APHASIA: SO MUCH CAN BE DONE BY A PHYSICIAN

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KEY POINTS
1. To be aware about epidemiological and humanitarian importance of aphasia
2. Not to miss the diagnosis of aphasia when present
3. Not to make a false positive diagnosis of aphasia, when not present
4. To always do a brief clinical examination to diagnose the severity and syndrome of aphasia.
5. To sometimes do a detailed clinical examination to create a profile of deficits and strengths in speech and communication
6. To do systemic general and neurological examination to assess the overall clinical situation and co-morbidities, as a background for aphasia.
7. To take into consideration psychosocial status of the subject and jointly workout realistic and meaningful goals.
8. To plan appropriate laboratory and imaging investigations.
9. To always refer the patient to speech therapist, seek feedback from him/her and actively interact with him/her.
10. To consider role of pharmacotherapy
11. To have up-to-date knowledge about and faith in the evidence base for efficacy of speech language therapy.
12. To become a bit of speech therapist yourself, while counseling the patient and caregivers.

TO BE AWARE ABOUT EPIDEMIOLOGICAL AND HUMANITARIAN IMPORTANCE OF APHASIA

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 other unusual ones such as sign language in deaf mutes. At the core of aphasia lies a disturbance in language functions.

Each year in the UK there are approximately 20,000 new cases of aphasia and about 100,000 new cases in the United States. In 1990 the National institutes of Health estimated that there was a population of one and a half million Americans living with aphasia (National Institutes of Health, 1990).1 Indian data are not available but can be extrapolated. The figures are particularly impressive when one considers that the aphasia population is larger than the combined populations of individuals with multiple sclerosis, muscular dystrophy, cerebral palsy, and Parkinson’s disease, conditions that have historically had the benefit of well-funded research and services, while aphasia has not.

Post stroke aphasia persists as a long term disability in about 25% of survivors (range 21 to 38%). A community based study from Sweden reported an incidence of 4/100,000 per year.2 Prevalence
is around 300 per million. Majority of persons with aphasia do not receive scientific therapy and rehabilitative aids. The treatment gap, much larger than the one encountered in epilepsy, is no less ironic because so much can be done.

**NOT TO MISS THE DIAGNOSIS OF APHASIA WHEN PRESENT**

A diagnosis of aphasia may be missed when the clinical deficit is mild and slowly evolving as in benign tumors or focal variants of neurodegenerative dementias like Primary Progressive Aphasia and Semantic Dementia. If one is listening carefully while asking history from patient, many observations can be made; pauses, hesitancy, word finding difficulty, need to repeat the question on realizing that patient has not understood it, wrong words and mis-articulations. The caregivers will tell that the patient struggles to get the words out, particularly the exact one he wants, talks in ways which is hard for people to follow, thinks that he is talking perfectly well, when he is not, has trouble in following conversation especially when tired or anxious or when listening long sentences with a background of noise, has trouble in reading comprehension and writing properly.

That the apparent *impairment of hearing* is in fact due to receptive aphasia can be ascertained by the observation that the patient will be able to listen and respond to nonverbal sounds including music. Anomia in subject with aphasia can be differentiated from *amnesia*. Intact memory can be inferred by indirect behavioral observations.

Alexia or impairment of reading comprehension is a common part of aphasia and subjects, may by mistake, seek ophthalmological consultation thinking that something is wrong with their vision. The visual acuity however will be normal for non-linguistic images.

Some patients with Wernicke aphasia speak fluently but the content of speech is meaningless, consisting of jumbled up words, circumlocutions, neologisms, repetitive meaningless function words and flouting of grammar. Such a speech is called jargon and patient are occasionally mislabeled as suffering from abnormal thought, abnormal behavior, psychiatric disorder, including *psychosis*.

**NOT TO MAKE A FALSE POSITIVE DIAGNOSIS OF APHASIA, WHEN NOT PRESENT**

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 anarchrha. 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 logorrheic 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. Patients with amnesia, dementia and Korsakoff 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 and 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.

TO ALWAYS DO A BRIEF CLINICAL EXAMINATION TO DIAGNOSE THE SEVERITY AND SYNDROME OF APHASIA.

