PROTOCOL TO IDENTIFY APRAXIA OF SPEECH IN BROCA'S APHASICS

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This is to certify that this dissertation entitled ***"Protocol to identify Apraxia of speech in Broca's aphasics"*** is the bonafide work in part fulfillment for the degree of Master of Science (Speech & Hearing) of the student with Register No. M 9906.

Mysore,

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This is to certify that this dissertation entitled ***"Protocol to identify Apraxia of speech in Broca's aphasics"*** has been prepared under my supervision and guidance. It is also certified that this has not been submitted earlier in any University for the award of any other Diploma or Degree.

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Declaration

This dissertation entitled ***"Protocol to identify Apraxia of speech in Broca's aphasics"*** is the result of my own study under the guidance of Dr. R. Manjula, Lecturer, Department of Speech and Language Pathology, All India Institute of Speech & Hearing, Mysore, and has not been submitted earlier in any University for the award of any other Diploma or Degree.

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INTRODUCTION

Motor speech disorders can be defined as disorders of speech resulting from neurologic impairment affecting the motor programming or neuromuscular execution of speech. They encompass apraxia of speech (AOS) and the dysarthrias (Duffy, 1995).

Darley (1969) was one of the major proponents of the concept of AOS. He defined AOS as a neurologic speech disorder resulting from impairment of the capacity to program sensori-motor commands for the positioning and movement of muscles for the volitional production of speech. It can occur without significant weakness or neuromuscular slowness, and in the absence of disturbances of conscious thought or language. This definition is largely consistent with those used by Darley, Aronson and Brown (1975) and Wertz, LaPointe and Rosenbek (1984).

The performance of apraxic speakers is characterised by a typical set of features which, distinguish them from other communication disordered patients and thus allow us in identifying the disorder as a specific entity. Apraxic speakers struggle to position their articulators correctly. They visibly and audibly grope as they struggle to produce correct articulatory postures and to accomplish a sequence of these postures in forming words. Their articulation is frequently off-target. They often recognize that they are off-target and effortfully try to correct the error. Their errors recur, nonetheless, but they are not always the same and the errors on a series of trials are highly variable. As patients struggle to avoid

articulatory error by careful programming of muscle movements, they slow down, space their words and syllables evenly and stress them equally. Thus the prosody of their speech is altered as well as their articulation (Darley et al., 1975).

The highest level of motor speech programming (MSP) is carried out in the left hemisphere in the right handed individuals. The MSP area in left hemisphere is the primary motor area especially the Broca's area (McNeil, 1997). The MSP involves sensory feedback, the basal ganglia, the cerebellar control circuits, the reticular formation, the thalamus, the limbic system and the right hemisphere. The MSP also depends greatly on the pre-motor and supplementary motor areas. The left hemisphere function of MSP is more strongly bound to the linguistic component of speech than its emotional components. The linguistic input to the MSP comes largely from the left hemisphere perisylvian area which includes the temporo-parietal cortex and posterior portion of the frontal lobe (Duffy, 1995).

The anatomical proximity of these language areas with those of the MSP makes it likely that damage to the perisylvian zone often results in a co-occurrence of language related deficits like aphasias and AOS (Darley et al., 1975). A Mayo clinic survey in the late eighties reported that nine percent of patients with left hemisphere pathology are found to have AOS. Consequently, the clinical manifestations of AOS are frequently buried within the generic heading of dysarthria and also frequently within categories of aphasia.

Differentiating AOS from the dysarthrias is easier and more clearly established than differentiating it from the aphasias.

Among all the aphasias it is most commonly found that AOS co-exists with Broca's aphasia (DeRenzi, Pieczuro and Vignolo, 1966; Kertesz and Hooper, 1982). However, it is also common to find Broca's aphasia and AOS existing independently. This is because AOS can be caused by damage to other areas of the brain apart from the Broca's area (Duffs', 1995).

