Brain and Language: A study of disorders

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Abstract

Linguists, Psycholinguists and Cognitive Neuroscientits have always tried to connect parts of brain to the various functional modalties of language. This can be done through various kinds of studies like studies of disorders, PET studies, fMRI data studies and others. In this paper we discuss two kinds of disorders: 1. One of them is a very specific disorder of language in children - Specific Language Impairment, 2. The other is a more general disorder and has a range of syndromes - Aphasia. Through these studies we will try to find out functional relationships between areas of brains and linguistic abilities.

1 Specific Language Impairment

Specific Language Impairment or SLI is a language deficit which is not accompanied by hearing impairment, low non-verbal intelligence test scores or neurological damages. Thus children with SLI show no signs of any other factor that usually accompanies language learning problems. The prevalence of SLI is about 7%. A child with a history of SLI in the family is a more likely candidate than a child with normal background.

1.1 History of SLI

Work in this area dates back to early nineteenth century. In 1822, Gall published a description of children who had clear problems in language but did not display any other known disorders - "There are many children who do not speak to the same degree as other children although they understand well or are far from being idiotic...". Galls report was followed by large number of case studies published mostly by physicians. The authors emphasized on apparently normal non-verbal intelligence but extremely limited speech output of these children. Earlier, the studies focused on children with severe output limitation. Gradually the focus widened to include children who produced multi-word utterances and the grammatical deficit in these children started getting the attention.

1.2 An Example

Here are some brief excerpts from one english speaking child with SLI, aged four years and three months. The child was shown sets of sequence pictures and asked some questions. These pictures depicted a story:
Adult: Ok, ready?
Child: Ready.
Adult: This is Jim. Tell me a story about Jim.
Child: Him going fishing. Jim holdwater. And ... go fish. And ... [unclear]
Adult: I did not hear this one.
Child: I dont know.

The childs utterances were quite short on average. Omission of grammatical suffixes and function words were very common. On some occasions over-regularization was also seen. The child in question enjoyed

interaction but initiations of verbal exchanges were very less. Communicative efforts were often abandoned if they were not understood on the first try.

1.3 Language Characteristics of SLI

Now we will look at the production and comprehension of English by children with SLI. We will try and bring out the linguistic strengths and weaknesses of English speaking children with SLI. We will limit our attention to the lexical and syntactic abilities of these children.

1.3.1 The Early Lexicon

Children with SLI appear to be late in acquiring their first words. Earlier case studies provided evidences for this fact. Bender(1940) observed a child who failed to produce his first words until four. Recent studies with large number of children confirms the earlier works made by Bender. In a study of 71 children with SLI, an average age of 23 months for first words was reported as compared to an average age of 11 months for normal children.

The types of words used by children with SLI seems to match the types observed in younger normal children. General nominals (names of objects, animals etc.) constitute 55% of the words whereas words referring to actions, properties constitute 12% each. Thus we can identify the deficiencies as a general lag. With the onset of multi-word utterances, children with SLI show marked deviation from normal younger children. The deficiencies go beyond the general lag evident so far.

Preschool Years The studies made by Chapman, Leonard, Rowan and Weiss (1983) revealed that children with SLI acquired as many words as normal children given a large exposure to words. This contradicted the earlier studies that suggested that lexical acquisition in children with SLI was slow. This led to a new concept of fast-mapping in children with SLI being taken up. It was found that on large number of repeated exposure to novel words, both the groups acquired almost same number of words. However, on smaller exposure (say 3 times) children with SLI showed poorer results. It was also found that action words were not retained even on large exposure to words. Thus the retention mechanism was also poor in such children.

School Years Studies of school going children with SLI gave an interesting comparison. It was found that children with SLI learned object names almost as well as normal children but their learning of action names fell well below par. The lexical limitation is generally identified as "word finding" problem wherein a child faces difficulty in generating a particular word called for in the situation. This deficit has also been described as "lexical lookup" problem and problems involving delayed speed of word retrieval. The chief symptoms of word finding problems are unusually long pauses in speech, frequent use of non specific words such as it or stuff. One possible explanation of word finding difficulty can be attributed to problems of retrieval. This suggests that words are present in the memory but children use an inefficient means of retrieving them. Yet another explanation is based on the connectionist approach which claims that SLI acts as a filter which weakens the network between phonology and semantics of the word.

1.3.2 Early word combinations

As expected, age of first word combination appears to be later in children with SLI than normal children. Studies have revealed that children with SLI show much narrower scope of word combination. There scope is narrower than the general notions of word combinations like agent + action or attribute + object. For a given notion of word combination like agent + action a child aged four years had only me as the agent (e.g., me do, me make etc.)

