Pakistan Journal of Neurological Sciences (PJNS)

Volume 9 | Issue 4 Article 13

12-2014

A clinical approach to disorders of speech

Apoorva Pauranik
Indore, India, apauranik@gmail.com

A CLINICAL APPROACH TO DISORDERS OF SPEECH

Apoorva Pauranik
Indore, India

Correspondence to: *Dr Apoorva Pauranik, House number 4, Ahilya Puri, Zoo Road. Near Residency Club. Indore. MP, India. Phone: + 91 9926634520. Email. apauranik@gmail.com*
Date of submission: *June 10, 2014,* ***Date of revision:*** *August 19, 2014,* ***Date of acceptance:*** *August 25, 2014*

ABSTRACT

Disorders of speech and communication, including Aphasia (mainly post stroke) are a major community health burden in terms of DALY (Disability Adjusted Life Years). Unfortunately they are relatively neglected with huge treatment gap. Traditionally aphasia related lesion sites have been grouped with reference to Rolandic & Sylvian fissures. However the theoretical models of language and speech organization in brain have undergone major conceptual shifts due to advances in cognitive neuropsychology, linguistics and functional neuro-imaging. Much more widely distributed overlapping neural networks are understood to be actively involved in various communication activities. The notion of speech centers or areas is somewhat outdated. Traditional aphasia syndromes may however be somewhat useful as a shorthand summary of the clinical profile, but are being considered now as of limited utility for planning an intensive, long-term speech therapy. Conventional description of aphasic deficits needs to be replaced by more detailed profiling which helps in better planning for speech therapy. The evidence base for efficacy of speech therapy is now robust. Management of aphasia, though time consuming, is worth the effort, in collaboration with speech therapist. Pharmacotherapy and transcortical magnetic stimulation or transcortical direct current stimulation may also have some role.

INTRODUCTION

Speech, communication and language disorders are many and heterogeneous. (Table 1) Aphasia is one of them and involves a deficit in expression (motor), comprehension (sensory) or both. The modes of communication affected could be verbal, written or both and even the unusual ones such as sign language in deaf-mutes. At the core of aphasia lies a disturbance in language functions. Language is an instinct whose biological substrate is also conducive to other learned traits like reading, writing and probably mathematics and music. Community burden of disorders of speech is significant when measured as Disability Adjusted Years of Life (DALY's). Aphasia persists as disability in 21-38% of stroke survivors. Community incidence is 43/100,000/year, and prevalence is 3000 per million (1). It is commoner than many neurological diseases like Parkinsonism, multiple sclerosis, motor neuron disease and muscular dystrophy combined together. Dementia, Head injury, brain tumors, encephalitis are additional causes of aphasia. Language learning disabilities including dyslexia (prevalence 5%) are a major community problem whose assessment and therapeutic rehabilitation is guided by cognitive neuropsychological principles, similar to those applicable to acquired aphasia and alexia. (Table 2) Non-fluent aphasia predominates in young patients suggesting an anterior lesion, while in elderly patients fluent aphasia is attributed to more frequent posterior infarct [(2)]. Clinical deficits in speech and communication are being increasingly recognized and studied in various types of dementias, motor neuron disease, extrapyramidal diseases, multiple sclerosis and others. While aphasia profiles in stroke reflect arterial topography, those in neurodegeneration reflect functional connections which are widely distributed in a non-random fashion. In Alzheimer's disease memory loss and general cognitive deterioration precedes language dysfunction. Naming and vocabulary becomes poorer in content. Repetition, oral reading, auditory comprehension and grammar are however preserved till late. The aphasia profile initially looks like anomic, later transcortical sensory and Wernicke, finally degrading into severely non-fluent output or muteness. Fronto-temporal degeneration is the second most common form of early onset dementia. Three subtypes of Primary Progressive Aphasias affect language & communication for initial 2 years or more, without other cognitive derangements. They can be differentiated on the basis of fluency, comprehension and repetition.