Understanding AOS when it exists in adults with Broca's Aphasia is challenging because, differentiating between the respective phonetic-motoric and linguistic impairments is difficult (Ballard, Granier and Robin, 1999). The few clinical descriptions available lack diagnostic power and fail to clearly differentiate between the components of each of the above two entities (Buckingham, 1979; Duffy, 1995; McNeil 1997). This is also because of the overlap of features in both these disorders. Some of these are as follows :

- Limited verbal output
- Effortful laboured speech
- Dysprosody
- Reduced phoneme length
- Slow rate
- Impaired repetition and confrontation naming

- Increase in error with increase in utterance complexity (linguistic or articulatory)
- Inability to increase speech rate effectively
- Reduced speed and timing of articulators
- High intra and inter-subject variability

(Darley, 1969; Rosenbek and Wertz, 1976; McNeil, Robin and Schmidt. 1997).

It is however possible to isolate features of AOS by using more qualitative descriptions rather than quantitative ones. A review of various tests available for AOS in adults shows that most of these tests lack normative data, and have inadequate psychometric properties of measures of validity and reliability. There is more focus on quantitative measures rather than qualitative descriptions and moreover the aphasic difficulties are not taken into consideration in various AOS test batteries.

Hence the present study aims to develop and evaluate a protocol containing tasks with specific keys to qualitatively analyse and isolate the features of AOS.

Limitations of the Study :

- The number of subjects evaluated with the protocol were limited (five subjects). The protocol needs to be administered and validated on a larger group of subjects for it to be standardized
- No control was established or considered to delineate the dysarthric speech errors if any in the subjects.
- Item analysis could have been a good measure for further validation of the protocol.

REVIEW OF LITERATURE

Speech is a complex action involving a number of levels of organisation and representative processes, eg., the cognitive, neuromuscular and musculo-skeletal activities (Duffy, 1995; Van der Merwe, 1997). It is the externalized expression of language. The sensori-motor control of speech can be defined as the motor afferent mechanism that direct and regulate speech movements (Netsell, 1982).

During the production of speech, the intended message has to be changed from an abstract idea to meaningful language symbols and then to a code amenable to a motor system. The identification of phases involved in this process, however remains unclear and debatable.

Most neurophysiologists recognize that the overall motor control process involves several phases or hierarchical levels of organisation (Lacquanniti, 1989, Jakobson and Goodale, 1991). The phases generally are identified as planning, programming and execution (Schmidt, 1978; Marsden, 1984: Brooks, 1986; Gracco and Abbs, 1987). A dysfunction at the level of planning and/or programming leads to apraxia of speech (Darley, 1969)

APRAXIA OF SPEECH (AOS)

Apraxia of speech also referred to as verbal apraxia, speech apraxia, phonetic disintegration and a host of other terms is a speech disorder of motor programming (McNeil and Kent, 1990).

The reference to AOS is not recent. Liepmann (1900) who developed the general construct of apraxia considered the speech behaviour of the Broca type patient to be an apraxia of the glossolabial pharyngeal musculature. Wilson (1908) said that "in motor aphasia we have a form of apraxia viz., apraxia of the speech musculature". Nathan (1947), Critchley (1952), Wepman and Van Pelt (1955), Wepman (1960) and Denny (1965) all have used some form of the term to clarify the nature of the phonologic impairment.

Earlier, researchers classified neurogenic speech production disorders as either linguistic (aphasias) or motoric (dysarthrias). However Darley (1967) observed that there was a clinical phenomenon of neurologic origin that did not fit into either of the above categories of aphasias or dysarthrias. Hence the subsequent research and reports subdivided motor speech disorders into the dysarthrias and apraxia of speech (AOS) implying that apraxia could also exist as a speech disorder also.

For more than a century, investigators were unable to agree whether AOS is a motor programming disorder devoid of linguistic components or a phonological disorder without a discrete separation from other language processes (Dunlop and Marquardt, 1977). Earlier investigators like Darley (1969) suggested that the term AOS would only be applicable when assurance could be given that the patient had the "intent", the underlying "linguistic representation" and the fundamental motor abilities to produce speech, but could not do so volitionally. He specified AOS as a disorder of speech that was

attributable to a disorder of the programming of the speech movements.