Verb Learning We have seen that lexical abilities of children with SLI is most hampered in the case of verb learning. It has been demonstrated that learning of verbs require exposure to the sentence frame in which the verb appears along with the event being described by the verb. This process of interpretation of meanings of verbs based on sentences might be the major weakness in SLI, hence accounting for the difficulty in verb learning.

1.3.3 Grammatical Morphology

It has been found that grammatical morphology constitutes a relative weakness in children with SLI. In a case study by Gopnik (1990) an eight year old child with SLI exhibited errors on many inflections and function words involving tense, person, number and gender. Children with SLI showed lower degrees of use of regular past ed with both real and nonsense verbs. They also showed fewer over-regularization error.

1.4 Neural Mechanisms

Now we will look into neural mechanisms used for encoding rules of languages in humans limiting ourselves to English past tense. We will look at the deficits in SLI in order to study the various models proposed for past tense inflection. Two models under consideration are the connectionist model and the symbolic rule model.

1.4.1 Brief Overview

Symbolic Rule Model This model proposes that the regular inflections are obtained by applying a symbol concatenation rule (-ed) whereas irregular verbs are in memory along with their past tense.

The Connectionist Model All morphological forms are processed within one type of processing mechanism (connectionist network) distributed across multiple brain regions. Past tenses of verbs are obtained by "convergence" of code for semantics and phonology. Since a verb and its past tense share regular similarities in both semantics and phonology, we can encode a rule for past tense by these statistical regularities.

1.4.2 SLI as a Rule-Learning deficit

Primary source of data about morphological deficits of children with SLI comes from studies using sentence completion task
e.g., The girl likes to walk. She did the same thing yesterday; she ....
Theories of SLI as a rule-learning deficit predict that children with SLI will have difficulty in producing the past tenses of regular verbs and will perform better with irregular verbs. However, studies have revealed that this is not the case.

Children with SLI make fewer over-regularization error than normal developing children. This suggests that their creativity in producing the past tense of unheard words is limited. Moreover these children are also limited in producing the past tenses of non-words (which should ideally be word+ed). This again suggests that they have not encoded a rule for generating past tenses. However, this hypothesis fails to explain the difficulty encountered by these children in producing past tenses of irregular verbs.

1.4.3 The Phonological Deficit

This is another hypothesis for explaining the morphological deficits in children with SLI. It is based on the connectionist model which says that past tenses normally arise through integration of phonology and semantics. This hypothesis proposes that a perceptual deficit leads to a phonological deficit which is the direct cause of language problems in SLI.

The deficit in phonology leads to a relatively small semantic representation which in turn impairs the ability to generalize from known forms. This limited semantic representation supports past tenses of familiar forms but does not come into play for unfamiliar forms. Since non-words rely completely on phonology, a phonological deficit will hamper the past tenses of non-words the most.

1.4.4 Modeling SLI

This modeling is based on the connectionist model and was done by Marc F. Joanisse (2000). It investigated the effect of perceptual deficit on learning past tenses. The network was trained to associate the meanings

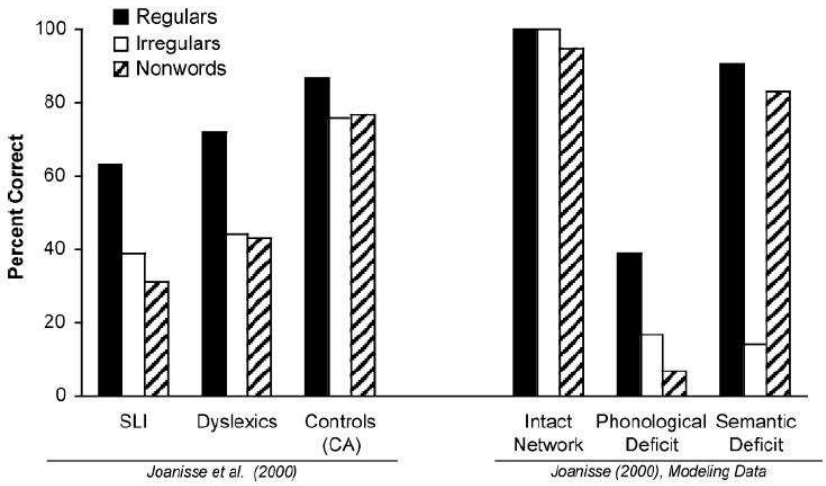

Figure 1: Results of the modeling

and sounds of English present and past tense verbs. During training, the network showed a slight delay in learning irregular forms relative to regular forms. At the end of the training, it had learned all tasks accurately. Now, the training process was altered a bit. Small amount of random noise were added to the phonological representations of the training words, thus simulating a deficit in perception of speech. This newly trained model showed a pattern of past tense production which was consistent with SLI. Compared with intact model, this model was poorer at learning all three types of past tenses (Figure 01). As it can be seen from the results, the model is consistent with children suffering from SLI.