One must begin with patient’s mother tongue and other languages, handedness and level of literacy. 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 hemiparesis, facial paresis, visual field defects, hemisensory loss and amnesia should be documented. A brief examination 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 spanning over 1-2 hours in one or more sessions will be required in most subjects for comprehensive profiling and planning the rehabilitative speech therapy.

A. ORAL EXPRESSION, ARTICULATION AND FLUENCY: Make your observations throughout history and try to ask questions which force the patients to utter some sentences. Like ‘tell me about your illness’, ‘your family’, ‘the work done by you at your job’.

An adequate sample of narrative speech is needed to detect the types of deficits. One can use picture description task. Aphasics produce incomplete, halting, sparse description, failing to grasp the totality of the environment despite being repeatedly pointed to the different parts of the picture.

Defective articulation and phonation are recognized during ordinary conversation. Test phrases or attempts at rapid repetition of consonant clusters bring out particu-
lar abnormality. Dysarthria is an important and common deficit in many aphasics particularly those with nonfluent speech. Pure dysarthria however occurs in many disorders other than aphasia. They involve basal ganglia (eg: Parkinsonism), descending corticospinal pathway (eg: pseudobulbar palsy), cerebellar hemispheres and connections to brainstem (eg: stroke, demyelination, tumors), cranial nerve nuclei and trunks of 5th, 7th, 9th to 12th nerves (eg: infective polyneuropathy), muscles of articulation in pharynx, palate, face, tongue (eg: myasthenia gravis). Dysarthria in aphasics is almost always accompanied by other deficits in language communication.

Reduced verbal output can be described as reduced phrase length, speaking monosyllables or single words or short sentences. Severe non-fluent aphasics sometimes repetitively utter a single word intoning it differently to convey some meaning like yes!, yes?, y..y..es ?.. Other manifestations of reduced verbal output are delay in initiation of a response, increased effort or apparent strain while generating the response, pauses in between words and sentences, interruption and word finding difficulties. The non-fluent speech in Broca’s aphasics is also described as “agrammatic”. Grammaticality means rule based use of many “function words” (conjunction, articles, prepositions, auxiliaries) to join “content words” (nouns, verbs, adjectives) into phrases and sentences. Agrammatic speech shows absence or paucity of function words. The short phrases are composed of content words only. Meaning is conveyed some-how in a manner resembling ‘telegraphic language’ as if to economize on words.

Circumlocution is replacing the unavailable word with round about description. Paraphasias exist, when instead of a word to be generated (target-word), another word is used. The distortion in the abnormal response word may be phonemic (sound related) or semantic (meaning related). Neologism means sound utterance or words, which do not exist in the vocabulary of the language being used by the patient. Jargon is a string of expression (verbal or written), which does not convey any meaning. Many of the so-called ‘words’ in a jargon are neologisms. Some others may be true words but are used in a jumbled up way without any context and without any grammatical rules hence no information is carried.

A small but significant minority of aphasics has fluent but empty speech. It is contrasting and opposite to non-fluent agrammatic speech. It appears to be fluent, effortless, with normal melody, accent, prosody and mannerisms. On close observation however speech is not normal. Phrases and sentences are of normal length or sometimes overly lengthy without a sense of completion. There are many paraphasias, circumlocutions and abundance of function words or grammar words whose information load is low. The content words, which really carry information, are in paucity and used flouting the rules of grammar. Jargon is common. Such patients have profound defects in auditory and reading compression. Classical example is Wernicke’s aphasia.

B. AUDITORY COMPREHENSION: Auditory comprehension is tested at one word level, sentence level and discourse level. At one word level the examiner speaks out the word at a slow and regular rate without repetition and asks the patient to point to corresponding picture or real object or body part. We should resist the temptation to act out the command ourselves. Patient’s performance is scored in terms of correct answers with respect to help or cues and time latency.

Sentence level comprehension is tested by multiple step commands: “fold this piece of paper and put into pocket”, “pick up the piece of cloth, put it into the glass and then invert the glass on the table”. Silly questions can be asked for which only yes or no answers are possible. Example: does the stone sink in water?, is elephant larger than an ant? A short story is narrated and questions are asked based on it.

Sentence level comprehension tasks also involve picture sentence matching. There can be one sentence and many picture choices or vice-versa.