Other subsequent investigations (Johns and Darley, 1970; Alen, Johns and Darley, 1971; Deal and Darley, 1973; LaPointe and Johns, 1975) have described AOS as a non-linguistic motor programming disorder without significant speech musculature weakness, slowness and inco-ordination.

Martin and Rigrodsky (1974) found that a group of aphasics with phonological impairments made more phoneme errors when they were asked to repeat meaningless stimuli rather than meaningful stimuli. They concluded that the semantic component of words would significantly aid in the motor production of phonemes.

Martin (1974) hypothesized that the influence of linguistic variables on phonological production demonstrated that motor acts cannot be discretely separated from other language, processes or their possible impairment. He suggested that the repetitions, blocks and groping behaviours of apraxics toward correct production were similar to other aphasic behaviours, clearly showing difficulty in processing of linguistic units.

Alen, Darley, Deal and Johns (1975) indicated that defining apraxia as a motor programming disorder, not primarily due to impairment of sensory, intellectual or higher language functioning, does not assert that speech and language are unrelated or that phonological impairment is wholly influenced by language functioning, but does suggest that programming of phoneme production can be selectively impaired without impairment of language functioning.

Recent investigations (Kelso and Tuller, 1981; Kent and Adams, 1989, McNeil, 1997) have interpreted the behaviours of individuals with AOS as impairment of linguistic phonological processing, motor control, or both. Acoustic, kinematic and perceptual studies of speech in recent years have lead to advances in the understanding of AOS and wide acceptance that it affects phonetic-motoric planning of speech.

To understand the important theoretical accounts which were put forth in the eighties and nineties it is essential to keep in mind the levels of speech sensori-motor control. The control of movements is considered to be exerted through a command (or sensori-motor) hierarchy that can be portrayed as highest, middle and lowest levels. The highest level is mediated by the association cortex (eg., prefontal, parietal and temporal lobes) which generates overall invariant motor plans. Motor plans are converted into motor programmes at the middle level, which consists of the sensori-motor cortex, the cerebellum and the putamen loop of the basal ganglia. At this level the specific parameters of the movement (eg., amplitude and speed) are defined. At the lowest level programs are translated into muscular activity and motor execution occurs (McNeil, 1997).

A brief review of the prevailing theoretical approaches to AOS claims that the processes that build the phonological representation of a message are intact but the phonetic-motoric level of production is disrupted (Ballard et al., 1999). Kelso and Tuller (1981) proposed the coalitional theory of AOS in which AOS is viewed as a breakdown in the interaction between an individual and his

environment that results in failure to meet behavioural goals. According to Kelso and Tuller (1981) for skilled actions to be co-ordinated, the neuro-muscular system must be organised into functional units. Numerous studies have shown that this finely tuned spatio-temporal co-ordination between articulators is disrupted in AOS (Freeman, Sands and Harris, 1978; Itoh, Sasanuma and Ushijima, 1979a; Kent and Rosenbek, 1983; Ziegler and Von Crammon, 1986; Kent and McNeil, 1987). Hence collectively these studies interpret AOS as a disorder affecting the phonetic motoric level of speech production. Furthering this thought, McNeil et al., (1997) stated that AOS is a phonetic-mot one disorder that affects the translation of an intact phonological representation of a message into the learned kinematic parameters for an intended movement.

Kent and Adams (1989) believe that there is inco-ordination of articulatory movements in AOS, which leads to variability the in production of target movement patterns. Shriberg, Aram and Kwiatkowski (1997a) referred to AOS as a deficit in sequencing the spatio-temporal aspects of movement at a pre-articulatory level.