1.5 SLI and Genetics

For many years genetic factors as a possible cause of SLI was ignored But when the focus shifted to childs family background, the concept of linguistic input to the child gained importance. Samples and Lane (1985) described a family in which all the six children had SLI It was reported that approximately 30% of the immediate family members of children with SLI had language problems.

However, studies have revealed large number of cases in which language problems are limited to the child. Thus we have dual findings. There can be two possible interpretation of this dual finding. One possible interpretation is that there are different causes of SLI, some are genetic, others not. Other possibility is that although genetic factor is present but with incomplete penetration. Thus there must be additional factors causing SLI.

1.6 Neuroanatomical correlates in SLI

An autopsy study of brain of four males with a history of SLI was made by Galaburda, Sherman, Rosen, Aboitiz, and Geschwind in 1985. One of the key findings was a symmetry of plana temporale. This is located in the upper portion of temporal lobe in each hemisphere. Typically the planum temporale of left hemisphere is larger than the right one. However, inn the case of these individuals the two plana were of the same size. This was due to abnormally large right planum temporale in these individuals.

1.6.1 MRI Scan

Plante looked for such left-right hemisphere symmetries in children with SLI using MRI techniques. The shape and location of planum temporale present obstacles to measurement from MRI scans. Hence, she measured a broader area around the sylvian fissure called the perisylvian area. Like plana temporale, the perisylvian area also shows left-right asymmetry with the left hemisphere exceeding the right hemisphere in size.

One of the studies involved a boy with SLI age 4;9 and his normally developing dizygotic twin. The child with SLI showed symmetry of left and right perisylvian area which resulted from abnormally large right perisylvian area. This finding was aided by subsequent studies of MRI scans from eight boys with SLI age 4;2 to 9;6.

1.6.2 Results

Six of the eight children showed deviation from usual left greater than right. Three out of these six showed symmetry of left and right perisylvian area due to unusually large right perisylvian area. The other three had the right perisylvian area larger than the left. Further, these studies were extended to parents. Seven of the eight parents studied, showed atypical configuration. This finding was consistent with the genetic aspect of SLI.

1.6.3 Remarks

The studies seems to establish that a larger than usual right perisylvian area constitutes a condition which disfavours language learning. However, there have been cases with atypical configuration with no language problems. Thus we can consider the above mentioned statement to be a bit too strong. Moreover, nearly one-fourth of the brains studied failed to show the normal configuration of larger left perisylvian area. This makes it controversial to classify symmetry as an abnormal condition. Thus it is debatable whether unusually large right perisylvian area is the biological reason for SLI.

2 Aphasia

Aphasia as defined by the Oxford Concise Medical Dictionary is a disorder of language affecting the generation of speech and it's understanding and not simply a disorder of articulation. It is caused by disease in the left half of the brain (the dominant hemisphere). It is commonly accompanied by difficulties in reading and writing. The Webster's Revised Unabridged Dictionary says that aphasia is the loss of the power of speech, or of the appropriate use of words, the vocal organs remaining intact, and the intelligence being preserved. It is dependent on injury or disease of the brain. As per the National Aphasia Association (NAA), it is an impairment of the ability to use or comprehend words, usually acquired as a result of a stroke or other brain injury.

2.1 Defining Aphasia: Positions, theories and their proponents

Aphasia has been studied for a long time now and diferent researchers have come up with their own theories to explain it. Here we briefly look at the history of the developments that have taken place:

Propositional	Jackson	1879	Impairment in ones ability to make propositional statements
Gestalt	Goldstein	1948	Inability to adopt an abstract attitude
Unidimensional	Schuell, Jenkins, Jimenez-Pabon	1964	General language impairment that crosses all language modalities
Multimodal	Head	1926	Disturbances of symbolic formulation and expression
Cybernetic	Porch	1994	A reduced capacity to store, switch and monitor and to do many other steps necessary for the brain to process information
Cognitive	Davis	1993	An acquired impairment of the cognitive system for comprehending and formulating language, leaving other cognitive capacities relatively intact
Psycholinguistic	Chapey	1994	An acquired impairment in language and cognitive processes that underlie language and is caused by organic damage to brain; characterized by reduction in and dysfunction of language content, form and use and the cognitive processes that underlie language such as understanding memory or thinking