Anatomical Substrate of Aphasia and Neuroimaging

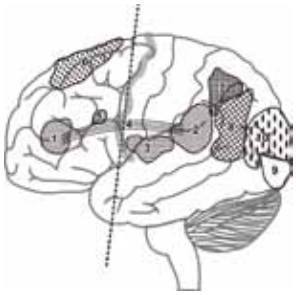
The anatomical substrate of aphasia had been conceptualized along three axis of dichotomy in relation to clinical picture (Fig.-1). The left hemisphere controls speech and language functions in 98% of right handed persons and also in about 50% of the non-right handed persons. Aphasia occurring in a right-handed person with a lesion in the right cerebral hemisphere (crossed aphasia) occurs only in 2-3%. The association cortex in the region of the left Sylvian fissure is mainly responsible for language capability. Many subcortical structures

(posterior thalamus, basal ganglia, deep white matter, temporal isthmus, insula) mainly on left or dominant side, are an important and integral component of language circuits. Aphasic syndromes due to discrete subcortical lesions are well described but they are generally less precise, not fitting into well established syndromes and usually recovers well in short period. The clinical deficits are more often due to associated reversible cortical hypo-perfusion or deactivation.

Figure- 1: Anatomical substrate of Aphasia

Dichotomy : A - Central Sulcus/ Rolandic Sulcus

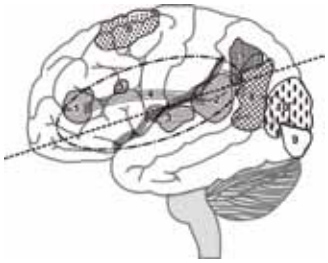
Anterior lesions Pre rolandic	Posterior lesions Retro Rolandic
Non-fluent, dysarthric, + Comprehension Normal Eg : Broca, Transcortical Motor.	Fluent : Non dysarthric Comprehension abnormal Eg. : Wernicke, Transcortical sensory.



1. Broca's Area, 2. Wernicke Area 3. Primary auditory cortex 4. Arcuate fasiculus. 5. Primary motor area : mouth and tongue 6. Motor association cortex and supplementary motor area 7. Supra marginal gyrus. 8. Angular gyrus 9 . Primary visual cortex 10. Visual association cortex.

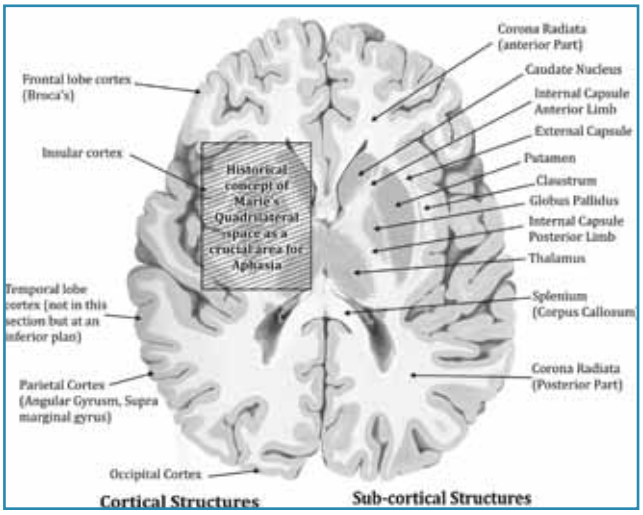
Dichotomy : B - Lateral Sulcus/ Sylvian Fissure

Peri-sylvian	Extra-sylvian
Repetition is poor Broca, Wernicke, Conduction	Repetition is preserved Transcortical aphasias, Anomic aphasia. Alexia without agraphia



1. Broca's Area, 2. Wernicke Area 3. Primary auditory cortex 4. Arcuate fasiculus. 5. Primary motor area : mouth and tongue 6. Motor association cortex and supplementary motor area 7. Supra marginal gyrus. 8. Angular gyrus 9 . Primary visual cortex 10. Visual association cortex.