C. REPETITION: The examiner asks the patient to repeat after him words and sentences. Initial stimuli are simple, short, common, and later they are made long, hard to pronounce and uncommon. The patient may totally fail, remain mute or come out with wrong responses. The errors may be sound disorders, incomplete responses, deletions or paraphasias. Some patient may perform fairly well on tests of auditory comprehension and also do well during daily life but fail miserably on repeating simple words or phrases (conduction aphasia). In contrast patient with transcortical sensory and mixed aphasia repeat long complex phrases mechanically or slavishly but do not follow or extract a bit of meaning from whatever they are listening and repeating.

D. NAMING: We begin with naming objects, pictures, body parts or persons. Examiner puts his finger on the object or photo and asks the patient to name it. Many patients with aphasia fail in different manner. Some may not come out with any response. There may be delay. The response word may be altered yet be related to target word either by sound (phonemic paraphasia) or by meaning (semantic paraphasia). The response word may be phonemic jargon (non word) or semantic jargon (dic-
Some patients may describe the word in a roundabout way (circumlocution). Cues or helps can be given. Phonemic cue means, uttering first one or two phonemes of the word or producing its rhythm. Semantic cue involves giving hint about synonyms or attributes of that word. In case of persistent failure, the examiner then offers the answer in two ways:

- Suggesting a wrong answer and asking if it is correct or not.
- Suggesting the correct answer and again looking if patient accepts it or not. In patients with total disruption of semantic memory of lexicon or access to it correct answers too many not be accepted.

The naming of objects can be made category-wise. Pictures or photographs of action verbs are one important category, others being colors, geometric figures, numbers, letters, body parts.

**E. READING:** The subject is made to demonstrate ability to recognize, match and discriminate the graphic symbols of alphabets. Then he is asked to read aloud words, phrases or paragraph without paying attention to the meaning. Reading comprehension involves word-picture or word-object matching and carrying out some commands written on a paper (eg: touch your hair, make a fist etc). Somewhat difficult task is to fill in the blanks in progressively difficult sentences from amongst a choice of detractors. Large paragraphs (unseen passage) are given to read and written questions are asked based on it. A mixed list of words and non-words is provided and patient is asked to separate them out.

**F. WRITING:** If the motor dexterity of writing hand is fair it is expected that patient would be able to reproduce his premorbid handwriting, at least in such mechanical tasks like copying from written stimuli, writing to dictation and serial writing of automatized sequences (like name, address, numbers, alphabets, weekdays, months). But patients with non-fluent aphasia fail in written expression too. In addition to various aspects of non-fluency (reduced output, laborious start, pauses, agrammatism), the mechanics of writing and calligraphy are also distorted. Fluent aphasics may write legibly but the written output will exhibit similar deficits of emptiness, paraphasias, jargon, circumlocution etc. Picture naming, object naming, describing the events going on in a painted or sketched scene are further tests of written expression.

Classification and Syndromes (Table 3) and (Figures 1a and 1b)

The classical aphasic syndromes have been grouped on the basis of speech and language performance in stroke survivors. Their distinction depends on the presence or absence of a constellation of speech behavioral aspects. The syndromes may be artefactual, arbitrary or artificial. New ways of classifying and describing aphasic phenotypes have been proposed. Yet they serve a useful purpose. Diagnosing an aphasia syndrome may have localizing value. Prognostication can be attempted on their bases.

Classification in a useful tool for summarizing the patient’s symptoms and for capturing some regularities of languages disruption due to the anatomical peculiarities of the brain vessels. However, there is overlap and many aphasias are unclassifiable.

**TO SOMETIMES DO A DETAILED CLINICAL EXAMINATION TO CREATE A PROFILE OF DEFICITS AND STRENGTHS IN SPEECH AND COMMUNICATION.**

More comprehensive detailed or focused clinical assessment of deficits in speech and communication are time consuming and require special interest, training and experience. However, a few more additional aspects of speech assessment, other than those described above, may occasionally be undertaken by physicians. The time taken gets reduced to some extent with practice. It is interesting and satisfying for the clinician and useful for the subject and family. Recently more
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 fasciculus, a white matter tract in deep temporal lobe. Also due to lesion in superior temporal or inferior parietal region.

emphasis is being placed on neurolinguistics and cognitive neuropsychology based approaches to assessment and therapy for aphasia. Linguistic analysis refers to theoretical concepts such as phonology (sound patterns, phonemes, phonetic features), morphology (word formation, morphemes), syntax (rule-based grammar, function words), semantics (lexicon, dictionary words, their meaning) and discourse (dyadic conversation, paragraphs, long speeches).