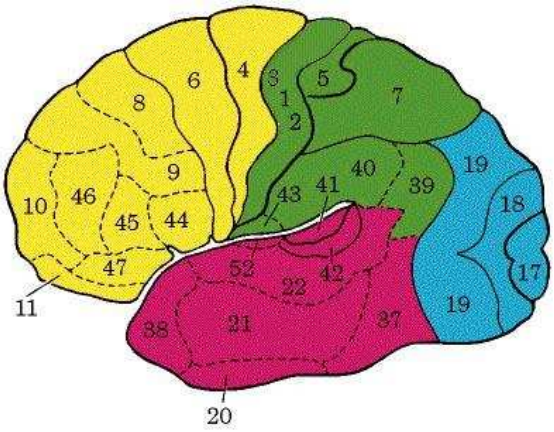

Figure 2: Brodmann's division of brain into areas

2.1.1 Aphasia: Syndrome Approach

The definition of aphasia which comes closet to the actual syndrome was given by Goodglass and Kaplan in 1981. According to them it is a neurological disorder resulting from damage to those regions of the cerebral hemispheres that form the anatomical basis for the human capacity for language. In 2001, the definition was revised - Aphasia refers to the disturbance of any or all of the skills, associations and habits of spoken and written language produced by injury to certain brain areas that are specialized for these functions. Disturbances in communication that are due to paralysis or incoordination of the musculature of speech or writing or to impaired vision or hearing are not, of themselves, aphasic.

2.2 Clinical Syndromes of Aphasia

We need to associate clusters of signs or syndromes with anatomy of the lesions in the brain producing them. Various classification schemes have come up because the lesions vary in exact size and location and response of individuals to the same lesions may also be different. As it is seen, children seem to show different responses to the same lesions. There are two emergent views on classification:

- Localizationists: Every type of linguistic behaviour can be localized in a particular part of the brain.
- Antilocalizationists: The brain is an integrated unit like a hologram and damage to one part will affect the functioning of a brain as a whole.

Our study in this paper would be in harmony with localization. On the basis of characteristics of speech output (results of the Boston Aphasia Diaganostic Examination), aphasias can also be broadly divided into:

1. Fluent aphasias: The flow of speech is not hampered, articulation is easy but the speech is not meaningful. There is difficulty in finding words and comprehension.
2. Non-fluent aphasias: The flow of speech is impaired (interrupted and awkwardly articulated) but meaningful. Comprehension appears to be better than production.

2.3 Further classification

Aphasias can be further divided into various types depending upon what signs the patients show. There can be reduced language output as well as reduced comprehension, repetition and naming. Apart from spoken language impairments, we also find additional impairments in reading and writing. If we consider naming impairments to be common to all aphasias, then we can have eight different syndromes. Again in thie classification, there are problems like: 1. These signs may vary across individuals, 2. Syndromes are not

stable even though the anatomy of lesions is, and 3. Most syndromes are polytypic that is they are defined by several criteria.
Here is a list of the different kinds of aphasia classified into various categories:

- Non-fluent:
  1. Broca's Aphasia
  2. Chronic Broca's Aphasia
  3. Acute Broca's Aphasia
  4. Transcortical Motor Aphasia
- Fluent:
  5. Wernicke's Aphasia
  6. Anomic Aphasia
  7. Conduction Aphasia
  8. Transcortical Sensory Aphasia
- Others:
  9. Global Aphasia
  10. Mixed Transcortical Aphasia
- Other Aphasias:
  11. Crossed Aphasia
  12. Aphasia in Left Handers
  14. Mixed Non-fluent Aphasia
  15. Subcortical Aphasia

2.4 Description of different types of aphasias

2.4.1 Global Aphasia

It is the most severe of all aphasias as there is significant impairment in all aspects of language. Language output is severely limited and comprehension is very impaired. There is absolutely no repetition, naming or writing. Along with global aphasia, buccofacial and limb apraxia are common, right hemiplegia may also occur.

Most typical lesion undercuts the entire perisylvian region. But much clinical variability is seen ranging from frontal lesions to deep subcortical temporal lesions. Prognosis is poor but eventually patients may improve comprehension and qualify for severe Broca's aphasia.

2.4.2 Broca's Aphasia

This is one of the most common of non-fluent aphasias. Language output is non-fluent, articulation is poor, and volume and speech quality are also reduced. Speech is telegraphic: the sentences are short (2-3 words) and grammatically simple (we generally find only noun-verb combinations). Relational words (articles, conjuctions, modifiers) are very uncommon in speech. Repetition is poor, literal paraphasia may occur and word finding is poor. Apraxia, right hemiparesis, depression, low frustration tolerance genrally accompany Broca's aphasia.

Brocas area can thus be related to articulation of speech (either engineering or producing articulation). Let us look at examples of children suffering from this order. It is seen that patients often do not hear their own verbalized conversation:

"What is your name?"
"Litha.
"Litha?
"No, Litha!"