Whiteside and Varley (1998) proposed a cognitive based account of AOS which posits two routes for phonetic encoding. The direct route is used for encoding frequently used phoneme sequences or syllables and utilizes minimal computational resources. The indirect route is used for encoding very low frequency or novel syllables and words involving compution of the phonetic representation on a phoneme by phoneme basis. On similar lines, Whiteside and

Varley (1998) claimed that individuals with AOS lose access to verbomotor patterns or motor programmes via the direct route and must hence compute phonetic representations phoneme by phoneme. This process explains the reduced coarticulation seen in AOS. (Ziegler and Von Crammon, 1985; McNeil, Hashi and Southwood, 1994; Mayer, 1995). Furthermore, the authors proposed that the indirect route is not used efficiently in compensating for the loss of the direct route of encoding This poor compensation is thought to result in articulatory groping, increased segmental and intersegmental duations, and interarticulatory dis-coordination.

Darley, Aronson and Brown (1975) suggest that the phase of spatial-temporal planning of movement corresponds to syntactic planning during speech production. Thus, the true nature of motor planning of speech movements is therefore neither adequately described nor differentiated from phonological planning (Me Neil, 1997). Rogers and Storkel (1998) observe that in AOS the phonological output buffer, holding the output of the speech programming processes is limited to one syllable. Hence there is slowed production which is evidenced in individuals with AOS, who cannot program two words or syllables into the phonological buffer at a time.

Apart from the above theories, the model developed by Van der Merwe (1997) has been widely accepted in explaining the AOS processing. Van der Merwe (1997) developed a model for considering diagnosis and management of motor speech disorders, including AOS and dysarthrias. This model includes

linguistic-symbolic planning, motor planning, motor programming and execution levels and relates these to respective neural substrates. This model offers detailed and comprehensive explanation of the impairments in the speech production process, relating neural structures, diagnosis and treatment of motor speech disorders (Ballard et al., 1999). This model clearly separates linguistic from motoric planning and motor planning from motor programming. The present study derives its basis from Van der Merwe's model and aims to develop a protocol to extract the programming level errors (AOS) if present in presence of a known linguistic symbolic planning deficit (aphasia). Earlier to this proposition, neurophysiologists deduced motor planning to be equivalent to linguistic symbolic planning (Van der Merwe, 1997). The model also states that linguistic-symbolic planning should be differentiated from phases of sensori-motor control. According to Van der Merwe (1997) the sensori-motor control of speech movements comprises of linguistic symbolic planning, motor planning, motor programming and execution phases. With the latter three phases especially involved in speech motor processing, a dysfunction at any of the three levels can disrupt the normal speech production.

With regard to the neural structures responsible for linguistic-symbolic planning, there are indications that the temporal-parietal area, the Wernicke's area and Broca's areas are involved. A dysfunction at this level would result in aphasias (Levy, 1977; Meyer, Sakai, Yamaguchi, Yamamoto and Shaw, 1980). Similarly a dysfunction at the level of motor programming would result in the improper selection and sequencing of motor programs of the muscles of the

articulators (including vocal folds) and improper specification of the muscle specific programs in terms of spatio-temporal and force dimensions such as muscle tone, rate, direction and range of movements. The areas in the brain involved in motor speech programming include the primary motor areas (especially the Broca's area), pre-motor areas, supplementary motor areas and temporo-parietal areas. It is a dysfunction in the motor speech programmer (MSP) that leads to apraxia.

Apraxia is often seen in association with aphasia although at times it appears independently. Similarly, not all cases of aphasia are associated with apraxia (Ajuriaguerra, Hecaen and Angelergues, 1960). It has been suggested that the close relationship between praxis and language may be related to sharing of the same neural structures (Kertesz and Hooper, 1982).

ASSOCIATION OF APRAXIA AND APHASIA

Association between the two disorders, aphasias and apraxia has been explained mostly in terms of:

A) Neuro - anatomical correlates.

B) Neuro - physiological correlates.

C) Behavioural manifestations of both the disorders together.

A) Neuro-anatomical basis for the association of apraxias and aphasias

Earlier studies used computerized tomography to locate lesions in the frontal and central opercula and anterior insula in aphasic and non-aphasic patients with buccofacial apraxia (Tognola and Vignolo, 1980). This was to investigate the probable anatomical basis of association between aphasia and apraxia. Aphasia and apraxia are commonly caused by left middle cerebral artery (MCA) occlusion that damages both language and praxis areas (Kertesz and Ferro, 1984)

Localization of the speech territory has been established by a variety of means. The most significant advances in early years were electrical stimulation studies of Penfield and Roberts (1959) and the study of small penetrating head wounds by Russell and Espir (1961). They and many other authors divide the speech territory into posterior and anterior areas.