Dichotomy : C – Cortical Grey matter & Sub Cortical Grey and white matter



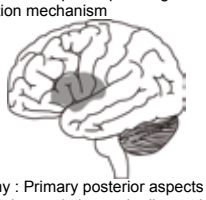
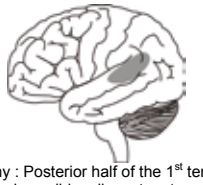
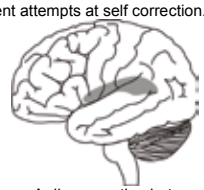
Clinicoanatomical correlations between lesions depicted on imaging and the aphasia profile are along expected lines in majority of the patients. However, about 30% cases exhibit misfits due to inter-individual variations in the anatomical substrate for language functions in brain. Factors such as handedness, dominance, age, sex, literacy and multilingualism which are demographically somewhat different in south asians as compared western. White population, may play a role in the degree of bilateral representation and other anomalous organization of speech function. Classical aphasiology was built over a period of one and half century upon evidence from postmortem brain lesion studies and their correlation with antemortem clinical picture.(Fig.-2) Modern neuroimaging has shown that in addition to the so-called classical speech areas, other more distributed bilateral brain regions play significant role in language processing. During functional neuroimaging for speech and communication specific linguistic tasks are performed by subjects while imaging study results are compared before, during and after the task. These regions also contribute to non-language higher mental function. It looks as if language emerged phylogenetically from pre-existing processing mechanism, initially meant for other, more basic cognitive functions. Functional neuroimaging has also shown that the dichotomy between motor or expressive or production on one hand and sensory or receptive or comprehension on the other, is rather artificial [3]. The apparent distinction between anterior-versus posterior brain region is blurred by studies showing simultaneous equal activation of brain regions across the central sulcus in different language tasks of production and comprehension or lower level speech sounds versus higher level word meanings.

Clinical Approach To Aphasia

Enquire about level of literacy and language(s) used at home and work. The handedness should be assessed by enquiring about the preferred use of a hand for many activities of daily-life in addition to eating and writing. Associated neurological deficits like hemiperesis, facial paresis, visual field defects, hemisensory loss and amnesia should be documented. A brief examination of speech can be completed in 5-10 minutes and will provide basic data about presence of aphasia, its severity and a tentative diagnosis of aphasia syndrome. More detailed assessments by speech therapist are required for comprehensive profiling and planning the rehabilitative speech therapy. The algorithm in figure 4 is useful but may be an oversimplification. Assessment of speech and language functions should involve multidirectional approaches. Traditional clinical neurological examination includes determining over all severity, fluency, articulation, information content, word finding difficulty, auditory comprehension at single word, sentence and story level, repetition, naming on confrontation, writing to dictation, copying and narrative writing. Cognitive psychologists are interested in probing

various theoretical issues like information processing, intelligence and computation. Hence they approach language function in different manner by examining functions like attention, motivation, perception, speed of processing, categorization, neural network theories, short term memory, implicit versus explicit memory etc. Linguists are interested in studying the effects of pathology at various linguistic levels like phonetic, phonological, morpho-syntactic, semantic, pragmatic and discourse levels. Speech therapists who are focused on rehabilitation, pay attention to overall communicative abilities and deficits. They welcome detailed input about specific strengths and weaknesses in a given individual at a particular time in course of illness so as to plan re-educative strategies and programme in a more efficient manner. Aphasia is not an exclusively language related problem. Many other higher mental functions are also affected: working memory, short term memory, recent memory, attention, executive skills, sensory perception, motor dexterity, apraxia, agnosia, spatial orientation. If a patient has problems in holding items in working memory he will have problems in sentence processing. Attention is essential to focus on communication partner's speech, particularly when surrounded by competing stimuli. Vigilance is attention sustained over long periods of time and is critical to hold a conversation.