It is also seen that children strongly resist grammatical correction by third party which is evident from

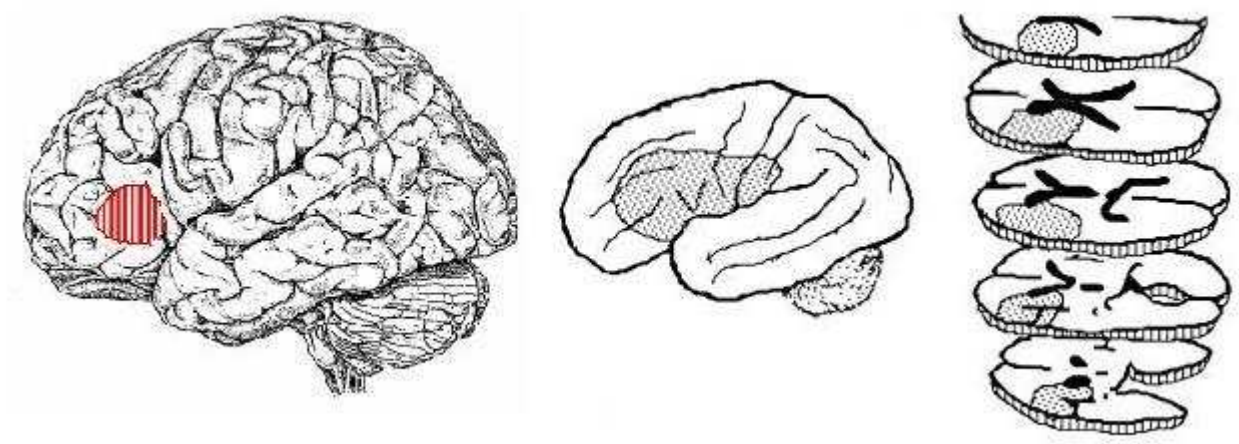

Figure 3: Lesion in Broca's Aphasia (left) and Chronic Broca's Aphasia(right)

this talk:

Child: "Want other one spoon, Daddy."
Father: "You mean, you want the other spoon."
Child: "Yes, I want other one spoon, please Daddy."
Father: "Can you say, 'the other spoon'?"
Child: "Otheronespoon." Father: "Say other.
Child: "Other" Father: "Spoon."
Child: "Spoon."
Father: "Other spoon."
Child: "Otherspoon. Now give me other one spoon."

Chronic Broca's Aphasia A study of fractional cases of Broca's aphasia might prove to be helpful. Chronic Broca's Aphasia often emerges out of global aphasia. There is damage to the dorsolateral frontal, rolandic, operculur and anterolateral parietal regions. Critical to this aphasia is the subcortical extension of the lesion.

Acute Brocas Aphasia It involves lesions in frontal operculum, lower motor cortex and subcortical white matter. Even after recovery, patients have paraphasias, speech impairment and impaired repetition.

Broca's Area Lesion The areas which can be associated with Broca's Aphasia are frontal operculum (Brodmann Areas 44, 46) [1] and Dorsolateral frontal cortex (Areas 44, 46, 6, 9). This suggests the existence of a frontal-caudate regional network required for complex output procedures: syntax and narrative discourse at the minimum. Damage to the lower motor cortex suggests the existence of a local (rolandic) network for articulation and some aspects of prosody (Figure 03). All these fractional and variant cases of Brocas aphasia show recovery sometimes by reorganizing cerebral functions to allow right brain control of speech.

2.4.3 Transcortical Motor Aphasia

Language output is non-fluent: there is an initiation block, reduction in phrase length and simplification of grammatical forms. Patients of TCMA are initially mute and may remain mute for many days. Echolalia (particularly incorporation echolalia - including a part of the question in the answer) is observed. Repetition, articulation and oral reading are normal.

The classical patient has a large dorsolateral frontal lesion extending deep into the white matter. Connections between the Broca's area and Brodmann Areas 6, pre-motor area or basal ganglia are severed. The fundamental deficits in TCMA are:

[1] Brodmann divided the entire brain into a number of areas. Look at Figure 02.

