also reported to be involved. The parietal lobe's angular gyrus and various parts of the supramarginal gyrus were reported to be involved to varying extents. According to Binder J,[13,19,26] the lesion almost always affects the posterior third of the insula and may extend back to the anterior aspect of the lateral occipital lobe.

More recently, non-invasive neuroimaging studies (Damasio H and Orlando F 1981,[27] Damasio H 1989,[28] Naeser MA *et al*. 1982,[29] Selnes OA *et al*. 1984,[30] Caplan D *et al*. 1995,[31] Kertesz A and Munoz D 1998[32]) confirmed the involvement of these additional areas besides the superior temporal gyrus proposed by Wernicke.

Although the anatomical boundary of Wernicke's area has become too broad to be meaningful, most neurologists and neuropsychologists locate the core of Wernicke's area in the superior temporal cortex posterior to the plane of primary auditory cortex (Galaburda AM *et al*. 1978,[33] Bogen JE and Bogen GM 1976,[11] Wise RJ *et al*. 2001[34]).

Wise RJ *et al*. (2001) [34]analysed four PET studies to identify anatomically separate functional subsystems in the left superior temporal cortex posterior to the primary auditory cortex. From the results, they identified a posterior stream of auditory processing. They hypothesized that the posterior superior temporal cortex is specialized for processes involved in sound mimicry, including repetition, and the specific role of the posterior superior temporal sulcus is to represent phonetic sequences, whether heard or internally generated and rehearsed. These processes are central to acquiring long-term lexical memories of novel words. **(Figure 3&4)**

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## C: Transcortical Motor Aphasia (TCMA)

A well-defined clinical syndrome of speech disorder was first described by Lichtheim L (1885).[1] Goldstein K (1917)[2] published a detailed monograph on the subject "Die transcortical Aphasia", where he differentiated two forms of TCMA, one caused by partial damage of the motor speech centre, which results in "heightening of the threshold of motor speech performance". The other type shows " an impairment of the impulse to speak at all" (von Stocker TR and Bader L 1976).[3] The syndrome is characterized by a lack of or limited spontaneous speech, intact repetition, normal articulation, and good auditory comprehension (Freedman M *et al*. 1984).[4] There is no problem with naming, no disturbance of grammar, and no lexicon in whatever was spoken. Paraphasia (literal and verbal) and word-finding difficulties were reported in five out of seven patients studied by Freedman M *et al.* (1984).[4] Word list generation was very poor.

According to Kaplan E and Goodglass H (1981),[5] TCMA is marked by an absence of spontaneous speech with difficulty initiating speech and limited ability to name or make brief responses. Auditory and reading comprehension and reading aloud are relatively intact. Repetition is remarkable in that it is prompt, well articulated, grammatically intact and free of difficulty in initiation, which marks all other speech. The syndrome is classified among "Non-fluent" aphasia. Besides the clinical features described above, another sign is the perseveration of words (Alexander MP and Schmitt MA 1980).[6] It may be pointed out that on a casual bedside examination, the condition may be mistaken for Broca's aphasia.

### Neuroanatomical Correlation

As initially indicated by Lichtheim L (1855)[1] the mechanism for TCMA production is in the left frontal lesions, some entirely outside the perisylvian speech area. Critchley M (1930)[7] described this syndrome due to the lesion of the anterior cerebral artery. This has been confirmed by Rubens AB (1975),[8] Alexander MP and Schmitt MA (1980).[6]

Schwab U (1927)[9] reported that 14 out of 21 patients who had speech disorder after surgical excision of epileptogenic foci from the left "frontal adversive field" had TCMA. Numerous reports characterise the alterations in verbal output secondary to manipulation of the supplementary motor area (SMA), which lies within the distribution of the anterior cerebral artery. Spontaneous seizures, electrical stimulation, or surgical manipulation of SMA produces repeated vocalization, speech arrest and bilateral mouth movement. It produces transient speech disturbances like a failure of initiation. Permanent speech disorders include difficulty in the initiation of speech [Penfield W and Welch K (1951),[10] Chusid JG *et al.* (1954),[11] Penfield W and Roberts L (1959),[12] von Stockert TR (1974),[13] Bensen DF (1981),[14] Alexander MP and Schmitt MA(1980),[6] Freedman SW (1985),[15] Freedman L *et al.* (1991)[16]].

However, Kaplan and Goodglass (1981)[5] analysing many cases fulfilling the standard definition of TCMA, found more than one lesion site for this syndrome. Some patients had changes in the frontal operculum, including the anterior portion of the Broca's area. Some damaged the dorsal mid-frontal region, which often projected into the white matter. Some had damage to the deep white matter, and some had lesions of the medial frontal cortex, including the supplementary motor area.

All seven patients with classical TCMA studied by Freedman L *et al*. (1984)[16] with CT demonstrated small lesions in the left frontal lobe in the white matter anterolateral to the left frontal horn. This lesion was usually most significant at the level of the lateral ventricle superior to the pars triangularis and pars opercularis of Broca's area. It never involved the site of classical Broca's area. Three patients had lesions in the anterior cerebral artery distribution, and one included supplementary motor area.

It is postulated that the syndrome results from the disruption of subcortical white matter connecting the supplementary motor area from Broca's area. The lesion is usually anterior to the Broca's area.

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## D: Transcortical Sensory Aphasia (TCSA)

Lichtheim, in 1885 (quoted by Lazar[1]) is credited with coining the term "Transcranial Sensory Aphasia." While Wernicke had not used this term in his original article on sensory aphasia, Lichtheim gave him credit for this concept.[2]

However, the condition was disputed by many contemporary neurologists, e.g. Henschen (1919),[3] Dejerine (1892)[4] and also quoted by Schmahmann and Pandya)[4,5] and several others (For a more detailed account of the history of this syndrome, see Alexander *et al*. 1990).[6] Clinical neurologists now recognise TCSA as a classic aphasia (Geschwind *et al*. 1970,[7] Heilman *et al*. 1981,[8] Kertesz *et al*. 1982,[9] Alexander *et al*. 1989[10]).

The syndrome is characterized by fluent aphasia with impaired auditory comprehension but normal repetition (which distinguishes it from Wernicke's aphasia) and usually severe alexia and agraphia. Its paraphasic substitutions are prominent. They are generally semantic (Heilman *et al*. 1981[8]). Often, the spontaneous speech of these patients is characterized by semantic jargon, but this feature is not obligatory for diagnosis of this syndrome (Kertesz *et al*. 1982, [9]Graff-Radford *et al*. 1985[11]). **(Figure 5).**

The syndrome has been correlated to various posterior brain regions (quite away from the classical sites of Broca's and Wernicke's aphasia). These include the posterior second and third temporal gyri, the temporoparietal–occipital junction, the inferior temporo-occipital region and the anterolateral thalamus.

Kertesz *et al*. (1982)[9] provided a well-illustrated CT/study of 31 patients. They concluded that the anatomical location of the lesion suggests that the syndrome is most often seen with infarction in the posterior cerebral artery territory or in watershed area lesions that involve the territory between the posterior cerebral and middle cerebral arteries. They described the occurrence of visual agnosia in many patients with TCSA. They observed that a unilateral dominant hemisphere lesion with callosal involvement can produce visual agnosia, although bilateral lesions are usually associated.

Heilman *et al*. (1981)[8] reported another group of such patients with a relatively intact ability to name. Their patients had posterior-superior parietal lesions.

Influenced by the original description by Lichtheim (1885), TCSA came to be recognized as a result of the disruption of the connection between Wernicke's area and the centre of concepts (Begriff S field).

Geschwind *et al*. (1968)[12] and Brown (1975)[13] mentioned "Isolation syndromes," implying the isolation of Wernicke's area is responsible for this syndrome.

Alexander *et al*. 1989[10] hypothesized that analysis of CT lesion sites in many patients with infarctions in the territory of the left PCA may distinguish the precise pathologic anatomy responsible for TCSA. He also proposed that these cases provide evidence for a specific functional brain system with one particular distributed anatomy. Damage to this functional system produces disturbances in semantic functions. The manifestations are disturbed semantic-lexical comprehension, recall and recognition. The distributed anatomy includes the posterior temporal association cortex, Brodman's area 37 and perhaps (at least in some individuals) an even larger crescent of the posterior association cortex, portions of Brodman's area 39 and 19, at the temporoparietal-occipital junction. The anatomy also includes some elements in the anterior or lateral thalamus, although exactly which one is still speculative. Finally, there are the white matter regions in which the afferent projections of these thalamic and cortical centres converge. The primary focus of these converging pathways seems to be in the posterior periventricular white matter adjacent to the proximal temporal horn, the immediately adjacent posterior portion of the temporal isthmus and along the posterolateral margin of the thalamus.

Coslett *et al*. (1987)[14] provided evidence of several subjects of TCSA.

From the above review, it is evident that while the clinical symptomatology is generally well-defined, its neuroanatomical correlation remains generally hypothetical and, at best, somewhat distributed (Alexander *et al.* 1989), **(Figure 6)**.[10]

Note:-Reference Figure 2, page 476 of the paper by Kertesz *et al*. 1982.

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## E: Subcortical Aphasia

"White matter pathways are critical structures in language disorders."

(Alexander *et al*. 1987).[1]

The occurrence of aphasia after subcortical lesions is well documented and around the same time, Marie (1926-28) recognized the potential role of subcortical lesions in producing aphasia. However, recently, especially after the advent of non-invasive neuro-imaging, many studies have highlighted the role of subcortical lesions resulting in speech disorders (Brunner *et al.* 1982,[2] Naeser *et al.* 1982,[3] Cappa *et al.* 1983,[4] Damasio *et al.* 1982,[5] Alexander *et al.* 1987[1]). According to Naeser *et al*. (1982),[3] the term Sub-Cortical Aphasia is used to refer to aphasia that results from lesion sites that may include internal capsule, putamen and subcortical periventricular white matter. In their series, approximately 10% of aphasia syndromes had subcortical lesions in the capsule putamen (C/P) areas. They observed three subcortical aphasia depending upon three lesion sites within this area. Patients within lesion sites with anterior superior white matter extension had good grammatical comprehension but slow dysarthric speech and lasting right hemiplegia. Patients with C/P lesion sites with posterior white matter lesion extension across the auditory radiation in the temporal area thus had poor comprehensive, fluent Wernicke's type speech and lasting right Hemiplegia. Patients with C/P lesion sites with both anterior superior and posterior extension were globally aphasic and with lasting right hemiplegia. Cappa *et al*. (1983)[4] confirmed these anterior-posterior extension syndrome regions were the critical ones for all components of aphasia. A review of 61 subcortical cases in the neurological literature for which CT and aphasia data were available supported these conclusions. The diversity of these lesions and their clinical features have been summarized in Table 1, modified from Figure 16 of Alexander *et al*. 1987.[1]

However, it was after the routine use of non-invasive neuroimaging for the investigation of patients with stroke, around the 1970s, that the frequency of such lesion-producing aphasia became obvious.