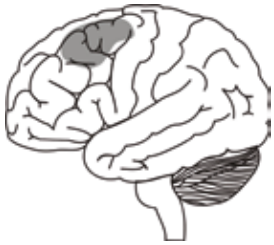
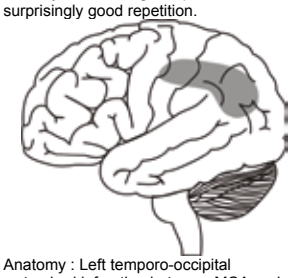
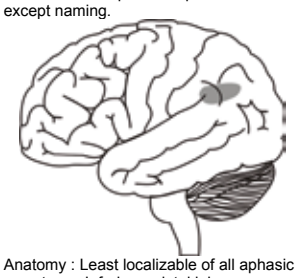
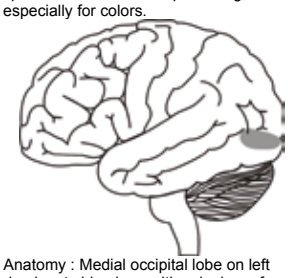
Figure 2a : Aphasia with perisylvian lesions

Broca's Aphasia : Clinical : Sparse, halting speech, often misarticulated, frequently missing function words and bound morphemes. Disturbance in speech planning and production mechanism  Anatomy : Primary posterior aspects of the 3rd frontal convolution and adjacent inferior aspects of the precentral gyrus. Prerolandic branch of middle cerebral artery. A lesion restricted to classical Broca's area causes a mild reversible nonfluent aphasia. More severe and well known type of classical broca's aphasia requires a much larger lesion surrounding regions other than Broca's area.	Wernicke's Aphasia : Clinical : Poor reading and auditory comprehension, fluent speech with phonemic, morphological and semantic paraphasias. Disturbance of permanent representations of the sound structures of words.  Anatomy : Posterior half of the 1st temporal gyrus and possibly adjacent cortex. Posterior temporal branch of left middle cerebral artery.
Global Aphasia : Clinical : Disruption of all language processing components and severe diminution or loss of all language .  Anatomy : Large portion of the perisylvian association cortex. Combined lesions involving Broca's and wernicke's area. Infarction due to occlusion of stem of left MCA or internal carotid artery.	Conduction Aphasia : Clinical : Repetition is severely impaired out of proportion to verbal expression, fluency and comprehension, literal paraphasic errors are common with frequent attempts at self correction.  Anatomy : A disconnection between wernicke's and broca's area due to arcuate fasiculus, a white matter tract in deep temporal lobe. Also due to lesion in superior temporal or inferior parietal region.

Classification and Syndromes

Syndromic classification has been useful as a short hand summary of patient's language disorder. However, they have lost their theoretical relevance and they need to be replaced by newer classifications based on cognitive neuropsychology [4].

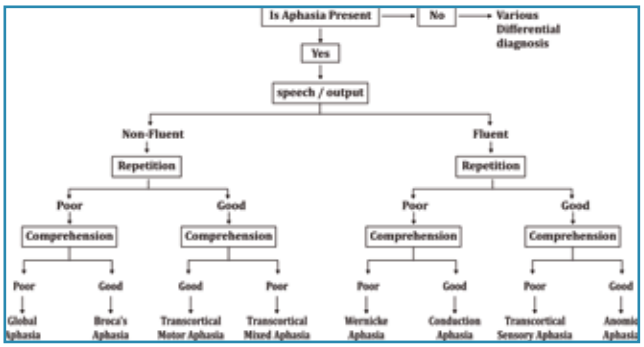
Figure 2b: Aphasias with lesions peripheral to perisylvian region