The classical anatomico-clinical approach provided knowledge about the relationship between lesion and function. Neuropsychologist found the existence of many selective deficits, suggesting that the brain is organized into distinctive systems or modules of relative functional independence.

Linguistically oriented aphasiologists are now keen to analyze the difficulties (faced by patients) in terms of the structure and systemic features that are disrupted or retained.

Neurolinguistics has also helped in speech therapy and rehabilitation. By more precise and detailed description of the language function of brain-damaged subjects, it is possible to
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Plan novel re-educative strategies and to monitor the recovery if performance.

**TO DO SYSTEMIC GENERAL AND NEUROLOGICAL EXAMINATION TO ASSESS THE OVERALL CLINICAL SITUATION AS A BACKGROUND AND CO MORbidITIES FOR APHASIA.**

Stroke being the commonest cause of aphasia, a clinical workup would naturally pay attention to and document in detail the common as well as uncommon (and modifiable as well as non-modifiable) risk factors for cerebro-vascular diseases. Associated neurological deficits in motor, sensory and cognitive functions will have a bearing upon speech-communication status and patient’s capacity to co-operate in rehabilitative speech language therapy.

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<tr>
<th>Transcortical motor</th>
<th>Transcortical Sensory</th>
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<tr>
<td><strong>Clinical:</strong> Similar to Broca’s aphasia with hesitant, telegraphic speech, normal comprehension and surprisingly good repetition.</td>
<td><strong>Clinical:</strong> Similar to Wernicke’s aphasia with fluent paraphasic speech, poor auditory and reading comprehension but surprisingly good repetition.</td>
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<tr>
<td><strong>Anatomy:</strong> Deep white matter in frontal lobe, anterior and superior to Broca’s area or near supplementary motor area. Anterior cerebral artery territory.</td>
<td><strong>Anatomy:</strong> Left temporo-occipital watershed infarction between MCA and PCA territories.</td>
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<tr>
<th>Anomic</th>
<th>Alexia without Agraphia</th>
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<td><strong>Clinical:</strong> Disturbance of the concepts of the words or the sound patterns of words or both. Most aspects of speech normal except naming.</td>
<td><strong>Clinical:</strong> Patient can write but cannot read even their own writing. Other aspects of speech are normal except naming, especially for colors.</td>
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<td><strong>Anatomy:</strong> Least localizable of all aphasic symptoms. Inferior parietal lobe or connection between parietal lobe and temporal lobe. Arterial territory : Angular branch of left MCA</td>
<td><strong>Anatomy:</strong> 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 hemisphere language centers.</td>
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Fig. 1b : Aphasias with lesions peripheral to perisylvian region

**TO TAKE INTO CONSIDERATION PSYCHOSOCIAL STATUS OF THE SUBJECT AND JOINTLY WORK-OUT REALISTIC AND MEANINGFUL GOALS.**

The premorbid educational, linguistic, occupational, economic, social, psychiatric and familial background should be carefully enquired into to gain insight into strengths and weakness of patient. Subjects who are highly educated, leading an intellectually rich life, having ample economic resources, good family support and highly motivated, will understandably have better response to speech language therapy. Attempts should be made to improve situation for those (unfortunately the majority) who are lacking in these attributes. Depression is common in stroke and aphasia, partly as a legitimate reaction to the disability and partly as neurobiological consequence.
of the lesion in the brain. Pharmacological (SSRI) and non-pharmacological (counseling) means should be employed. Many sessions with family will be needed to anticipate possible goals of rehabilitation over an extended period, of many months and years.

TO PLAN APPROPRIATE LABORATORY AND IMAGING INVESTIGATIONS.

Laboratory investigations will include general workup and evaluation for stroke-risk factors. The imaging of choice is MRI brain and MR angiogram to document the location, size and morphology of the lesions in brain and its vascular supply.