Alexander *et al*., (1987)[1] in a very detailed paper, reported CT findings in 19 cases with subcortical infarction or haemorrhage with several components of aphasia syndrome, especially sentence length and grammatical form (together comprising fluency), ease of speech initiation, articulation, voice volume and auditory comprehension. Each component had a specific lesion site correlation, and the lesion components of aphasia in various deep periventric white matter regions were critical.

Naeser MA *et al*. (1982)[3] and Alexander MP *et al*. (1987)[1] described aphasia due to capsulostriate area, while several authors (Bogousslavsky J *et al*. 1988,[6] Bruyn RP 1989,[7] Gold M *et al*. 1997[8]) analysed aphasia syndromes associated with lesions of the thalamus (Ojemann GA 1983,[9] Penfield W and Roberts L 1959,[10] Archer CR *et al*. 1981,[11] Lazzarino LG *et al*. 1991[12]).

Another detailed study by Naeser MA *et al*. (1982)[3] reported "no single neuroanatomical area that contained an extensive lesion, which could be used to discriminate the most serious cases (of aphasia) from the least severe. The two groups were separate, however, based on a CT scan where the extent of the lesion in two subcortical white matter areas was combined (1) the most medial and rostral part of the subcallosal fasciculus plus (2) the periventricular white matter near the body of the lateral ventricle deep to the lower motor/sensory cortex area for the mouth (Probably the same area highlighted by Alexander MP *et al*., 1987[1]). Extensive lesions in only one of the two white matter pathways areas, alone was not sufficient to

produce long-lasting severe limitation in spontaneous speech." Mega and Alexander MP (1994)[13] reaffirmed the occurrence of aphasia after subcortical lesions. Acknowledging the existence of disagreement about the precise characteristics of "subcortical aphasia" based on detailed study of 14 patients with aphasia after subcortical lesions they observed, "The clinical profiles of the patients were quite similar varying in severity in rough proportion to the lesion size and varying in quality in proportion to anterior paraventricular extent."

They quoted the reports by Naeser MA *et al*. (1982),[3] Alexander MP *et al*. (1987)[1] on aphasia due to lesions affecting the capsulo-striate region, and aphasia due to lesions of the paraventricular white matter earlier reported by Alexander MP *et al*. (1987).[1] They provided a detailed clinical manifestation following the subcortical infarcts characterized by impairment of generative language ability and lexical selection anomia of varying severity. The generative-language problem produces an abnormality of verbal fluency tasks, deficient sentence generation with increased latencies, perseveration and occasional bizarre content despite a generally grammatical and fluent conversation. Confrontational naming and conversational word retrieval are reduced. Phonemic paraphasia is uncommon, and word repetition, oral reading, and comprehension are generally intact. They suggested that "damage to a frontal-caudate functional system underlies a 'Core' aphasia profile in these patients."

A decade later, Nadeau SE and Crosson B (1997),[14] in a rather lengthy paper, strongly disagreed with Alexander MP *et al*. (1987)[1] and claimed, "To date, there is no compelling evidence that non-thalamic subcortical structure is directly involved in language functions exists." Not denying the occurrence of aphasic disorders in subcortical lesions, they claimed that non-thalamic subcortical aphasia due to ischaemic lesions mainly results from some combination of sustained cortical hypoperfusion. In support of their hypothesis, they quoted the work of Olsen CO *et al*. (1983),[15] Larson D *et al*. (1983),[16] Weller M JF *et al*. (1993),[17] Démonet *et al*. (1992),[18] Caplan D *et al*. (2007).[19]

However, several other reports on subcortical aphasia do not support the hypothesis proposed by Nadeau and Crosson (Basso A *et al*. 1985,[20] Cappa SF *et al*. 1983,[4] Démonet JF *et al*. 1991[21]).

It may be pointed out that both Alexander MP *et al.* (1987)[1] and Nadeau SE and Crosson B (1997)[14] agree the lesions of the basal ganglia (Caudate and Putamen) do not produce aphasia. Crosson B (1985)[22] proposed that the basal ganglia regulate the release of cortically generated language segments but do not participate in the actual formulation of these segments. However, the thalamus plays a significant role (See under thalamic aphasia, Chapter 6g).

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## F: Conduction Aphasia

Originally described by Wernicke C (1874)[1,2] as a symptomatically distinct form of aphasia, who termed it "Leitungsaphasia" or "Conduction Aphasia," Lichtheim L (1885)[3] preferred to term it "Commissural Aphasia" to make a distinction between aphasia due to damage to the centres (Broca and Wernicke) versus interruption of the commissural pathway between the centres (Anderson JM *et al*. 1999,[4] Benson DF *et al.* (1973)[5] supported the hypothesis that the syndrome is the result of disconnection between the Wernicke's and Broca's area as initially proposed by Wernicke. However, Anderson JM *et al*. (1999),[4] based on their study of electrical stimulation during epilepsy surgery, have suggested that alternatively, conduction aphasia might be induced by cortical dysfunction alone.

The posterior portion of the superior temporal gyrus is connected to the anterior perisylvian area (Petrides M and Pandya WA 1988)[6] by a white matter bundle called the arcuate fasciculus. Geschwind N (1965),[7] in his anatomical studies of language dysfunction, proposed that a lesion of the arcuate fasciculus was responsible for conduction aphasia.

Conduction aphasia is characterized by fluent but hesitant (dysprosodic) speech with paraphasias and near-normal spoken language comprehension (unlike Wernicke's aphasia). There is gross difficulty in repetition, confrontation, naming and reading aloud. Writing is notably disturbed by poor spelling, reversal, and substitution of letters.

Note: References to Figure 2 of Anderson *et al*. 1999[4]

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## G: Thalamic Aphasia

The role of the human thalamus in brain mechanisms underlying speech has been controversial. Some pioneer neurologists like Head H (1963),[1] Nielsen JM (1946)[2] and, Geschwind N [3] as late as 1970, did not find any speech function in the thalamus. However, aphasic disorders in patients with left thalamic tumours, haemorrhage, infarction, following thalamotomy and electrical stimulation have been well documented (Smythe and Stern 1938; quoted by Mohr JP *et al*.,[4] Cheek and Taveras JM 1966,[5] Reynolds MW *et al*. 1979,[6] Bell DS 1968,[7] Ojemann GA *et al*. 1968,[8] Van Buren JM and Borke RC 1969[9]). After the introduction of non-invasive brain imaging, it became possible to detect and delineate well-circumscribed ischemic/haemorrhagic lesions in the thalamus and study their neurolinguistic correlates (Archer *et al*. 1981,[10] Bogousslavsky *et al*. 1988,[11] Graff-Radford *et al*. 1985,[12] Cohen *et al*. 1980,[13] Gorelick *et al*. 1984[14]).

Archer CR *et al*. (1981)[10] reported a single case of aphasia due to a discrete CT-proven thalamic infarct. Detailed psychometric data of the patient's speech and memory disorders obtained during acute and chronic stages were correlated with the evolution of the lesion. They observed that discrete focal lesions in the dominant thalamus usually produce only temporary speech impairment. The most striking residual deficit (in the chronic stage) was the impairment of verbal recall. Mild anomia was detected 6 months after the ictus.

A detailed study of 40 patients with CT-proven thalamic infarct by Bogousslavsky J *et al*. (1988)[11] delineated four arterial thalamic territories (inferolateral, tuberothalamic, posterior choroidal, paramedian) that corresponded clinically to four different syndromes. Although "the study was not specially directed to speech disorders, it reported the following:

Inferolateral infarcts: Neuropsychologic evaluation revealed only one patient among 18 who had reduced verbal output with semantic paraphasia and mild impairment of comprehension.

Tuberothalamic infarcts: Among 23 patients in this group, neuropsychological dysfunction was the primary disturbance, with either dysphasia (in the four patients with a left-sided infarct) or hemineglect with impaired visuospatial processing in the patient with a right-sided infarct). Dysphasia was characterised by hypophonia, reduced output, verbal paraphasias, moderate comprehension impairment, and preserved repetition. Posterior choroidal infarcts: There were only 3 patients in this group, and only one patient had mild dysphasia. Paramedian infarct was observed in 9 patients unilaterally and 5 patients bilaterally. Impairment of consciousness was usually followed by neuropsychological disturbances: in unilateral infarcts, these disturbances found only in 2 patients were similar to those observed in the tubero-thalamic infarcts.

Electrical stimulation of the thalamus by Penfield and Roberts (1959),[15] Ojemann GA *et al*. (1968),[8] and Van Buren JM and Borke JM (1969),[9] demonstrated disturbance of speech. A summary of the results emanating from these studies, as provided by Ojemann GA and Ward AA (1971)[16] is quoted below:

"Thus, the pattern of speech representation in the left lateral thalamus that emerges from the present study (Ojemann

GA and Ward AA 1971)[16] complemented by that of Ojemann GA *et al*. (1968)[8] includes pulvinar and *en passage* fibres related to the centrum medium and dorsal medial nuclei. This is strikingly similar to the mode for thalamic speech representation proposed by Penfield W and Roberts L (1959)[15] who suggested that the centrum median and dorsal medial nuclei conveying were related to the frontal (Broca) cortical speech area, and the pulvinar to the parietal-temporal (Wernicke) cortical speech area."

Penfield W and Roberts L (1959)[15] attributed an integrative role for the thalamus for speech between the frontal (Broca) and parietal-temporal (Wernicke's) speech areas.

Based on their stimulation studies, Ojemann GA and Ward AA (1971)[16] concluded, "Within the left ventrolateral thalamus, anomia and perseveration were evoked from the medial central portion of that nucleus but not from areas superior-inferior, medial or lateral to the electrodes." Along with an earlier study, (Ojemann GA *et al*. 1968)[8] concluded that these two studies together suggest that within the left lateral thalamus, the anatomical substrate for speech includes pulvinar and *en passage* fibres related to the centrum median and dorsal medial thalamic nuclei.