Transcortical motor : Clinical : Similar to Broca's aphasia with hesitant, telegraphic speech, normal comprehension and surprisingly good repetition.  Anatomy : Deep white matter in frontal lobe, anterior and superior to Broca's area or near supplementary motor area. Anterior cerebral artery territory.	Transcortical Sensory : Clinical : Similar to Wernicke's aphasia with fluent paraphasic speech, poor auditory and reading comprehension but surprisingly good repetition.  Anatomy : Left temporo-occipital watershed infarction between MCA and PCA territories.
Anomic : Clinical : Disturbance of the concepts of the words or the sound patterns of words or both. Most aspects of speech normal except naming.  Anatomy : Least localizable of all aphasic symptoms. Inferior parietal lobe or connection between parietal lobe and temporal lobe. Arterial territory : Angular branch of left MCA	Alexia without Agraphia: Clinical : Patient can write but cannot read even their own writing. Other aspects of speech are normal except naming, especially for colors.  Anatomy : Medial occipital lobe on left dominant side along with splenium of corpus callosum. Within posterior cerebral artery territory. It is a disconnection between intact right visual cortex and left

Traditional methods of assessment of aphasia may still be valid and useful for localization and prognosis but major conceptual shift has occurred since 1980s in the way the patients are studied. The reference model is no longer the anatomy but the functional components of normal cognitive system. Many testing protocols straddling across and beyond the traditional aphasia syndrome and based on cognitive analysis have been developed [5]. Additional methods of clinical examination relating to different components of cognitive neuropsychological model of language in brain (figure 4) have been developed in last few decades. (Table 3) gives some examples of errors based on such a model and their localizing value. An analogy can be given with epilepsy. The classification of seizures has been revised. More emphasis is on seizure seimiology (actual description of the whole event as seen or recorded on video) rather than pigeon-holing subjects into categories like

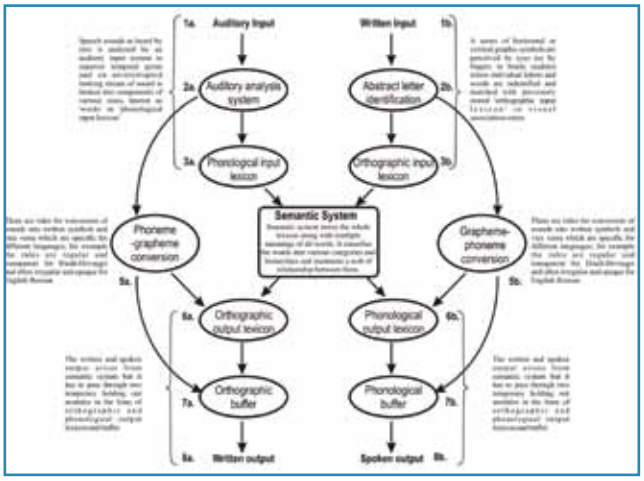
partial simple, partial complex etc. Something similar to video monitoring is happening in aphasiology. When one argues that the classical Aphasia syndromes are outdated, that does not necessarily mean that some perfect or better alternative is available. The subject is passing through a phase of transition. We must go for detailed description instead of mere labeling or else and use labels only as a summary title stem and expand upon them.

Figure 3: Algorithm : Aphasia Syndrome Diagnosis



This algorithm is an oversimplification. The so-called classical aphasia syndromes are rarely true or pure to themselves. Overlaps or mixed profiles are more common. If at all, we use the syndrome label only as a short hand summary. Always make it a habit to describe the salient clinical features in the terms mentioned in table

Figure 4: Cognitive Neuropsychological Model of Lexicon (The vocabulary or store house of words)



It is possible to examine in detail the linguistic performance of a subject and pinpoint the boxes or arrows in the diagram, where one thinks that the functional deficit is present. The tasks are simple and not too lengthy. They ingeniously make use of reading aloud and writing to dictation words and nonwords, regular and irregular words and exception words.