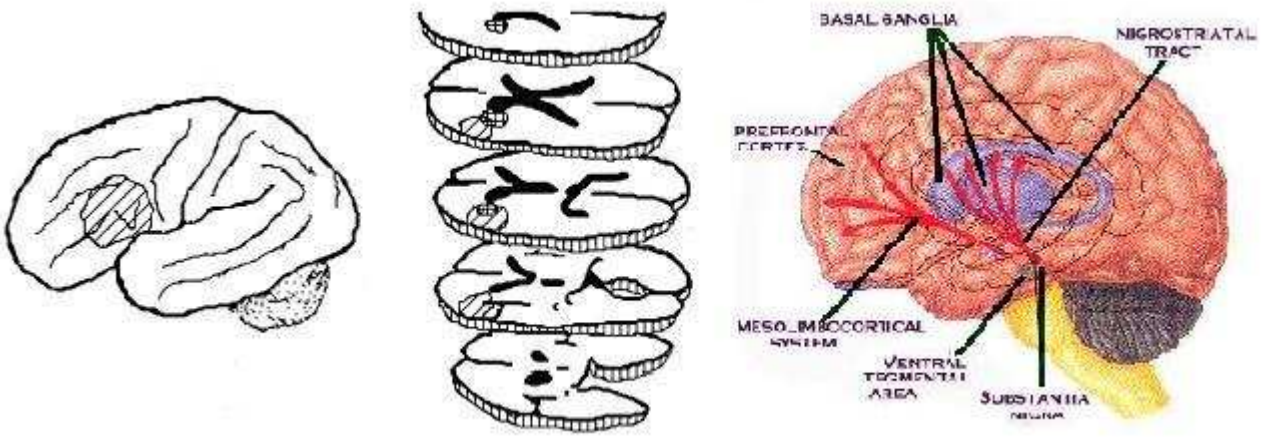

Figure 4: Lesions in case of Transcortical Motor Aphasia (left) and Dopaminergic Pathways (right)

Generative language tasks The capacity to generate complex syntax is limited. Patients cannot respond to open ended questions. This is attributed to large dorsolateral frontal lesions.

Reduced activation There is also a reduction in activation to speak (or write) which points towards medial frontal damage. Reduced activation is due to loss of ascending dopaminergic pathways (Figure 04) supported by improvement in fluency and speech rate after administration of direct dopamine agonists.

2.4.4 Wernicke's Aphasia

It is the most common of the fluent aphasias. Language output is fluent. The major impairment is semantic - content seems to be meaningless (often referred to as jabberwocky) or empty. An example is:
"The stockety wance on my holiday, it ate up the laddersby until, you know, we fell it over and then he danced wither the meal."
Paraphasias are common. They can be verbal: cup or knife for spoon, literal or phonemic: smoon for spoon, or neologistic: snopel. Speech is paragrammatical because of semantic ambiguity. Repetition and naming are poor. Anosognosia (lack of awareness) of their communication problems is common.

Wernicke's Area Lesion The lesion is generally in the area of the superior temporal gyrus to the end of the sylvian fissure (Figure 05). Auditory comprehension is severely impaired and so auditory language system must be temporal. Key regions for word retrieval are inferior temporal and middle temporal/ angular gyrus transition. Lexical- semantic function is broadly distributed in the posterior association cortex. PET studies show that recovery of comprehension is proportional to the recovery of blood flow in the left hemisphere. Recovery is also related to shift in activation to semantic tasks from left temporal to right temporal region. This shows the importance of posterior association cortex for recovery of comprehension.

2.4.5 Anomic Aphasia

It is a much less homogeneous grouping than other classical syndromes. Naming or word retrieval is the only deficit. Circumlocution is common. For example, a patient saying "had one of them up there" would mean that he has had a surgery in the head. Comprehension and repetition are good and paraphasias are rare.

It can be localized with the least reliability of all syndromes. When caused by the temporal parietal area there may be alexia and agraphia. When caused by dorsolateral frontal lesions, there are no accompanying signs. When it is the residual of Brocas aphasia, accompanying signs are as expected of those disorders.

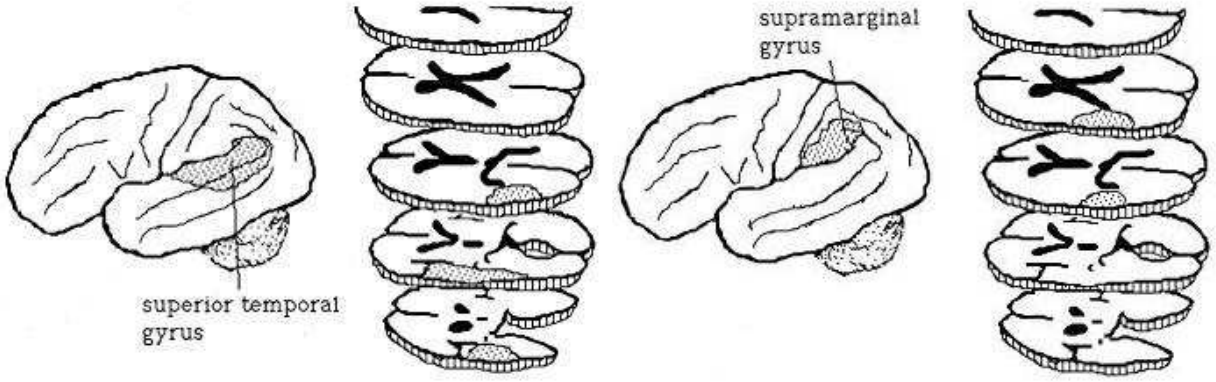

Figure 5: Lesions in case of Wernicke's Aphasia (left) and Conduction Aphasia (right)