## Evidence from Thalamic Surgery for Treatment of Dyskinesias

The general experience of several neurosurgeons performing ventrolateral thalamotomy for Parkinson's disease was that for a period after surgery, the patient may have slight difficulty in expressing words. At the same time, the understanding of spoken or written speech was never impaired" (Selby G 1967[17]). However, there were reports by Bell DS (1968),[7] Samra K *et al*. (1969[18]), and others which indicated more frequent disturbances of speech following a lesion in the thalamus of the dominant hemisphere than on the other side. However, most reports found the generally evanescent nature of these symptoms (Cooper IS 1959,[19] Gillingham FJ *et al*. 1960,[20] Bell DS 1968,[7] Samra K *et al*. 1969[18]).

## Evidence from Lesions of the Thalamus

The evidence from pathological lesions in the thalamus, e.g. ischemic infarcts, haemorrhage, and degenerative disorders, also fail to provide unequivocal evidence of speech defect. Thus, in a series of 7 cases of autopsy proved thalamic haemorrhage and 13 cases with clinical observations alone, Fisher CM (1959)[21] reported some instances of dysphasia. The dysphasia was usually characterized as moderate in severity, with calculations relatively well preserved. Speech examinations in these patients would not have been carried out using psychological or neurolinguistic studies. However, cases of autopsy proved gross or total destruction of the left thalamus due to haemorrhage, which presented no speech defect before death, have been documented.

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## H: Acalculia

*Number rules the Universe: Pythagoras*

### Historical Review

Henschen (1920, cited in Henschen S, 1924),[1] based on his study of approximately 110 abstracts and sources on the subject, concluded that acalculia included disturbances in number recognition and arithmetical operations (Cohn R 1961).[2] Henschen also published detailed accounts on the subject in 1919[3] and 1924.[1] Before Henschen's monumental report, Lewandowsky M and Stadelmann E (1908)[4] reported on the first published cases of acquired calculation disorder. There were also few other contributions as well, e.g., by Sitting (1917, quoted by Kahn HJ and Whitaker HA, 1991)[5] and Peritz (1918, quoted by Kahn HJ and Whitaker HA, 1991[5]). A detailed historical review on the subject has recently been published by Kahn HJ and Whitaker HA (1991).[5] Table 1 (Chapter 4a) summarises some earlier reports based on this and other significant publications. (The present author did not have access to the publications by those in Table 1; however, their source of information is included in the list of references reviewed by the author).

### Classification

Clinically, it is well recognized that acalculia could not exist as an entirely independent entity manifest only as some fractionation of the syndrome (Warrington EK 1982)[6] or, more often, coupled with other symptomatology. While authors like Lewandowski MS (1908),[7] Singer and Low AA (1933),[8] Cohn R (1961),[2] and Benson DF and Denckla MB (1969)[9] claimed that no case of "pure" acalculia is recorded, There are well-studied reports of acalculia as a well-defined entity (Benson DF and Weir WF 1972,[10] Gitelman DR 2003).[11]

Quoting a detailed account of acalculia by Hecaen H *et al*. (1962),[12] Benson DF and Weir WF (1972)[10] summarized three varieties of acalculia:

1. Aphasic acalculia is when several languages are disturbed
2. Visuospatial acalculia is when alignment and place-holding values are abnormal
3. Anarithmetia, is a disturbance of the essential ability to perform computation. In a detailed paper, the authors provide examples of each category.

### Neuroanatomical Localization

One of the most debatable aspects of acalculia is its neuroanatomical localization in the human brain. On the one hand, Henschen S (1919)[3] concluded that "a distinct and independent cortical network existed for arithmetic functioning and therefore acalculia could exist as an entirely independent finding of brain damage or could be coupled with other symptomatology. However, according to Cohn R (1961),[2] Henschen S (1919 and 1924)[1,3] concluded that "though acalculia in *(this)* broad sense was observed as a clinical manifestation of lesions located in widely disparate regions of the brain, the common anatomical involvement was the caudal portion of the left hemisphere.

On the other hand, Kahn HJ and Whitaker HA (1991),[5] after a detailed account of Henschen's reports (1919,1924),[1,3] concluded that Henschen "had thought the angular gyrus to be always implicated in calculation". A host of others, including some more recent ones like Cohn R (1961),[2] Benson DF and Denckla MB (1969),[9] claimed that "No case of "Pure" acalculia is recorded and even case reports in which computation defect is the major or out-standing feature are uncommon. Kahn HJ and Whitaker HA (1991),[5] yet again, in their detailed historical review of cerebral localization of acalculia, concluded that "A summary of cases of acalculia since Henschen's work, leads to the conclusion that regardless of the functional modular nature of calculation ability, there is neither a localized region nor a specific hemisphere uniquely underlying the disorder."

In between these two extreme views, some of the well-quoted publications on acalculia from the early contemporaries of Henschen S (1924)[1] to Luria AR (1966)[13] reflect the diverse opinions on the cerebral localization of lesions resulting in acalculia [See Table 1, Chapter 4a]. Thus, Lewandowski and Stadelmann (1908; cross referenced from Ardila A *et al.*, 2002)[14] reported a left occipital hematoma to be responsible for acalculia, while Berger H (1926)[15] mentioned it to be due to (i) a glioma of the left hemisphere reaching into posterior temporal and occipital lobes (ii) a glioma of the left paracentral lobule invading funicular gyrus, corpus callosum and left occipital lobe; and (iii) a left thalamic glioma, to be the pathologies found in three of his patients studied in detail.

Head H (1926),[16] in his book on "Aphasia and Kindred Disorders," made only a brief reference to calculation. He considered each variety of aphasia associated with a distinct form of arithmetic disorder. Goldstein K (1948),[17] in his book, "Language and Language Disturbances, proposed that acalculia typically followed lesions in the "parieto-occipital region and occasionally the frontal lobe." However, he stated that most published cases had lesions in the occipital lobe. Hecaen H *et al*. (1961)[18] attributed that most cases of acalculia exhibited bilateral lesions primarily in the parieto-occipital region. They argued that in all instances, parietal lobes constituted the central zone for calculation processes. They also proposed that an arithmetical dysphasia would only be found in cases of different cortical damage. Cohn R (1961)[2] reported that acalculia was a clinical manifestation of lesions located in widely disparate brain regions, and the common anatomical involvement was the caudal portion of the left cerebral hemisphere. Lesions disturbing the physiology of the central visual apparatus profoundly alter the processes of arithmetical order. Luria AR (1966)[13] similarly proposed that the parieto-occipital region and its role in linguistic operations were equally critical for arithmetic.

Most of these studies based on autopsy dealt with extensive lesions. Unlike Broca and Wernicke, they did not attempt to ascribe acalculia to a tiny part of the pathology. Of course, these studies were carried out before the modern non-invasive imaging studies mentioned in other chapters. However, unlike many other cognitive functions, including different types of aphasia, the introduction of recent neurodiagnostic techniques has failed to provide a definite answer to the anatomical localization of various aspects of numerical cognition. This

is despite many such studies, including detailed accounts by Kahn HJ and Whitaker HA (1991),[5] and Gitelman DR (2003).[11]

Corbett AJ *et al*. (1986)[19] described a single case of acalculia following dominant hemisphere subcortical infarct involving the head of the left caudate nucleus, the anterior superior putamen, and the anterior limb of the internal capsule extending superiorly into the periventricular white matter.

Whitaker HH and Ivers R (1985)[20] and Hittmair-Delazer M *et al*. (1994)[21] made a more or less similar observation. Interestingly, Ojemann GA (1974)[22] reported interference with mental arithmetic during human thalamic stimulation, implying some role for the thalamus in numerical cognition.

Kahn HJ and Whitaker HA (1991),[5] in their extensive historical review of acalculia localization, commented. "The site of lesions responsible for acalculia has been ascribed to many regions in the brain." Although calculation deficits occur more often with left hemisphere lesions, they can also be seen with right hemisphere injury. Several other authors reported acalculia from either hemisphere.

Dehaene and colleagues in a series of fMRI studies, provided a detailed account of localization of calculation and mathematical function in regular healthy volunteers and patients with acalculia (Dehaene S and Cohen L 2007,[23] Dehaene 2005 S [24]). Following a survey of sources of mathematical thinking, behavioral, and brain-imaging, Dehaene, *et al*. S (1999)[25] concluded, "Our results provide grounds for reconciling the divergent introspection by showing that even within the small domain of arithmetic multiple mental representations are used for different tasks. Exact arithmetic emphasizes language-specific representations and relies on the left inferior frontal circuit also used for generating an association between words." Several other domains of mathematics, like symbolic arithmetic calculus, etc., may use different neural systems.

Table 2 of Chapter 4a summarizes the findings of the recent studies using recent neuro-imaging techniques.

Some more recent studies have only added to the confusion rather than arriving at a consensus. Thus, using PET studies, Zago *et al*. (2001)[26] observed the cerebellum's role in speech processes related to calculation. They found cerebellar activation to be seen when relatively complex or novel computations were carried out compared with simple numerical perception tasks. In addition, Zago L *et al*. (2001)[26] also noted that the left precentral gyrus, intraparietal sulcus, bilateral cerebellar cortex, and the right superior occipital cortex were activated in several instances. They referred to the previous studies by Dehaene *et al*. (1999)[25] and Dehaene S *et al*. (1999),[25] which reported similar findings. In a PET study, Menon DK *et al*. (2000)[27] saw bilateral mid-cerebellar activation when the subjects performed the most challenging computation tasks. Pesenti M *et al*. (2001)[28] reported that the right prefrontal and medial temporal cortex sustained mental calculation in a prodigy.

In a very detailed essay, summarising a large number of reports well illustrated by figures showing cortical and sub-cortical activation for a variety of numerical-functions Gitelman DR (2003)[11] confirms the diversity of anatomical sites in the human brain (in both hemispheres). His Figures 7.5 to 7.8 are very informative with their detailed text. To quote two of his significant observations, he indicated that figures 7.6 and 7.7 show the set of regions showing the most frequent activation across studies, including the bilateral dorsal lateral pre-frontal cortex, the premotor cortex (precentral gyrus, and sulcus), the supplementary motor area, the inferior parietal lobule, the interparietal sulcus, and the posterior occipital cortex-fusiform gyrus. Compared with the data from studies of exact calculations [Figures 7.6 and 7.7], approximation and magnitude operations [Figures 7.8 and 7.9] show relatively more parietal and occipital and less frontal activity.

To add to the confusion, Grafman J *et al*. (1982)[29] described a patient with near destruction of the left cerebral hemisphere, leaving only the occipital and parasagittal cortex as a result of a gunshot wound. Despite an inability to perform multi-digit calculations, he could compare multidigit numerals with excellent accuracy, suggesting that intact right hemisphere mechanisms were sufficient for performing this comparison task.