Differential Diagnosis of Aphasia

Aphasia is an acquired disorder in a person who had attained certain degree of speech development. Hence developmental disorders of speech are not aphasia. Many children with brain damage (antenatal, perinatal, postnatal) and mental retardation learn speaking, reading and related skills late or incompletely or not at all. They are not aphasic. A developmental suppression of the motor fluency of speech is very common. Stuttering or stammering is not a part of the aphasia rubric. Early damage to the function of hearing influences the development of speech and an extreme example is congenital deaf-mutism. Pure dysarthria hampers the pronunciation and intelligibility of speech but language, vocabulary, grammar, reading, writing, and auditory comprehension are intact. In severe cases, hardly any sound emanates from the articulatory apparatus and results in anarthria. Disorders of phonation occur due to diseases of the larynx, vocal cord and respiratory weakness (no enough air during expiration). In all the above conditions, the cortical areas concerned with cerebral organization of linguistic functions are intact. Written expression (if the patient is literate) is normal while in aphasia written and verbal deficits almost always run parallel to each other. Auditory and/or reading comprehension are also normal while in patients with aphasia these are often abnormal. Regression of normal acquired speech and communicative ability, and other cognitive functions occur in a group of pervasive disorders in children such as autism, Asperger syndrome and Rett syndrome. Landau-Kleifner syndrome in children is also a disorder of acquired aphasia and epilepsy. Patients with conversion reaction may become mute, speak in whispers or in a bizarre, abnormal manner. Rarely, some patients may behave as if they are not able to listen or comprehend any speech. This should raise the possibility of malingering or Munchausen syndrome. Speech in psychosis, particularly schizophrenia is sometimes confused with aphasia. Verbal or written output may be sparse or excessive, bizarre and absurd. Responses may sound like jargon or other less severe distortions described in oral expression. Flight of ideas, pressure of speech and evasive, tangential answers may create an impression of logorroheic speech or Wernicke aphasia with defects in auditory comprehension. Howsoever absurd the thoughts and speech in a patient with schizophrenia, grammar and logic are largely retained, which is not the case in those with aphasia. One of the important prerequisites for a definition of aphasia is a state of normal alertness or sensorium and normal intellectual or cognitive functions. It does not mean that the two cannot coexist but in such a situation it becomes difficult to decide about the relative contribution of each of them to the clinical deficit. Patients who are stuporose, drowsy or

have an acute confusional state or delirium may be labeled as aphasic due to delayed and incorrect responses. Irrelevant muttering may be mistaken as fluent jargon or vice versa. Aphasic patients with defective auditory comprehension may be wrongly considered as having hearing impairment. Loss of the naming function in aphasia is commonly mistaken as loss of memory. The contrary may also be true. Patients with amnesia, dementia and Korasokoff psychosis may involuntary fill in the gaps in their memory and be diagnosed as suffering from anomic or Wernicke aphasia. A patient may fail to comply with a verbal command due to motor apraxia and not because of a defect in auditory comprehension. Visual or tactile agnosia may be responsible for a naming defect rather than aphasia. Total loss of speech or 'mutism' is a clinical situation caused by a very wide variety of disorders, aphasia being only one of them. Congenital deaf mutism is easily identified since early childhood. Patients with severe Broca's and global aphasia may be mute for many initials days to weeks. Anarthria & aphonia (laryngeal) may resemble muteness. Frontal lobe dysfunctions are known to cause akinetic mutism. Psychogenic syndromes (conversion or dissociative states, catatonia) can also render a person totally speechless for a variable period of time.