Clinic anatomical correlations between lesions depicted on imaging and the aphasia profile are along expected lines in the majority of the patients. However, about 30% exhibit misfits such as non-fluent aphasias with retrorolandic or posteriorly placed lesions, fluent aphasias with prerolandic or anterior lesions, instance of the lesion being present in an unexpected area, discrepancies between lesion size/volume and clinical severity of and recovery from aphasia. The most probable explanation is inter-individual variations in the anatomical substrate for language functions in brain. Factors such as handedness, dominance, age, sex, literacy and multilingualism may play a role in the degree of bilateral representation and other anomalous organization of speech function.

MRI has a high sensitivity and is better for three-dimensional volumetric analysis of various discrete areas of the brain. Functional neuroimaging (fMRI), PET, SPECT techniques are now paving the way for defining discrete neural modules or networks (not areas or centers) concerned with cognitive functions such as speech. Well-defined linguistic tasks are performed by subjects while imaging study results are compared before, during and after the task.

TO ALWAYS REFER THE PATIENT TO SPEECH THERAPIST, SEEK FEEDBACK FROM HIM/HER AND ACTIVELY INTERACT WITH HIM/HER.

Therapy requires teamwork involving a physician, psychologist, speech therapist, social worker and the relatives and colleagues of the patient. Aims of all therapies for people with aphasia should be to improve their functioning in the real social world, to enable them to cope with barriers to participation. Speech therapy is an interactive process and it is not a mechanical application of a predefined behavior. During therapy patient must be an active participant.

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 not because a performance consists of playing scales, but because doing this develops skills that results in better performance.

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.

The methods of speech therapy and material used are tailored according to the individual patient’s needs. Psycholinguistically based therapeutics is in its infancy, but some early reports are encouraging.

For patients with very severe aphasia, pragmatics based approaches are recommended which relate to analysis and repair of conversation in holistic sense. The therapist follows conversational coaching approach with the help of different themes or scripts, video tapes and replay and turn taking.

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.

TO CONSIDER ROLE OF PHARMACOTHERAPY

Many medicines have been studied in treatment of aphasia. The results are not impressive. But pharmacological augmentation of speech therapy may be more robust and reliable with stimulants, Cholinesterase inhibitors, dopamine agonists and other medications. Pulvermuller (2008) reported that memantine enhanced experience dependent neural plasticity elicited by intensive constraint induced aphasia therapy (CIAT). The authors discussed about neurofunctional links between language and motor action and described that somatotopic activation of motor areas occurs not only during simple repetitive movements but also when passively reading related action words for example: face (lick), arm or hand (pick) and leg or foot (kick). Methylphenidate given ½ to 1 hour before a session of speech therapy may help in consolidation of learning by improved attention.

Various medications have been used to enhance the effects of the language therapy. Studies of bromocriptine have had mixed success; some show improvement in speech fluency or other aspects of language, while other show no benefit of bromocriptine plus therapy over therapy alone. More recent studies have shown that piracetam, cholinesterase inhibitors, selective serotonin reuptake inhibitors, tricyclic antidepressants, or stimulants may be useful adjuncts to therapy. Several studies have investigated the effects of
the utility of speech therapy, as it satisfies the stringent criteria of evidence-based medicine. 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 the non-dominant (right) hemisphere. It is a wrong question to ask whether speech therapy (in general) is effective for aphasic patients (in general)? The right question should be: which specific modes of speech therapy can be beneficial to which particular types of patients?

When it comes to evidence base, it is true that there is not much in the form of randomized controlled trial to show for efficacy of aphasia therapy in general and therapy based on cognitive neuropsychology in particular. The type of conditions which justify use of RCT are (a) homogeneity of populations studied (aphasics are a heterogenous lot) (b) homogeneity of the treatment given (the methods are as varied as the patients) (c) sensitivity of assessment techniques (many are inappropriate and insensitive).

There are many reasons for the sorry state of therapeutic nihilisms amongst the minds of physicians and speech therapists. Different types of evidence and different methods are needed. Not enough has been done. The critical mass of adequately done therapy in sufficient numbers of subjects is missing. Wrong question has been asked – is speech therapy, in general, effective for aphasia, in general? The correct question should have been – are these and these practices of therapy useful in a aphasic subjects with such and such types of deficits? Not enough attention has been paid to many factors affecting outcome of therapy.

Several recent investigations have shown that intense treatment of at least 2 hours per day for at least 4 days per week for short period of 3-4 months is more effective than same duration of therapy spread out over a longer period (Bhogal SK 2003). A Constraint Induced Aphasia Therapy (CIAT) is an intensive form of language action therapy performed in small group settings. Participants have to request picture cards from each other by using descriptions of the depicted objects and understand request made by others and therapists. Other forms of communication (gesturing, drawing, writing) are not permitted.