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## I: Primary Progressive Aphasia

Non-fluent/agrammatic primary progressive aphasia (PPA), also known as progressive non-fluent aphasia (PNFA), is a progressive neurodegenerative condition most prominently associated with slowed effortful speech that is produced at about one-third the rate of healthy adults.

The current recommendation for identifying PPA emphasizes three clinical features: Effortful speech, a grammar disorder and apraxia of speech (Grossman M *et al*. 1996,[1] Ash S *et al*. S2006[2]). Speech is produced at an average rate of about 45 words per minute. The variety of grammatical forms in sentences is impoverished.

Gorno-Tempini ML *et al*. (2004)[3] performed a comprehensive cognitive, neuroimaging and genetic study on 31 patients with PPA, a decline in language functions that remains isolated for at least 2 years. Detailed speech and language evaluation was used to identify three different clinical variants: non-fluent progressive aphasia (NFPA, $n$ = 11), semantic dementia (SD, $n$ = 10), and a third variant termed logopenic progressive aphasia (LPA, $n$ = 10).

Isolated speech and language difficulties are often the first symptoms of focal forms of neurodegenerative disease, particularly from front-temporal lobar degeneration (FTLD) (Neary D *et al*. 1998)[4] and cortico-basal degeneration (CBD) Kertesz A *et al*. 2000).[5]

In Alzheimer's disease, when speech and language deficits remain the only complaint for at least two years, the term PPA has been applied (Mesulam MM 1982,[6] Greene JD *et al*. 1996,[7] Croot K *et al*., 2000[8]).

Gorno-Tempini ML (2004)[3] and Grossman EJ (2013)[9] observed that taking into consideration the three variants of PPA (described above), the focal pathology is found in the left inferior frontal and anterior insular regions in NFPA, bilateral temporal lobes in SD and left temporoparietal in LPA.

A relatively uncommon speech and language disorder, usually a feature of a chronic neurodegenerative disorder, may precede the latter for a couple of years, resulting in a complex clinical diagnosis. Like several other aphasia syndromes, the pathological site of the syndrome is also distributed to several different brain regions.

The disruption of large-scale neural networks in non-fluent/agrammatic variants of primary progressive aphasia is associated with frontotemporal degeneration (Grossman EJ *et al*., 2013).[9]

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## J: Word Deafness

### Introduction

Although the clinical syndrome, "Word Deafness," was briefly described by Lichtheim L (1885),[1] over the years, there has been confusion regarding the clinical entity. Two relatively recent publications, Goldstein JL (1973)[2] and Buchman AS *et al*. (l986),[3] provide a wealth of historical and clinico-pathological information on this condition. The Table 5 of the paper by Buchman AS *et al*. (1986)[3] includes a list of 34 previous publications on Word Deafness, and It is a valuable source of information on the subject. Notwithstanding other terms like auditory agnosia, cortical deafness, and "Pure word deafness," it is generally recommended that the term "Pure word deafness" be retained for this syndrome.

Thus, according to Lichtheim L (1885),[1] "word deafness" is diagnosed when the ability to understand and repeat speech and write from dictation is lost. In pure or isolated word deafness, reading, copying written material, spontaneous writing and speech remain largely unaffected (Goldstein K 1948[4]). Defective repetition differentiates this condition from transcortical sensory aphasia, while preservation of reading, writing and spontaneous speech differentiates it from Wernicke's aphasia (Buchman *et al*. 1986).[3]

It may be pointed out that while Lichtheim L [1] has been given full credit for defining this syndrome. Kussumaul has been credited to have recognized auditory agnosia in 1877 (cited by Shaw E, 1893).[5] Kussumaul thought the word deafness to be the significant result of destruction of the first left temporal gyrus. At the same time, he described word blindness as due to lesions of the left angular and supramarginal gyrus (Goldstein K 1974).[4]

It is not surprising that word-deafness has often been considered a part of Wernicke's aphasia.

Marie (1906), who earlier questioned Broca's description of the third frontal gyrus as responsible for motor aphasia, called the syndrome of word-deafness a myth (Quoted by Goldstein K 1974).[4]

The recent advances in audiology, neurolinguistics, and non-invasive neuroimaging have, on the one hand, added additional features to the syndrome, confusing its earlier defined clinical features. These also advanced our knowledge of its neuro-anatomical correlates. Thus, patients whose audiometric findings cannot account for their auditory comprehension deficits may be said to have an agnosia for sound. When this deficit is limited to linguistic sound, it is pure word deafness or auditory verbal agnosia (Buchman AS *et al*. 1986).[3]

It may be mentioned that this syndrome, usually caused by ischemic/haemorrhagic lesions, is common in age groups prone to varying degrees of deafness. Hence, a comprehensive audiometric examination is essential before arriving at the diagnosis of word deafness.

According to Buchman AS *et al*. (1986),[3] "a review of the literature of cortical auditory disorders establishes that no patient previously reported with word deafness, who have had formal testing of linguistic and non-linguistic sound comprehension and musical abilities have had "pure" word deafness. All patients consistently demonstrated more pervasive auditory agnosia, ranging from complete deafness to more subtle auditory processing disturbances.

### Neuroanatomical Correlates

There has been no unanimity regarding the site/sites of lesions responsible for the word deafness. Several studies reported that the lesion accountable for word deafness is subcortical in the left temporal lobe, disconnecting the auditory association area from the auditory inputs from both the right and left hemispheres (Hensche 1919, Ziegler DK 1952,[7],Kanshepolsky J *et al*. 1973,[8] Goldstein K 1948,[4] Maffei C *et al*.[6]). On the other hand, several others reported that autopsies proved such unilateral lesions produced word deafness. Some of these patients had significant hearing loss, ascribing the condition to peripheral sense organ disabilities (Bay E 1957[9]). However, Brain L (1965),[10] concluded that hearing impairment plays no part in producing word deafness.

Generally, the disconnection theory described above is favoured by most recent authors. Geschwind and others proposed that word-deafness was caused by disconnection or isolation of Wernicke's area from auditory input. (Geschwind N,[11-15] Goldstein,[2,4,16] Brick *et al.* 1985 cited by Buchman AS HB *et al*.[3]).

The bilateral primary auditory cortex may cause disconnection. Lesions or strategically located left subcortical lesions cut off the "ipsilateral and transcallosal auditory projection to the Wernicke's area. However, the unilateral lesions responsible for word deafness are uncommon. Generally, the syndrome is caused by bilateral lesions. Coslett (1984)[17] reported pure word deafness after bilateral primary auditory cortex infarcts. It must be pointed out that cases of word-deafness reported based on autopsy studies often demonstrate additional involvement of adjacent frontal, parietal and deep subcortical structures (Wohlfart G *et al.* 1952,[18] Jerger J *et al.* 1972,[19] Buchman AS *et al.* 1986[3]).

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# Recent Advances in the Neural Basis of Speech and Language: Implications for Education

Educational neuroscience is an emerging multidisciplinary field that links basic research in neuroscience, psychology and cognitive science with educational technology (Tandon PN and Singh NC, 2016).[1] As late as 1997, Jerome Bruer (1999),[2] a cognitive scientist, in an oft-quoted paper, "Education and the brain: A bridge too far," challenged the neuroscientists with a rather critical statement, "Educational applications of brain science may come eventually, but as of now neuroscience has little to offer to teachers in term of informing classroom practice that can serve as a basic science for development of an applied science and instruction. However, he also added, "Recently, however, interest in how neuroscience might improve education has moved from the margin to centre stage."

Fourteen years later, enough knowledge had accumulated for Stanislas Dehaene to state that "Human cognitive neuroscience has made enormous strides in understanding the specific cerebral circuits underlying particular domain of education such as mathematics, reading, second language education" and that "recent discoveries that demonstrate how the brain is changed by learning to read and how these results illuminate the specific hurdles that children face as they learn to read. Dehaene was convinced; "that empowering teachers with appropriate knowledge of the principles of human neuroplasticity and learning will lead to better classroom practices" (Dehaene S 2010,[3] 2002[4]).

Bruer JT (1997)[5] distinguished between mental science (cognitive science), basic brain science (neuroscience), and psychology (child psychology) as distinct disciplines. However, there is a great deal of overlap between these disciplines. Thus, scientists from the Institute of Leaming and Brain Science at the University of Washington, in a series of papers, agreed that new insights from many fields are converging to create a new science of learning that may transform educational practices" (Meltzoff AN *et al*. 2009,[6] Meltzoff AN 2013,[7] Kuhl PK 2004,[8] 2011,[9] Kuhl PK *et al*. 2008[10]). Others like Galaburda AM *et al*. (2006)[11] have extended the field from genes to behaviour in exploring the learning disability in dyslexia.

A detailed review of recent advances in the neuroscience of speech and language that have direct implications for education prompted this review of the current status of this field "Educational Neuroscience. Two main streams of knowledge link neuroscience to education:

(i) The brain structures responsible for various educational processes like speech, language acquisition, reading, writing, calculation, attention and memory, etc. and,
(ii) How do educational processes affect brain structure and function?

(Dehaene S *et al*. 2010,[3] Brem S *et al*. 2010,[12] Kuhl P and Rivera-Gaxiola M 2008,[13] Draganski B and May A 2008,[14] Zamarian L *et al*. 2009[15]).

Much of the knowledge on brain mechanisms underlying speech, language and learning has been acquired recently as a result of advances in non-invasive neuroimaging and allied techniques like PET, fMRI, EEG, EMG, SPECt, Near Infrared Spectroscopy, and Event-related Evoked Potentials. These studies have been conducted on young infants, adolescents, and adults (Raichle ME *et al*. 1994,[16] Cabeza R and Nyberg L 2000,[17] Imada T *et al*. 2006,[18] Dehaene-Lambertz G 2011,[19] Prather JF *et al*. 2017[20]). (Please also refer to the chapter 5 on Functional Development of Speech in the Human Brain in this monograph for details).

Educationists need to know that these studies have revealed that a structural organization of the neural network for speech is present in newborns, even preterm infants. Behavioural studies indicate that infants recognize their mother's voice in contrast to other feminine voices in the native language (De Casper AJ and Fifer WP 1980,[21] Mehler J *et al*. 1978[22]). Neuroimaging studies using fMRI showed several differences in the activation measured when infants listened to their mother's voice compared to an unfamiliar speaker. The mother's voice elicited a higher activation in the left posterior temporal lobe for language (Decasper AJ 1986,[23] Mehler J *et al*. 1978[22]).