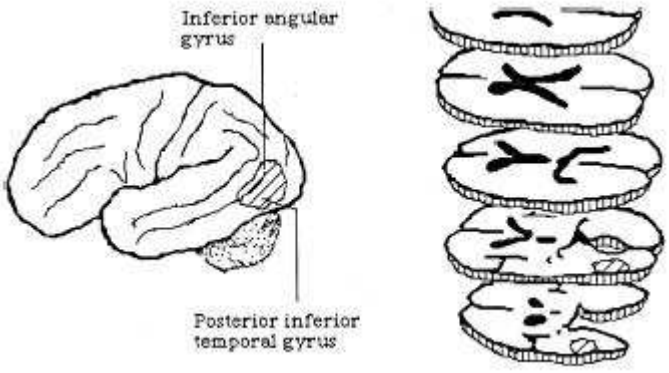

Figure 6: Lesions in case of Transcortical Sensory Aphasia

2.4.6 Conduction Aphasia

Language output is fluent. Content is paraphasic usually literal/ phonemic. The patient attempts to produce repeated approximations of the word, which is called conduit d'approche. Repetition is poor but auditory comprehension is normal.

Generally there is damage to supramarginal gyrus (Figure 05). Classical correlation was with the arcuate fasciculus, a bundle of nerve fibers that lies below the supramarginal gyrus in the temporal lobe and connects Broca's and Wernicke's areas. This highlights that supramarginal gyrus and temporoparietal short association pathways are critical areas for phonological processing.

2.4.7 Transcortical Sensory Aphasia

Repetition is preserved but there is no comprehension and propositional speech. Semantic processing is affected and hence semantic paraphasia dominates.

It occurs when Broca's area, Wernicke's area and the arcuate fasciculus are undamaged but are cut off from the rest of the brain by infarcted tissue (Figure 06). Lesions are in the middle and inferior temporal gyri. Such lesions would be found in Brodmann Areas 37, 22, and 39.

2.4.8 Mixed Transcortical Aphasia

Comprehension is impaired and naming is poor. Repetition is preserved. Echolalia and fragmentary sentence starters are common. MTA requires a combination of the lesions of TCMA and TCSA. Patients are mute initially and when they speak it is like patients with TCSA. Most cases are due to large frontal lesions in

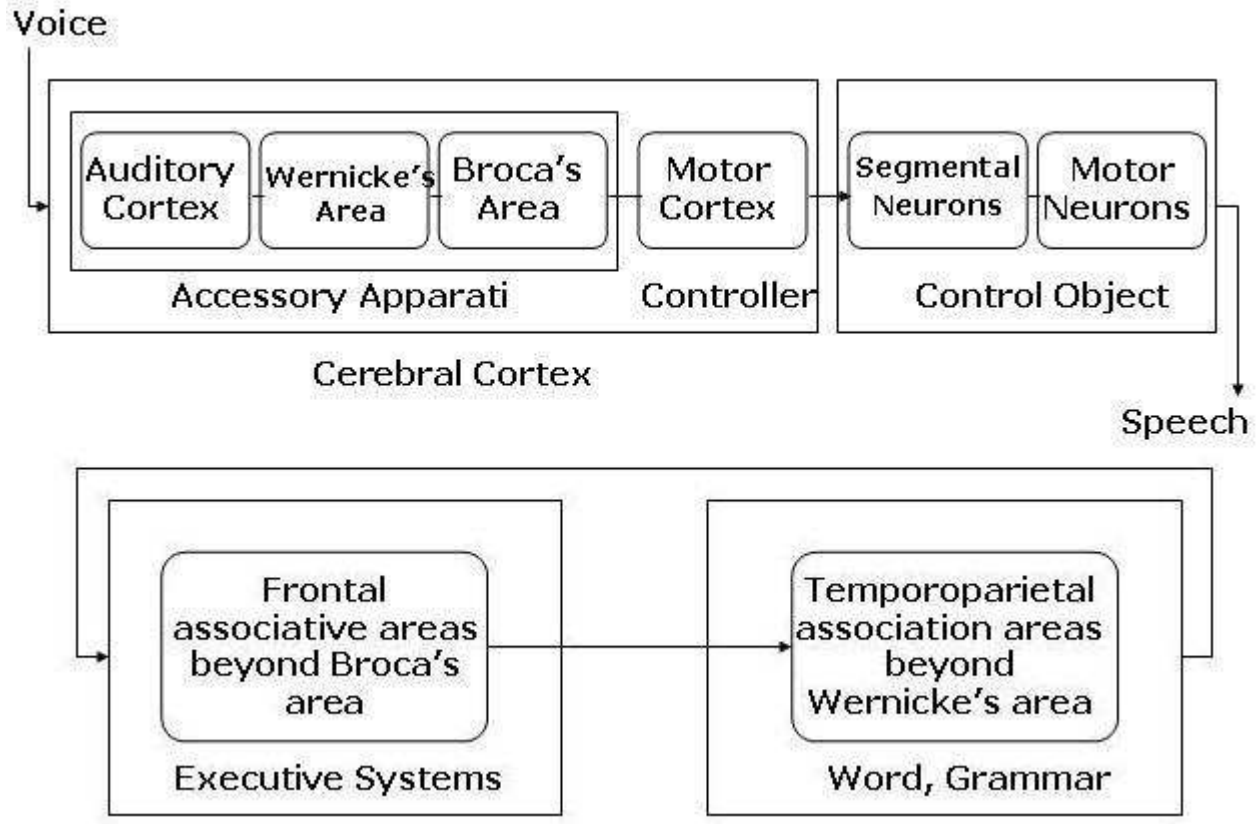

Figure 7: Models for Hearing and Speech (left) and Verbal Thought (right)

the region of TCMA lesions.