The posterior areas comprise the mid and posterior temporal lobe, the adjacent inferior parietal lobe and the adjacent anterior occipital lobe. This posterior zone is loosely equated with but is larger than Wernicke's area. The posterior area functions as an analyzer for speech reception and an integrating processor (CLP-Central Language Processor) for all modalities of language. The anterior zone includes the foot of the third frontal convolution, Broca's convolution. The apparent interconnection between the posterior and anterior zones is the arcuate fasciculus. The function of the anterior area is programming of motor speech (Darley et al, 1975). Hence, keeping the anterior, posterior

division in view it can be said that patients with AOS may exhibit alterations of language also as-

(a) Damage to the anterior area (Broca's area) can lead to both MSP deficits as well as Broca's aphasia.

(b) A lesion damaging the anterior speech area may extend posteriorly and also damage the posterior language area.

(c) The damage to the anterior (MSP) area impairs the operational efficiency of the posterior area.

(d) Feedback of the arcuate fasciculus to and fro the anterior posterior zones may be affected.

(e) Damage to Broca's area may disturb the balance between the two hemispheres leading to interference by the non-dominant lobe. (Darley et al., 1975).

Darley et al., (1975) also posit that the motor association area for non-speech oral movement is anterior to the motor cortex. Its functional relationship to Broca's area is uncertain. Damage to this area produces oral apraxia for non-speech movements. AOS may occur without oral apraxia but when oral apraxia occurs it is commonly in associated with AOS (Tognola and Vignolo, 1980). Nevertheless oral apraxia has been reported in patients with AOS or limb apraxia (DeRenzi et al., 1966). These findings suggest that the MSP projects directly to the motor cortex rather than relaying through the motor association

area for non speech oral movements.

Alexander, Benson and Stuss (1989) in their review of frontal lesions and language, equated aphasia (agrammatic language disorder) with AOS. The principle features of the two disorders were certainly similar especially the non-fluent speech output. Duffy (1995) states that apraxia and aphasia can be frequently associated because there are no significant differences between the disorders that may cause them (usually cerebro-vascular accidents). Moreover, there are no significant differences between the two disorders in terms of their gross anatomic or vascular characteristics. Other left frontal and deep basal ganglia syndromes are said to be less closely related to AOS according to Kirshner(1992).

B) Neurophysiologic correlates

Based on numerous cortical mapping studies Ojemann (1984) showed that motor and language mechanisms share many common neural sites. The two general areas suggested were:

(i) Ventrolateral nucleus of the thalamus

(ii) The lateral perisylvian cortex of the dominant hemisphere (anterior and posterior).

The latter, according to Ojemann (1984) was thought to be responsible for sequential motor movements and language decoding He also speculated that a "precise timing mechanism" may underlie certain aspects of language as well as

motor control.

Metter, Riege, Hanson and Phelps, (1983) corroborated Ojemann's conclusion that motor functions and language processes may share common neuroanatomical sites. Results of their glucose metabolism investigations indicated that the caudate, a structure traditionally felt to be active in the programming of learned movements, was also active during some language activities, especially some which were traditionally included in aphasia batteries.

C) **Behavioural manifestations of both the disorders together**

Liepmann (1905) surveyed a series of 89 patients and found the incidence of aphasia in 14 out of 20 left hemisphere damaged patients who had left sided apraxia and right hemiplegia. He termed this as Sympathetic dyspraxia. He pointed out that impaired comprehension was not the cause of apraxia. He also concluded that the left hemisphere was dominant for purposeful movements for both sides of the body and called attention to the importance of the corpus callosum in the neural mechanism of apraxia.