Non-invasive brain imaging studies revealed a highly structured early organization of brain networks for language with hemispheric specialization and fast maturation in the first months of life. Battro AM (2011),[24] Dehaene-Lambertz G *et al*. (2006)[3,25] pointed out a very complex set of interacting brain systems in the initial acquisition of language early in infancy, similar to that reflected in adult language processing. Language exhibits a critical period' for learning. In the language domain, infants and young children are superior to adults despite their cognitive superiority (Knudsen EI 2004[26]). While infants have the universal phonetic capability at birth, in that they can initially hear all phonetic differences (Eimas PD *et al*. 1971[27]), between 6 and 12 months of age, non-native discrimination declines and between 9-12 months of age, native language speech perception shows a significant increase (Kuhl PK *et al*. 2004[8]). This knowledge is essential for the debate concerning primary education in the native language. At the same time, it suggests that learning a foreign language is best to start early in life. Infants can learn from natural exposure to a foreign language, and this learning is durable (Kuhl PK 2011[9]).

Another important observation from these studies revealed that a human being interacting with the infant during language exposure, while not required for simple statistical learning tasks, is critical for learning complex natural language-learning situations (Kuhl PK 2003[28]). Language material presented simultaneously and at the same rate via television or audio tape (in the absence of a human teacher) showed no learning. They also tested whether phonetic learning in infants is triggered

by hearing language by presenting a language material via DVDs compared to a live presenter. They found that at this age, learning is influenced by the presence of a live person. Questioning what a live person provides that a DVD cannot, they suggested that specific social cues may be critical. A live human being generates interpersonal social cues that attract infant attention, which motivates learning (DeLoache JS *et al*., 2010[29]). Battro AM *et al.* (2010,[30] 2011[24]) emphasized that experiments in second-language learning demonstrate that passive exposure to language is ineffective and that social interaction with an active tutor is essential. Experiments confirm the importance of teachers and families as providing a social environment optimally conducive to learning. These observations, if confirmed in more extensive series by other investigators, would pose a significant concern for present-day educators given the current increasing use of "Virtual" education (online education, unmanned education) owing to the shortage of teachers in large parts of the world).

The recent lockdown forced by the pandemic increased. The commercialization of electronic "Self-learning devices'. These studies, including those discussed earlier, highlight the value of the 'mother's voice' and indicate the importance of native language learning and the need to start education early in childhood. According to Battro AM (2011),[24] scientific knowledge is sufficient to conclude that early education can profoundly impact brain organization throughout life. An element not in the realm of neuroscience and not discussed in any detail here is the role of social environment on early life education as highlighted by several investigators (Knudsen EI 2004,[26] Duncan GJ *et al*. 1994,[31] Walker RB *et al*. 1994,[32] Meltzoff AN and Moore MK 1983,[33] Meltzoff AN *et al*. 2009,[6] Neville BA *et al*., 2011,[34] Noble KG *et al*. 2005,[35] Kishiyama 2010).

Knudsen EI (2004),[26] in his paper on "Sensitive periods in the development of the brain and behaviour," pointed out that "The growing body of work suggests that early language environment has a significant effect on the trajectory of language learning. It is well established that a more natural social setting extends the learning period and that manipulating other social features can either shorten or extend the optimum learning period". Meltzoff AN (2009)[6] pointed out that empirical evidence from his laboratory shows that "Human education fundamentally depends on social cognition. In this respect, he claims his studies illustrate the philosophy that education "begins at birth." Based on many personal studies and an extensive review of literature, Patricia Kuhl concluded, "We assert that talking to children early in life, and doing both these things while interacting socially with children around language and literacy activity, creates the milieu in which plasticity of brain during the critical period can be maximized for all children (Kuhl PK 2011).[36]

## Effect of Education on the Brain

The brain changes induced by education have been studied using non-invasive neuroimaging investigations. These studies revealed the significant impact of early education on domains such as language literacy, arithmetic, and reasoning. The brain of illiterate adults differs in several identifiable features from that of literate adults (Battro AM *et al*. 2011).[24]

A comparison of the literate and illiterate brains revealed how reading acquisition changes the brain within the visual and phonological systems. Massive changes are needed at the phonological and visual level before children can master the skill of reading. Recent studies indicate a very complex set of interacting brain systems during the initial acquisition of language early in infancy, many of which appear to reflect adult language processing (Dehaene-Lambertz G *et al*. 2006[25]). Interestingly, such changes were also seen in formerly illiterate adults who learned to read in adult life (Dehaene *et al*. 2011[19]). Mark Johnson (from the Centre for Brain and Cognitive Development School of Psychology., Birkbeck, College, University of London) described the "Functional brain development in humans"[37] and explained that "The infant's interaction with its environment helps to sculpt inter and intraregional connections within the cortex, eventually results in the highly specialized adult brain."

Human postnatal functional brain development is not just the passive unfolding of a maturational sequence but is an activity-dependent process, albeit guided and constrained by initial biases. It is becoming evident that new cognitive functions during infancy and childhood might result from emerging patterns of interactions between different regions of the brain. Some of these changing patterns of interactions between areas might also be characteristic of perceptual and motor skill learning in adults. By directing the infant to orient and attend to certain external stimuli, some brain systems effectively 'tutor' others with appropriate input for subsequent specialization. In this sense, the human infant is active in their functional brain specialization. Raichle ME *et al*. (1994)[16] described practice-related changes in human brain functional anatomy during non-motor learning. A meta-analysis by Macnamara BN *et al*. (2014)[38] revealed that deliberate practice and performance in music, games, sports, education and professions results in plastic changes in specific neuronal circuits. Hartzell L (2018)[39] found that the brains of verbal memory specialists like Sanskrit Pandits, who are trained to memorising some 40,000 to 1,00,000 word oral text, had massive grey matter density and cortical thickness increase in language, memory and visual systems, including in bilateral temporal cortices and the anterior angulate cortex and hippocampus regions. Structural changes were observed in the hippocampus of taxi drivers as a result of their navigation-related activities (Maguire EA *et al*. 2000[40]). Experience-dependent structural plasticity in the adult human brain has been confirmed by May A (2011).[41] A better understanding of structural and functional brain development in human infants and children due to learning will profoundly affect educational policies.

Dehaene (2010)[3] focuses recent discoveries demonstrating how the brain is changed by learning to read and how these results illuminate the specific hurdles children face as they learn to read. He is convinced that empowering teachers with appropriate knowledge of human neuroplasticity and learning principles will lead to better classroom practices.

Cognitive neuroimaging in literate adults has clarified how reading operates at the cortical level. An extensive set of neurons in the left hemisphere are identically activated when we read sentences and listen to them (Buchweitz A *et al*., 2009[42]).

This language network comprises temporal lobe regions, most prominently the entire length of the superior temporal sulcus from the temporal pole to the posterior temporal and parietal junction and the distinct regions of the left inferior frontal lobe. These regions are thus not unique to reading. Instead, they are spoken language areas, and reading provides them access through vision. Overall, comparing literate and illiterate brains emphasizes how reading acquisition changes the brain, not just within the visual word form area but also in the visual and phonological systems. These systems are highly plastic in that virtually all of the changes were seen in partial form in adults who learned to read in adulthood. Most of the research thus far regarding language learning and the brain has focused on one or two languages. However, a large part of the world is multilingual, and in the next section, we discuss multilingualism and multiliteracy and its impact on the brain.

## Multilingualism and Multiliteracy

Multilingualism is the ability of societies, institutions, groups and individuals to communicate in more than one language in their daily lives (European Commission, 2007:6). While the term multilingualism varies across studies (Cenoz J, 2013[43]) wherein in some cases, multilingualism is the ability to speak more than two languages, in other cases, it is used interchangeably with bilingualism. Here, multilingualism refers to individuals fluent in more than two languages. The opportunity and affordability, along with new communication technologies provided by the internet, have increased the relevance and importance of multilingualism in education. Due to migration, several school children have different home languages from the ones used at school, influencing their language learning process. The structure of school curricula mandated that students learn the language(s) of instruction in addition to that spoken at home. Neuroimaging research from language production and word recognition in multilingual (e.g., Poarch GJ and van Hell JG, 2012a;[44] 2012b[45]) shows that they simultaneously activate all of their languages, even when behavioural demands are only for a single target language. Thus, when multilinguals encounter a spoken word in any one of their familiar languages, words from non-target languages are also simultaneously activated, even though they might not be relevant. This has led to the hypothesis that (a) languages are likely to be represented in similar brain areas in multilingual and (b) multilingual proficiency must require managing interference from non-target languages. This has led to extensive research on cognitive control mechanisms in multilingual (Bialystok E *et al*. 2012),[46] wherein conflict monitoring and attentional control are critical factors for multilingual proficiency. Multilingualism may be early, late, sequential, and simultaneous. When it is concurrent, languages are learnt concurrently, while when it is sequential, literacy is first acquired in one language, followed by others.

Even though multilingualism is prevalent to a large extent, research in this area is scarce. With increasing globalization, future research must focus on this to enable evidence-based learning strategies for learning multiple languages in education.

## Technology and Language Learning

Speech and language (verbal and written) are the primary modes of education. Past decades have witnessed the introduction of various technologies to supplement or replace the classical teaching and learning methods. These technologies offer advantages, but many studies have highlighted their inappropriate and excessive use. A recent UNESCO report (GEM Report 2023)[47] provides valuable insight. Some selected key messages are produced below.

### Key messages of GEM report 2023 (UNESCO)

1. There is little robust evidence of digital technology's added value in education. Technology evolves faster than it is possible to evaluate it. Education technology products change every 36 months on average. In the United Kingdom, 7% of educational technology has conducted randomised controlled trials, and 12% have used third-party certification
2. Technology offers an education lifeline for millions (as happened during covid pandemic) but excludes many more. In the US, an analysis of over two million studies found that learning gaps widened when instruction was exclusive
3. Technology can harm inappropriate and unsupervised use
4. Large-scale international assessment data, such as that provided by the Programme for International Students. Assessment (PISA) suggests a negative link between excessive ICT use and student performance. Mere proximity to a mobile device impacted learning in 14 countries, yet less than one in four have banned smartphone use in schools. The harmful aspects of using digital technology in education and society include the risk of distraction and lack of human contact
5. The unregulated use of technology even poses threats to democracy and human rights, for instance, through invasion of privacy and stoking of hatred. Clear, evidence-based objectives and principles are needed to ensure that technology benefits learning and avoids harm
6. Issues of equity and inclusive access for disadvantaged groups have been raised. A wide range of technology brings education to hard-to-reach learners.