2.4.9 Crossed Aphasia

The review which we have done is valid for right-handers with lesions of the left hemisphere. About 2 to 5 % of the right-handed population (though it can range from 1 to 13 %) becomes aphasic after a lesion in the right hemisphere. These patients fall into two categories: About 70% have standard aphasias associated with corresponding lesions in the left hemisphere. The rest 30% have striking anomalies in the aphasia-lesion relationship. In this group, mild syndromes can occur despite large lesions.

Conduction aphasia has been seen despite large perisylvian lesions. In other patients with similar lesions, transcortical sensory aphasia or anomic aphasia have been described. Patients with crossed aphasia generally have a better capacity for recovery. These anomalic cases may suggest possible lateralizations of phonologic and semantic functions. Alexander and co-workers even propose a genetic basis for inheritance of handedness and laterality. The biological basis of crossed aphasia however remains unknown.

2.4.10 Aphasia in left-handers

Left-handers make up 10% of the total population but are a much more heterogeneous group. Again, about 70% of left-handed aphasics have left-brain lesions and 30% have right-brain lesions. About 15% would be aphasic after a lesion of either hemisphere (have bi-lateral language representation). Proportion of cases with anomalic aphasia-lesion relationships is higher than in right-handers. It is also claimed that they have better recovery than right- handers. Since the biological basis of handedness and lateralization is unknown, it is an open question as to how do these anomalies occur.

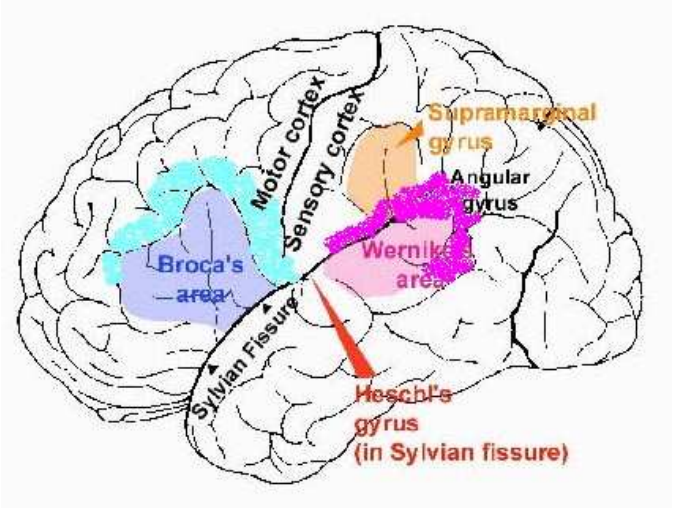

Figure 8: Areas of Brain and their language associations

2.5 Classic Model of Language Organization

The classical model for language organization (Figure 07) has been proposed by Alec Marantz et al. It is analogous to that for voluntary limb movements. The cerebral cortex is the controller while segmental and motor neurons (in medulla, cervical cord, orofacial and laryngeal muscles) are the control object. The command is sent to the control object and generated by the activities in auditory cortex, Wernicke's and Broca's areas. Neural system for verbal thought or comprehension is similar to association cortex functions for thought processes in general. Frontal association cortex is the controller while temporoparietal association cortex contains the control objects (words, the conceptual tree etc. called the "thought models").

2.6 Conclusion

On the basis of the lesions in different cases of aphasias, we can come up with some generalizations about brain-language associations. They have been tabulated in the table below:

1	Supramarginal gyrus, temporoparietal short association pathways	phonological procesing
2	Broca's Area	articulation of speech
3	Local rolandic network	articulation and some aspects of prosody
4	inferior temporal region, middle temporal/angular gyrus transition	word retrieval
5	Frontal-caudate regional network Dorsolateral frontal areas	syntax and narrative procedures
6	posterior association cortex	lexical-semantic function
7	Temporal regions	auditory language system
8	Medial frontal region	activation to speak and write

ACKNOWLEDGMENT

We would like to thank Prof. Harish Karnick and Prof. Achla Misri Raina, our course instructors for guiding us throughout the study of the topic. Prof. Karnick also helped us by providing the links where we began our work. He also suggested us about books on aphasia.

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