The relationship between aphasia and ideational apraxia (Ajuriaguerra, et al., 1960; De Renzi Pieczuroand Vignolo, 1968) and between the expressive difficulties in aphasia and apraxia was emphasized by several studies (Nathan, 1947; Alajouanine and Lhermitte, 1960; De Renzi et al., 1966). Goodglass and Kaplan (1963) examined 20 mild to moderate aphasics and did not find significant correlation between the severity of aphasia and the gestural scores.

They suggested that although there is no causal relationship, the association of language and praxis is related to the contiguity of the neural structures involved. Bay (1964) in a study of 80 unselected aphasic patients reported a distinct subgroup with distinct apraxia of the articulatory muscles and impaired tongue movements evidenced in the glossogram. Schuell. Jenkins and Jimenez-Pabon (1964) in their study of aphasic patients identified a group who presented articulatory problems seemingly independent of their aphasic impairment. The oral and limb gestures of 105 aphasics were studied by DeRenzi et al., (1966) who found oral apraxia in the majority (90%) of Broca's aphasics but only 33% of conduction aphasics. Similar results were also obtained by Kertesz and Hooper (1982).

DISSOCIATION OF APRAXIA AND APHASIA

Ojemann (1984) cautioned that not all perisylvian sites responsible for language were also responsible for motor operations. He stated that one should not expect every lesion that produces a language deficit to produce a motor apraxia but rather only those that damage the common language motor system.

Square-Storer, Roy and Hoggs (1990) view apraxia as a disorder which is distinct from aphasia. According to them, in an efficient symbolic communication, cognitive processes precede the motoric operations for all the intentional movements. The stage of "representational translation" (wherein there is a translation of a designatum i.e., idea, feeling or percept into a representational

communicative symbol/sign i.e., a word, phrase etc.) precedes the planning, programming and execution. An impairment of the translational stage may be inaccessibility of semantic memory store or wrong selection of a symbol once within a semantic memory store (eg. substitution). This is a characteristic aphasic error. On the other hand incorrect undifferentiated and amorphous limb/orofacial responses may also result from motor planning or programming disturbances. Hence, in the former case the production of a wrong representational gesture may be falsely considered as an apractic error in patients with left hemisphere damage. Therefore, according to Square-Storer et al. (1990) language/symbolic processing is independent of motor processing.

Aphasia without apraxia

Heilman, Rothi, Campanella and Wolfson (1979), Kertesz et al. (1984) reported patients who were moderate to severely aphasic but who demonstrated none or only mild praxic deficits. Kertesz et al. (1984) quantitatively investigated the functional anatomical relationship of aphasia and apraxia. Of their 177 patients, six were found to be severely aphasic with no apraxia. The latter were assessed using 15 limb and five orofacial items. Imitation was used to reduce effects of auditory verbal comprehension disorder.

The above findings were substantiated with neuroanatomical explanations:-

- Four out of six atypical skull asymmetries indicated that the visuokinesthetic patterns may be represented bilaterally rather than being

dominant in the left hemisphere.

- The fifth patient had a lesion which spared left pre-motor, fronto-parietal, sub-cortical connections and callosal connections.

- The sixth patient had severe Wernicke's aphasia resulting from a small lesion in the superior temporal gyms sparing the occipital, frontal and parietal lobes and their connections.

Hence the above neuroanatomical correlates support sparing of the praxis performance.

Apraxia without aphasia

In the literature a few histories of patients who demonstrated apractic symptomatology, but none or mild aphasia has been reported (Hecaen. 1978; Square, Darley and Sommers, 1981; Seines, Rubens, Risse and Levy, 1982; Kerteszetal. 1984).

Shankweiler and Harris (1966) showed that severe articulatory difficulty occurred in their five apraxic patients without impaired recognition of speech sounds. Johns and Darley (1970) found that the ten apraxic patients whom they studied were generally better in visual and auditory perception of speech stimuli than in oral production. According to Aten et al. (1971), AOS can occur in relatively pure form in the absence of auditory perceptual impairment. Hecaen (1978) felt that apraxia could occur in the absence of aphasia as the result of a callosal lesion.