Leora Cherny (2008) did a systematic review of effects of intensity of treatment and constraint induced language therapy for individuals with post-stroke aphasia and concluded that modest and preliminary evidence exists for more intensive treatment and CIAT.

In a recent Cochrane Review Kelly et al (2010) analyzed 30 trials with 41 paired comparison, 14 sub comparison, and 1064 participants. Speech Language Therapy (SLT) was compared with either no therapy or social support and stimulation or two different approaches of SLT. There was a consistency in the direction of results which favored intensive SLT over conventional SLT. Therapy by a trainee and a volunteer were also as effective as the one provided by a professional.

TO BECOME A BIT OF SPEECH THERAPIST YOURSELF WHILE COUNSELING THE PATIENT AND CAREGIVERS

All physicians by default are counselor and therapists. They should repeatedly reinforce following every day tips to be given to family members of aphasic patients

- Stress the language functions that the patient already
has. One can reinforce and promote the use of residual language.

- Comfort the patients’ saying that it is all right to communicate in whatever form they can or want to.
- Expose the patient to adequate language stimulation such as interesting radio and television programs.
- Encourage the patient to participate in speaking situations such as marriages, parties, picnics and other social get-togethers.
- Stimulate real life communicative situations, which the family and patients can act out by pantomiming and role paying.
- Promote rhyming, singing and imitating for the patient.
- Ask the patient to speak at a slow rate (20-25 words a minute) if the speech is imprecise or slurred.
- It is normal if a patient’s performance fluctuates from time to time. Aphasics are easily tired and perform better after a rest or a nap. Choose a time for working with the patients when they are the most responsive.
- Patients with aphasia follow and communicative better when the conversation involves only two persons. They get confused if more people are around or if there is background noise.
- The following cues can hierarchically be used to help elicit words from patients who have difficulty in finding words or object names. However, never demand an answer from the patients. Variability in behavior is common in aphasia, where the patients can retrieve one day and may not the other day;
  - Asking the object name. What is this called?
  - Direction to state the function of the item. For example, what do we do with it?
  - Direction to demonstrate the function of the object. For example, show me what you do with it.
  - Statement of the function by the family. For example, one writes with it.
  - Offering phonemic cue. The family provides the initial phoneme of the word. For example, “K.” for ‘Kitaab’
  - Supplying the first syllable. The family provides the word initial syllable. For example, “ta” for ‘Table’.
  - Sentence completion. The family provides the sentences without the target word and the patient completes the sentences by supplying the word. For example, “Main parta huun”
  - Praise all of the patient’s communicative efforts regardless of how small they seem. Aphasic patients are sensitive to positive reinforcements.
  - Encourage the patients to write or gesture. In the presence of hemiplegia, encourage them to use the non-dominant hand.

- Ways for communicating with an Aphasic
  - Listen attentively to the patients.
  - Do not interrupt. Allow the patients adequate time for responding.
  - Avoid supplying words as soon as you feel that the patient cannot find the word.
  - Accept all efforts of communicative regardless of how significant they are.
  - Speak in a quiet place, free from background noises. (i.e. the television, traffic and people)
  - Control the amount of your own verbalization.
  - Speak naturally at a slow rate (not more than 20-25 words a minute).
  - Speak at a normal volume (do not shout).
  - Communicate with the patients using:
    - Simple one-clause utterances
    - Familiar and concrete nouns and verbs
    - Limited information
    - Gestures, if they are more effective
  - Communicate when the patients is receptive and not tired.
  - Make sure you have the patient’s attention before you begin talking to the patients.
  - Attend to the meaning of the message and not its correctness

The internist and for that matter any medical practitioners caring for a patient with aphasia should be more knowledgeable about and interested in the subject. She or he should be convinced that speech therapy works and rehabilitation can be improved by it and other simple advices. Physicians 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. While referring and re-referring the persons with aphasia to the speech therapists, interested internists shall seek and provide feedback in a more detailed and precise manner. That in turn will increase the interest and commitment level of the therapists and finally the patient and caregivers will realize that something important and meaningful is being offered. A team will be built with limited resources.

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