Management, Speech Therapy and Rehabilitation

Therapy requires teamwork involving a neurologist, psychologist, speech therapist, social worker, linguist and the relatives and colleagues of the patient. Various methods of speech therapy can be grouped into two: those reducing specific impairment and those aiming for overall functional social gain. There is no contradiction between the two. In music, one practice scales or ragas not because a performance consists of playing scales, but because doing this develops skills that results in better performance [(6)]. The intensive phase of speech therapy usually lasts a couple of months. The frequency of sessions could be twice a week to twice per day, each session lasting for about an hour. Take-home exercises are also given, which should be carried out under the supervision of family members. It is difficult to evaluate the effectiveness of formal speech therapy because so many factors influence the recovery from aphasia. Yet in recent years, a few studies have supported the utility of speech therapy, as it satisfies the stringent criteria of

Table :1 Disorder of speech language and communication

Type of Disorder	Comment
Disorders of phonation Aphonia Dysphonia	Almost always due to laryngeal dysfunction (local, non neurogenic or neurogenic paralysis of vocal cord muscles)
Disorders of articulation Anarthria Dysarthria	Non neurological local causes : Neurological Causes : Muscles : - polymyositis Neuromuscular : – myasthenia gravis Neuropathic : – GB Syndrome Brain stem, Cerebellum, Basal ganglia CVA, neoplasia, infection, degeneration (Parkinsonism), trauma, developmental.
Disorders of fluency Stammering (Stuttering) Cluttering	Usually developmental No visible underlying pathology in most
Disorder of language Aphasia Alexia/ Dyslexia Agraphia	Cortical speech areas : Stroke Other causes less common causes Developmental : Mental retardation, cerebral palsy Degenerative : Young Age : Autism, Rett syndrome, leuko & polio dystrophies Old age : Alzheimers and other dementias

Table :2 Community Burden of aphasia and other disorders of speech and communication.

S.No.	Disease Condition	Over all burden	Percentage with aphasia
1.	Stroke	Prevalence 0.5%	25%
2.	Head injury	Prevalence 1.2%	5 – 10 %
3.	Dementia	10% above the age of 60 years	Almost all have variable impairment of language functions depending on severity.
4.	Mental retardation	5% (all) 1% (severe)	Almost all have variable impairment of language function depending on severity.
5.	Learning disabilities (eg: dyslexia)	5%	All have variable handicap in reading and writing skills.
6.	Fluency and articulation disorders: Stuttering, stammering lisping, cluttering, lalling	1%	Variable handicap in all.

evidence-based medicine or practice. Training –induced plasticity in the brain during recovery from aphasia has now been documented with the help of positron emission tomography (PET) scan. It shows significant activation of ipsilateral and also non-dominant (right) hemisphere. Technological advances have improved the quality of life of many patients with aphasia. Audiovisual augmentative methods of communication are now possible and affordable. Computers, multimedia, speech synthesizers, touch screens and special software are being used. Melodic intonation therapy and music therapy were reported to be useful for patients with non-fluent aphasia to convey meaning through variations in tone and accent, despite a very limited repertoire of vocabulary. Drug therapy : Randomized controlled trials have been reported with dopamine agonists (brombocriptine), nootropics (piracetam) and cholinesterase inhibitors (donepezil). There is weak evidence in favour of a mild efficacy particularly in nonfluent aphasias. However a few recent studies have shown that pharmacotherapy in combination with intense speech therapy is superior than either of the two alone. Transcranial magnetic stimulation and transcranial direct current stimulation have been reported to be useful in a few case reports on chronic global aphasics. The effects are long lasting. TMS may modulate or suppress counterproductive or inhibitory over activation in the surviving left and right hemispheric neural networks for naming.

The South Asian Context

The burden of aphasia and other disorders of speech and communication is high in developing countries. We have higher proportion of younger patients as compared to developed countries. Only a few clinicians have been working in the field of aphasiology in South Asia. No indigenous test battery in the South Asian languages has been developed, which fulfills the stringent requirements of originality, sound theoretical basis and documented standardization and validity. Some local adaptations of the Boston Diagnostic Aphasia Examination or Western Aphasia Battery are in use. Bi or multilingualism is a rule, rather than exception in South Asia. It probably helps in better recovery and delays cognitive impairment. The language organization in brain may be different from monolinguals. The clinical profile of aphasic deficits, its recovery and response to therapy are variable or different across the languages. Multilingual persons who are fluent and literate in languages as diverse an Urdu, English, Punjabi, Bengali or Hindi are likely to provide fascinating original inputs in understanding the language function in brain. Illiteracy is reducing but still a challenge and opportunity for South Asian researchers to explore differences, if any between speech and communication performance between illiterates and literates [7]. There is a paucity of speech pathologists and therapists in South Asia. Speech therapy is laborious, time-consuming and requires motivation on the part of patient as well as therapist. Positive results may not be forthcoming in a large minority of patients. Physicians should be familiar with the assessment required for aphasia and its therapy. Clinicians will do well if they carry a pocket book of testing cards. The tools of speech assessment can be used for therapy to