Generative artificial intelligence is the latest technology touted as having the potential to transform education. In contrast to conventional ICT, which has been used in various forms for many years, using new generative AI-based solutions like Chat GPT in education has opened up a pandora's box, which has already alarmed its founders. Sam Altman, one of the founders of OpenAI, pleads to develop international rules for its regulation. Some countries like Italy have already banned the use of chat GPT, while others like the USA, Europe and India are seriously considering regulating it. Systemic reviews over the past two decades of technology's impact on learning find small to medium-sized positive effects compared to traditional instruction.

Chat GPT is already used in many countries and India for a variety of purposes by both teachers and students, especially in higher education. This includes helping generate ideas for tackling complex problems, summarising long texts, and creating quizzes at the end of the lesson, as teaching aids for personalized tutoring. Chat GPT can produce exceptional written text which has been misused for answering question papers. It can analyse large amounts of data, identify patterns

and recommend the most efficient solution to complex problems. Several AI tools are already available for specific purposes, and more are being added. While this takes away students' original, independent thinking, its use results in identical essays, and similar arguments being submitted.

A college teacher in Delhi, where students are increasingly using such AI technologies, succinctly commented. "AI is here to stay. AI can become a useful tool in education. The thing is not blindly resisting it but incorporating it with calibration and synergy." Significant revisions would have to be made to curricula, and the examination methods must be thoughtfully modified. The problem is that technology is changing faster than a pedagogical modification to adjust to these changes, which is, at best, too slow. If a tool can produce a classical text in no time, it can create original linguistic text requiring original ways of understanding it. Are we prepared for it, or are we even initiating programs to deal with it?

In conclusion, education policy and practices should be continuously updated to reflect the best, most reliable and replicated practices in language learning. The role of technology cannot be ignored since it is here to stay and is a part of people's lives. Instead, by understanding its benefits and being alert to its harms, its use must be regulated and monitored, especially in the early years. Finally, a recent report entitled the International Science Evidence-based Education Assessment (ISEEA), a mammoth exercise undertaken by the UNESCO Mahatma Gandhi Institute of Education and Sustainable Development (MGIEP) summarized the best learning practices provided by the neuroscience of learning. The link to the report is https://mgiep. unesco. org/iseereport. This resource is of great value for teachers, policymakers and researchers and its use and reference are greatly encouraged.

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# EPILOGUE

Speech evaluation is essential to clinically examining patients with suspected neurological disorders. This, then, was part of our daily professional activities. Yet it was only recently, years after giving up professional work, that the present author realised how little he knew about speech and language except for various types of dysphasia and some knowledge of their neuroanatomical basis, primarily the contributions of Paul Broca (1861, cited in Neuropsychology review in 2011,[1] from his article in 1865. See also Tandon PN, 2024[2]) and Wernicke C (1874)[3,4] two hundred years ago. Many clinico-pathological studies that followed after this were of concern only for the experts in the field. As described in "Genesis of this monograph," the author got interested in the subject, reviewed a lot of recent literature (and some standard earlier ones) and realised that while the subject was of interest to scholars thousands of years ago, before Broca and Wernicke.

Thus, philosophers, psychologists, anthropologists, and linguists, besides physicians and neuroscientists, studied various aspects of Speech and language, mostly as isolated individuals and few in some well-known "schools" (See Tandon P and Chandra PS 2022).[5] Much later, clinicians (neurologists, neurosurgeons, neuropsychologists, and neuropathologists) provided new insights.

Broca's (1861) assertion that the integrity of the posterior part of the third frontal convolution was indispensable to articulate Speech laid the foundation of its cerebral localisation. However, the absence of a complete dissection of the autopsied brains resulted in the original concept being based on incomplete knowledge of the extent of the lesion. Already challenged by contemporary colleagues like Charcot (1825-1893), Pierre Marie (1853-1940), Hughling Jackson (1835-1911) and others, the recent neuroimaging techniques have provided a comprehensive account of the neuroanatomical basis of verbal Speech (all cited by Tandon and Chandra in Evolution of Neurosciences.[5] See also Chapter 4a-c).

Like Broca's aphasia and Broca's area, it is now well established that neither the clinical syndrome nor its neuroanatomical correlation was as well restricted as described by Wernicke C (1874).[4] This was already pointed out by Charcot and Marie (1888, cited by Brais B 993),[6] Henschen S (1919),[7] Henschen S (1924),[8] Geschwind N (1971),[9] and Bogen and Bogen JE (1976).[10] A recent review by Margolin DI (1991)[11] concluded that the classic Wernicke's syndrome reflects damage to no less than nine distinct language processing modules.

In addition, it has helped linguists and sonologists to study in detail the parameters which delineate the physical features of speech, revealing its tone, quality of sound, dialect and a sonological feature which have been used for centuries for speech therapy and legal purposes. More recently, it has been used to fine-tune brain-machine interactions and diverse aspects of AI, believe it or not.

The present monograph highlights that the science of speech and language has seen spectacular advances in recent years. Most of the new knowledge from collaborative efforts has been added since the late 1990s. Mithen S (2024)[12] mentions around 2020, "I began to suspect that embedded in the recent research by linguists and archaeologists, computer scientists and anthropologists, philosophers, psychologists, and geneticists were fragments of comprehensive accounts for language evolution."

An account that could build the necessary bridges between disciplines and would stand the test of time despite the inevitability of discoveries and new ideas. Finding those fragments from within so many disciplines was only half the challenge.

The US National Institute of Health and nearly one dozen other institutions have recently been trying to develop AI programs to use these applications that will diagnose diseases through speech analysis. Every nuance of Speech will be scrutinised, from volume, pace, and intonation to vocal cord vibration, to diagnose not just speech disorders but also neurological disorders, mental health and respiratory problems and spectrum disorders like autism. Based on a large amount of data collected from all over, it is proposed that it is becoming close enough to detect a range of diseases just by hearing a few seconds of voice clips. It is claimed that the AI tools can pick up type 2 diabetes, Parkinson's disease, Alzheimer's disease, depression and schizophrenia, bipolar disorder, cancer of the larynx and vocal cord paralysis by utilising the speech spectrum. A study recently published by the Mayo Clinic supports these possibilities.

However, as late as 2011, Kuhl PK [13] highlighted, "We are still just breaking ground regarding the neural mechanisms that underlie language development and its critical period. In the last decade, brain and behavioural studies indicated a complex set of interacting brain systems in the initial acquisition of language early in infancy, many of which reflect adult language processing."

This knowledge is not only of academic interest but has permitted diagnosis for safe surgery in the dominant hemisphere (Tandon PN *et al*. 1986,[14] Garfield J 1987,[15] Duffau H *et al*. 2002,[16] 2015,[17] Chandra PS *et al*. 2008,[18] 2015[19]).

In his erudite book, "The Language Instinct: How the Mind Creates Language," Steven Pinker (1994)[20] pointed out that, some thirty-five years ago, a new science was born now called "Cognitive Science." It combines the tools from psychology, computer science, linguistics, philosophy and neurobiology to explain the location of human intelligence" to a defined area of the brain. It is essential to remember Critchley's (1970, cited by Abbamonte in 2007 translating Critchley's work)[21] observation in his book "Loss of ability to use of language has

been recognised long before Broca's description of it being due to a localised brain lesion. And almost all forms of dysphasias had been described before 1800."

While there is much commonality between speech and language puzzles, it is clear that they are not synonymous. Speech, the vehicle of language, involves a different neural substrate than language, a complex and well-defined communication system used by the community of human beings to express their thoughts, ideas and emotions. It has rules, conventions, and grammar that enable them to understand each other's communication, whether vocal, written, or written (Scott SK and Johnsrud IS 2003).[22]

Notwithstanding the recent spectacular advances, it is humbling to recognise our ignorance about "How language became human's superpower? How were words formed? How did we talk our way out of the stone age?" as discussed by Mithen S (2024).[12] According to Mithen, "The origin of language is one of the most contested problems in science. Linguistics, neuroscience, anthropology have all claimed pieces of jigsaw, but the picture is only clearing now". The author traces the steps from the early vocalisation of our ancestors to words, metaphors and writing, leading to cognitive and social change". Needless to say, it is still far from being completely understood.

Over 400 languages worldwide are derived from a single extinct language from the Bronze Age-Proto-Indo-European. As people dispersed east and west, their language changed through mistakes and contacts with other people.

Despite long-standing studies of evolution biology, it would be generally accepted that our knowledge of human speech and language evolution is still primitive. We know little about how the babel of an infant is converted to vocalisation, which can develop into the language of a poet, a philosopher, or a scientist. With all the advances in computer science, machine learning and AI, many advances have been made in understanding the basic mechanisms underlying this unique human trait.

As briefly summarised in the chapter on Functional Development of Speech and Language in the Human Brain (Chapter 5), we have come a long way from the authoritative views of Broca and Wernicke about the localisation of speech in a small restricted area of the cortex. Evidence exists to challenge this revolutionary concept. Recent studies of speech function of healthy volunteers and patients with well-defined focal lesions as revealed by modern neuroimaging have shown that Broca's original description of the brain lesion responsible for aphasia has several limitations, Mohr *et al*. (1978),[23] Goodglass H *et al*. (1986),[24] Petersen and Petersen (1989),[25] Nadeau SE (1988).[26] We know that many more cortical areas than the conventional Broca's and Wernicke's areas are involved in speech and language processing. These include the supplementary motor area, insular cortex, the corpus callosum, the cerebellum, parts of the thalamus, basal ganglia and limbic system, in addition to the whole new field of connectomics, including the role of white matter fasciculi. The visual and auditory cortex also plays an additional role in the neuronal substrate for speech and language perception and processing. It is equally valid for the neuronal substrate of memory as an essential part of the total speech and language function. However, we still know little about how the functions of these units are integrated to provide a coherent expression of human thought, wisdom, emotions, scientific, technical intuition, and mathematical equations.

According to Pinker S (1994),[20] the most authoritative views in this century come from Naom Chomsky, the linguist who first unmasked the system's intricacy and perhaps the person most responsible for the modern revolution in language and cognitive science. Chomsky called attention to two fundamental facts about language. First, virtually every sentence a person utters or understands is a brand-new combination of words, appearing for the first time in the universe's history. Therefore, a language cannot be a repertoire of responses. The brain must contain a recipe or program to build unlimited sentences from a finite list of words. The program may be called a "mental grammar." The second fundamental fact is that children develop these complex grammars rapidly and without formal instruction. With my scientific background, this is undoubtedly thought-provoking but still fails to answer the fundamental question of how and what neural mechanism develops this "grammar" and converts it into a new language. But I have learnt that by now, the community of scientists studying the questions has increased to numbers in thousands.