some extent. There is great need for aphasia workbooks in the South Asian languages, which can be given to patients for home exercises. Computer-based and web-based programs for diagnostic evaluation and therapeutic practices have to be created in the Indian languages. A lot may be done even with limited resources. Group therapy sessions can be useful in two manner (i) patients with nearly similar profile are offered targeted specific practice sessions (ii) heterogenous groups are offered general purpose learning and practice opportunities (pragmatics based) to improve the overall communication skills. Family members, caregivers and volunteer with short term training can act as bridge between professionals and persons with aphasia by supervising and facilitating home based practices and exercises. Tele-therapy is feasible and not very expensive where in video conferencing may improve compliance by reducing costs and time incurred in travelling to the therapy center. Computer and internet based exercises and practices can be and must be created in Indian languages to fill the gaps. Neurologists should play some role of therapists. With increasing awareness about efficacy of physical therapy and occupation therapy for motor deficits in conditions like hemiplegia or paraplegia, more and more neurologists have developed interest and expertise in rehabilitation and restoration. They take pride in their specialization. They do not look down upon this type of work as something lowly and to be done only by physiotherapists. Similar change in the mindset is warranted with respect to aphasia.

REFERENCES

1. Engelter ST, Gostynski M, Papa S, Frei M, Born C, Adjacic Gross V, et al. Epidemiology of aphasia attributable to first ischemic stroke : incidence, severity, fluency, etiology and thrombolysis. Stroke 2006;37:1379-84
2. Bhatnagar SC, Jain SK, Bihari M, Bansal NK, Pauranik A, Jain DC, et al. Aphasia type and aging in Hindi-speaking stroke patients. Brain Lang 2002; 83:353-61.
3. Lee A, Kannan V, Hillis AE. The contribution of neuroimaging to the study of language and aphasia. Neuropsychol Rev 2006; 16:171-83.
4. Ardila A. A proposed reinterpretation and reclassification of aphasic syndromes. Aphasiology 2010; 24:3, 363-394.
5. Spreen O, Risser AH. Assessment of aphasia, Oxford; Oxford University Press; 2003.
6. Basso A, Aphasia and its therapy. New York Oxford university Press; 2003.
7. Karanth P. Multilingual/multi-literate/ multicultural studies of aphasia – the Rosetta stone of neurolinguistics in the new millennium. Brain Lang 2000; 71:113-15

Table – 3: Error Types and localizing value.

Error Type	Locus of damage	Examples
Semantic	Semantic system Phonological output lexicon	Table – Chair Five – Fifteen
Phonological		
In general	Phonological output buffer Phonological output lexicon	Chidiya(Bird) - Chiriya
If present only during repetition	Phonological input to output conversion mechanism	
If present only during reading	Grapheme to phoneme conversion mechanism	
Morphological	Phonological output lexicon Orthographic output lexicon	Chain (patience) – bechain (impatience) Chhutti (holiday) – Chhuttiyan (holidays) Shiksha (education)-Shaikshanik (Educational) Dikhana (to show) – Dikhava (showmanship)

Conflict of interest: Author declares no conflict of interest.

Funding disclosure: Nil

Author's contribution:
Dr. Apoorva Pauranik: Study concept and design, protocol writing, data collection, data analysis, manuscript writing, manuscript review