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# ABOUT THE EDITORS

Dr (Prof.) PN Tandon (born 13 August 1928) is a highly celebrated Indian neuroscientist, neurosurgeon, academician and teacher of teachers. He graduated with flying colors (Hewit medalist) MBBS and MS from the King George's Medical College (KGMC now KGMU), Lucknow in 1950 and 52 respectively. He then went to United Kingdom for further studies and obtained his FRCS in 1956. He further obtained specialist training in neurosurgery in Oslo, Norway under Dr Kristiansend and then under Dr Penfield at the Montreal Canada where he was awarded the Penfield Medal for his contributions.

He was driven by a passion to develop the speciality in his own country and on returning to India, after a brief tenure as a professor at KGMU (1963-1965), he moved to the All India Institute of Medical Sciences (AIIMS), New Delhi where he founded the neurosurgery department as a professor of neurosurgery. During his tenure at AIIMS, he served as Honorary surgeon to the President of India (1977-1980) and member of Science Advisory Council to the Prime Minister (1986-1989).

After his super annuation from AIIMS, he continued to contribute to the field as Professor Emiritus and Bhatnagar fellow, Council for Scientific and Industrial Research (CSIR). Professor Tandon, an elected fellow of the National Academy of Medical Sciences, was the President of the Indian National Science Academy (INSA) in 1991-92, Co-Chair Inter-academy Panel of the World Academics of Sciences 1994-2000, member of the University Grants Comission 2004-2007 and President of Indian Academy of Neurosciences 2007 & 2008. Prof Tandon also serves as teh President of the National Brain Research Society, Manesar, Haryana, India. In recognition of his remarkable contributions, he was awarded the Padma Shri (1973), Padma Bhusan (1989) and Padma Vibhushan (2006) by the Government of India.

He achieved internation recognition as member of Indo-US Vaccine Action Program for two decades, member of the indo-US Science and technology forum and member of the International Bioethics Committee (UNESCO) 2004-2007. He was elected Fellow of the 3rd World Academy of Sciences, Royal Society of Medicine, Norwegian Academy of Science and Letters and Foreign Member of the Society for Neurosciences, USA. He has around 350 publications to his credit including 34 books and monographs, 55 book chapters and 250 original research papers.

Dr (Prof) P Sarat Chandra is a world-renowned neurosurgeon and is currently Professor and Head of the Department Neurosurgery at the All India Insititute of Medical Sciences (AIIMS), New Delhi. He graduated from MKCG Medical college, Odisha (1991) with flying colors (Pfizer medal awardee for best graduate) and then completed his neurosurgical training from NIMHANS,Bangalore (1996).

He completed his post Doc fellowship from UCLA. He is best known for his path breaking work in the field of epilepsy surgery, craniovertebral junction anomalies, complex spine and minimally invasive neurosurgery. He has described several minimally invasive and safer surgical techniques, which are now followed world over. He has had a huge experience of more than 25,000 surgical cases consisting of a wide spectrum of neurosurgical pathologies that include epilepsy and functional, complex spine, tumors, vascular, skull base, and endoscopic surgeries. He is a well-known lecturer having given more than 500 lectures world-wide has over 590 publications including some of the highest rated journals like NEJM and Nature and have over 14 extramural research grants and 7 patents. He is a recipient of several National and International awards (to mention a few Asiano-Oceanic Epilepsy Congress/ International League Against Epilepsy outstanding award, AIIMS Excellence award, Vasvik industrial award, Sun pharma award for the best researcher, Career 360 excellence award) and has delivered several orations (KS Mani oration, AD Sehgal oration, IES; Newton-Dave oration, KGMU; JIPMER research day oration; BS Sharma oration, BC Bansal/ Uma Bansal oration, PGI-Rohtak, Clinical grand rounds (UCLA, UCSF, New York Presbyterian University, Thomas Jefferson University).

He has leadership positions in reputed scientific societies (President, Indian Epilepsy Society and Neuroendoscopic Society; Past President, Cerebrovascular society and Skull Base Society, Past President of Asian Epilepsy Surgery Society, Co-Charman, AOEC, ILAE).

He established the country's first magnetoencephalography at NBRC, Manesar and the country's first Epilepsy Neurobiology lab, which performs intra-cellular electrophysiological studies on 'live' in vitro brain resected specimens to study brain connectomics.

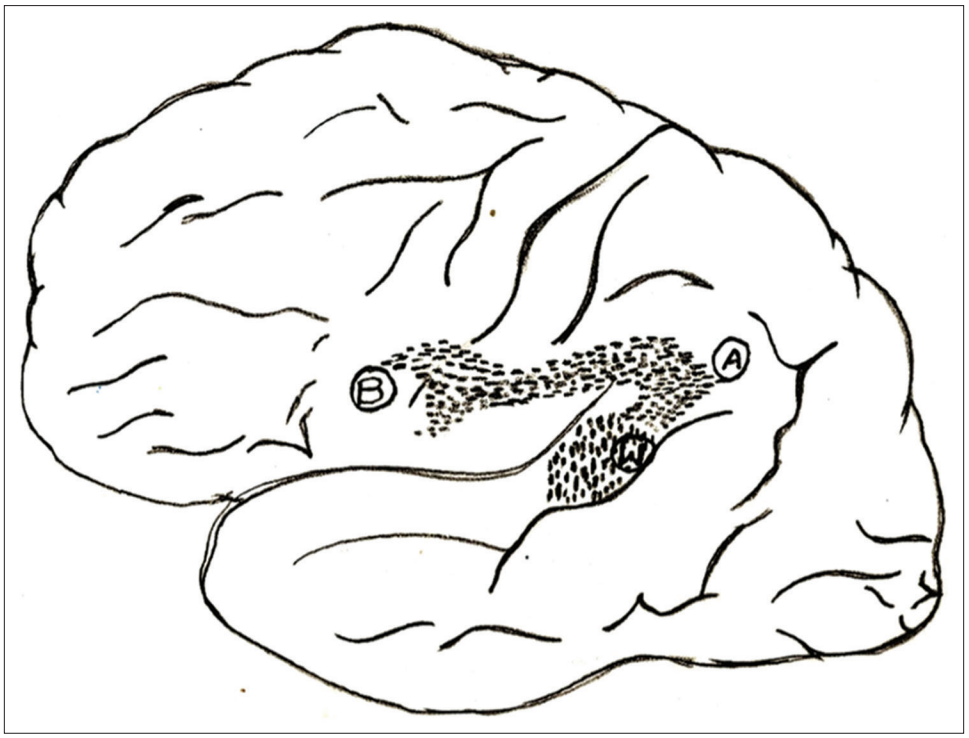


**Figure 3:** Schematic diagram of Geschwind's diagram (1970) depicting conduction aphasia pathway. B-Broca's area, W-Wernicke's Area, A-Arcuate Fasciculus. Despite the good comprehension of spoken language there is a gross defect in repetition. The lesion for this disorder typically lies in the lower parietal lobe, and is so placed as to disconnect Wernicke's area from Broca's area. The disorder in repetition exhibits some remarkable linguistic features which are not yet explained.
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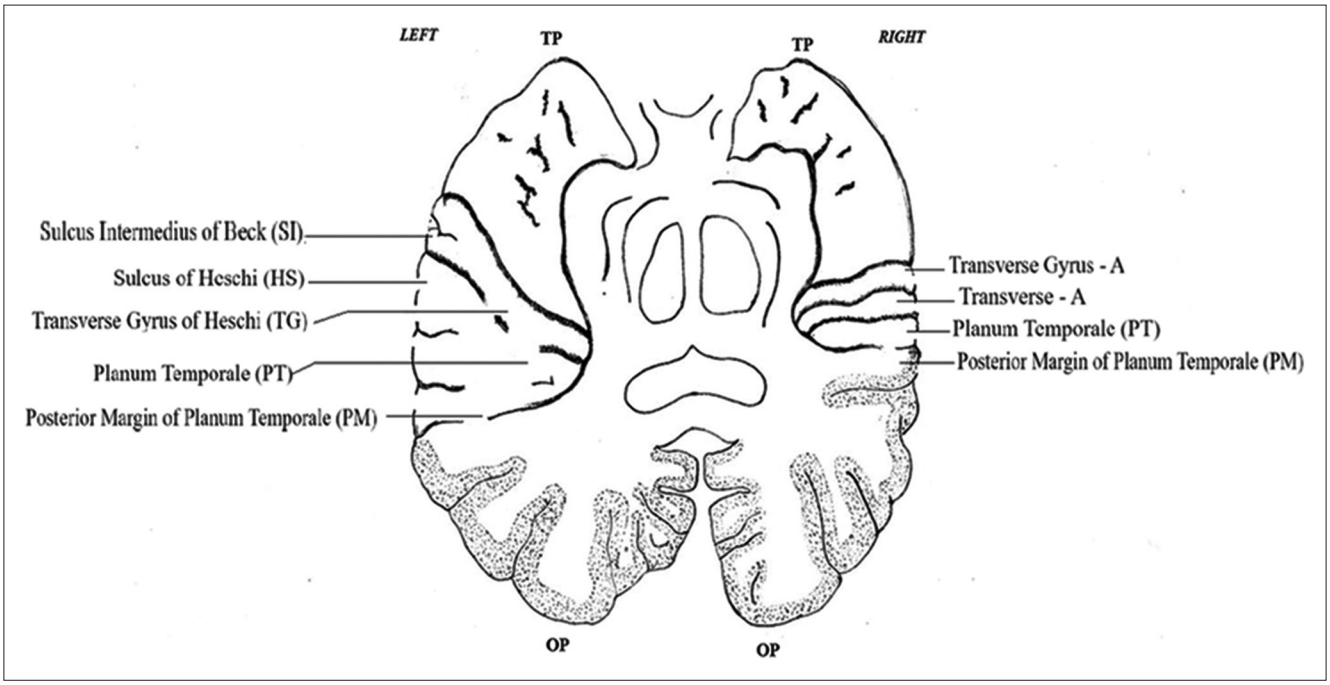


**Figure 2:** Schematic diagram showing left-right hemisphere surface area differences at the level of superior temporal surface. Figure shows Steep slope of Posterior Margin of planum temporale (PT) compare to right and steep slope of planum anterior margin (formed by Sulcus of Heschl (SH) on the left compare to right.
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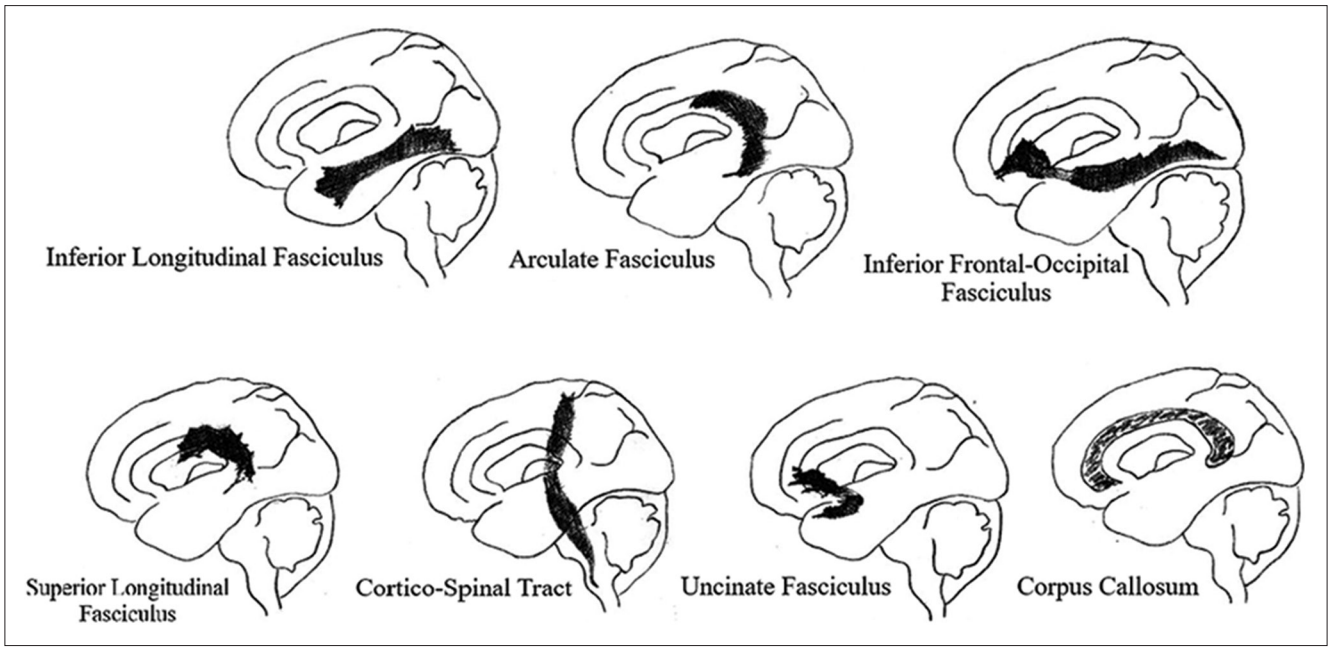


**Figure 1:** Schematic Diagram of various white matter tracts as seen in diffusion tensor imaging of MRI.
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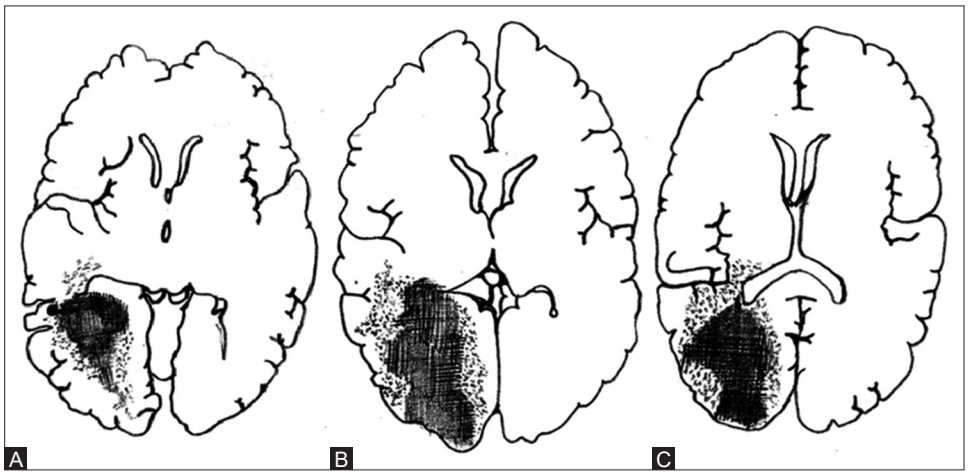


**Figure 4:** Schematic Diagram showing computed tomographic images at 15 degree. Sequential cuts above Orbitomeatal line. A – at the level of Broca's area, B – at level of Wernicke's area, C – at the level of Supramarginal gyrus. Figure shows sparing of occipital cortex and lesion involves left Parieto-Occipital region and Occipito-temporal fasciculus regions.
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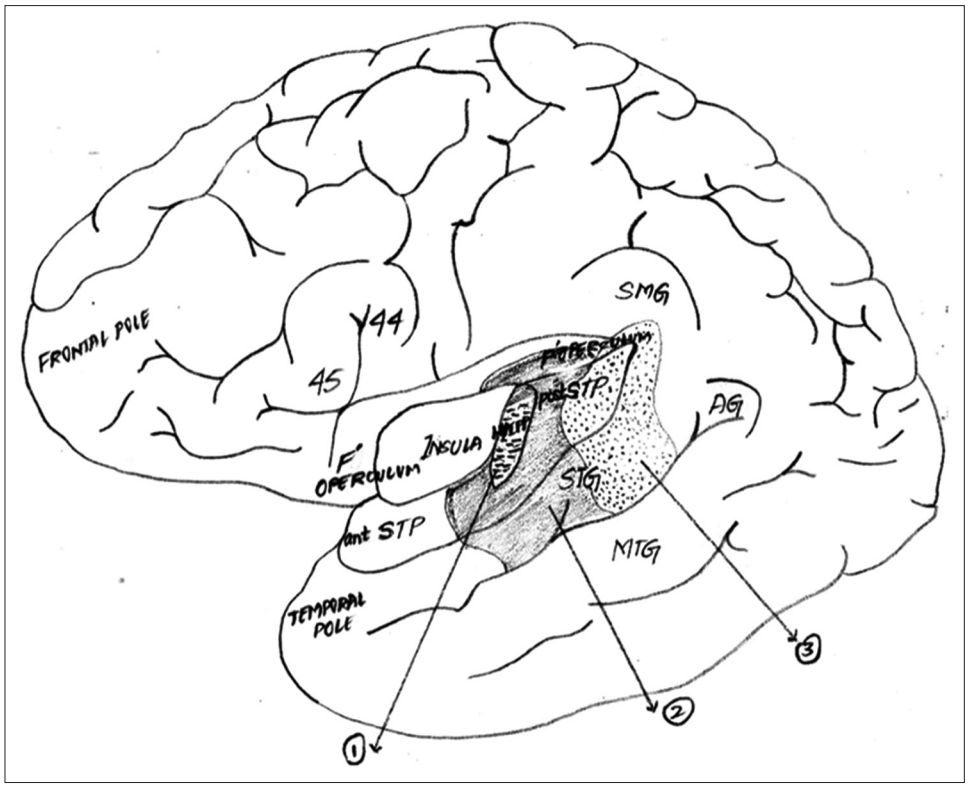


**Figure 5:** Schematic Diagram showing lateral view of left hemisphere with structures inside the exposed Sylvian Fossa. F' Operculum-frontal operculum, P' operculum-parietal operculum, SMG-Supramarginal Gyrus, AG-angular gyrus, MTG-Middle temporal gyrus, STG-superior temporal gyrus, HIPP-Hippocampus, ant STP-anterior superior temporal plane, post STP-posterior superior temporal plane, 44-45 corresponds to Broca's area. Shaded areas correspond to rough Cytoarchitectonic representation labelled as 1-Primary, Koniocortical Fields, 2-Secondary Parakoniocortical Fields, 3-Transitional Fields representing transition between unimodal sensory cortex to multi modal integration cortex.

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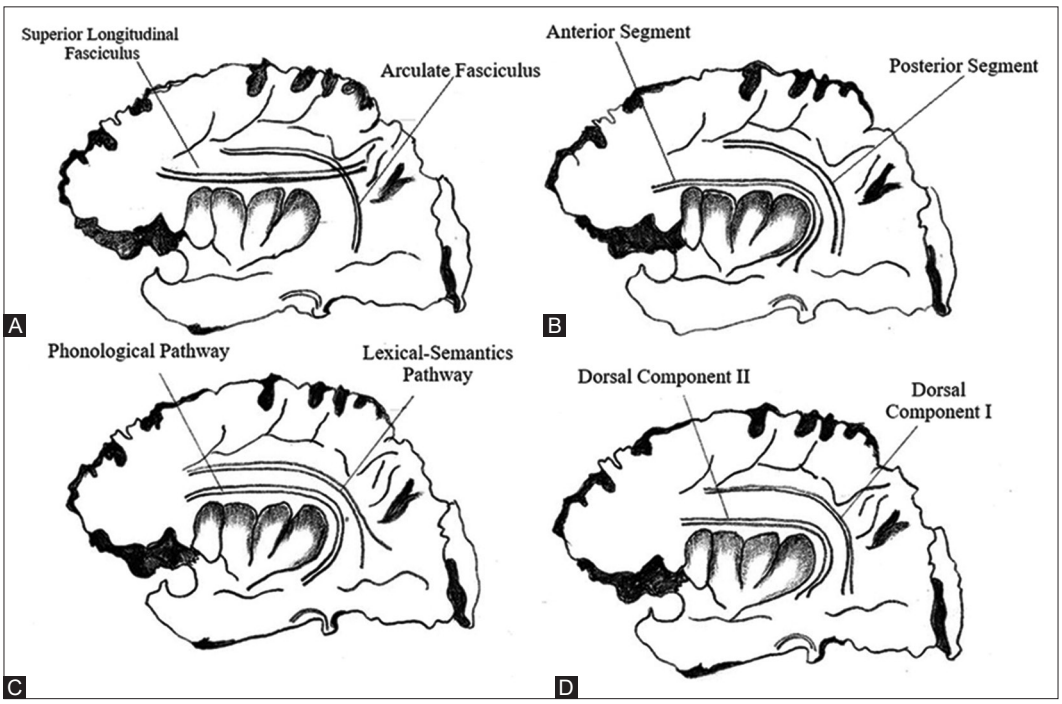


**Figure 2: A:** Three segment model by Catani-*et al.* **B:** Two segment model by Glasser and Rilling – 2018, **C:** Two segment model by Makaris-*et al.* – 2005, **D:** D- Two segment model by Friedericz-*et al*. (Chapter 4c,Page no.72)

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