Seines et al. (1982) presented a case that represented the opposite dissociation of severe apraxia with a mild recovering aphasia. A large suprasylvian infarct seemed to spare Wemicke's area. They postulated a bilateral representation of language but unilateral kinesthetic motor engrams. They interpreted their findings as a dissociation of language and praxis mechanisms.

In those studies in which apraxia has appeared as the exclusive disorder, sub-cortical motor structures have often, but not always, been identified as lesion sites; the sites included being, basal ganglia and thalamus (Square et al. 1981; Agostini, Colleti, Orlando and Tredici, 1983; Square-Storer, Darley and Sommers, 1988). These authors also report of other pure cases of apraxia without aphasia having parietal lobe lesions.

ASSOCIATION OF AOS WITH BROCA'S APHASIA

Cortical damage to the inferior posterior region of the frontal lobe in the left hemisphere can impair oral movements, in particular articulate speech production which is known as AOS (Martin, 1974; Johns and LaPointe, 1976; Benson, 1979). The controversy continues as to whether the AOS or disturbance in articulatory programming is the same as Broca's aphasia or is a condition that can occur in isolation of language impairment.

According to Marie (1906) left frontal lobe lesions produce only anarthria. Similar to this proposition even in the 1960s, the view was that non-fluent aphasias of Broca's type do not represent true aphasias or language disorders but rather an apraxic misreading of phonemic expression. However, modern

descriptions of Broca's aphasia not only reports disturbed phoneme articulation but also a grammatical language disturbance. Hence, Broca's aphasia is an aphasia and not merely an AOS.

Wertz, Rosenbek and Deal (1970) found that 65% of patients with AOS also demonstrated aphasia, 14% a combination of apraxia and aphasia and dysarthria, 13% had apraxia only and 8% showed apraxia combined with dysarthria.

Extensive research and observation by investigators of the seventies and eighties led to the opinion that AOS is a non-linguistic speech disorder. It was observed to co-exist with other disorders and was frequently observed with aphasia and/or dysarthrias (Darley et al., 1975; Wertz et al., 1984)

McNeil and Kent (1990) opine that it is reasonable to conclude that people with Broca's and nonfluent aphasia usually have an accompanying AOS and also state that AOS may be an integral part of the syndrome of Broca's aphasia.

Most definitions of Broca's and non fluent aphasia along with their descriptions of agrammatic and syntactic errors do not give overt recognition to the existence of a motor speech programming deficit. They do, however, describe patients speech as slow laboured or effortful, reduced in phrase length, abnormal in prosody and having poor articulatory ability. These characteristics are consistent with those of speakers with AOS (Duffy, 1995). Duffy concludes that, AOS is not synonymous with Broca's aphasia because the aphasic

component of the syndrome includes deficits that are not explainable by AOS and also because AOS can occur without any manifestations of aphasia. According to him, both AOS and aphasia are discrete entities which however frequently can co-exist because they share the same neural structures.

McNeil et al. (1997) state that by definition, AOS is a motor planning or programming disorder but the arguments about whether AOS is a phonological disorder or motor programming disorder still exist. Many authors hold the view-that defining apraxia of speech has been done but the issue of specifying to whom the term applies is still a difficult task. This implies that identification of AOS in an aphasic is a challenging task, which needs indepth study of the characteristics of AOS and how they can be extracted in the presence of an already existing aphasic problem, especially Broca's aphasia.

According to Kent and Rosenbek (1983), AOS is the impaired volitional production of articulation and prosody. The articulation and prosodic disturbances, however do not result from muscle weakness or slowness, but from inhibition or impairment of the CNS's programming of oral movements.

Wertz et al. (1984) state the salient, clinical characteristics of AOS as follows.

a) Effortful trial and error, groping articulatory movements and attempts at self correction.

b) Extended periods of abnormal rhythm, stress and intonation.

c) Articulatory inconsistency on repeated production of the same utterance.

d) Obvious difficulty in initiating utterances.

The characteristics of AOS can be briefly reviewed under the following titles:

A) ARTICULATORY CHARACTERISTICS

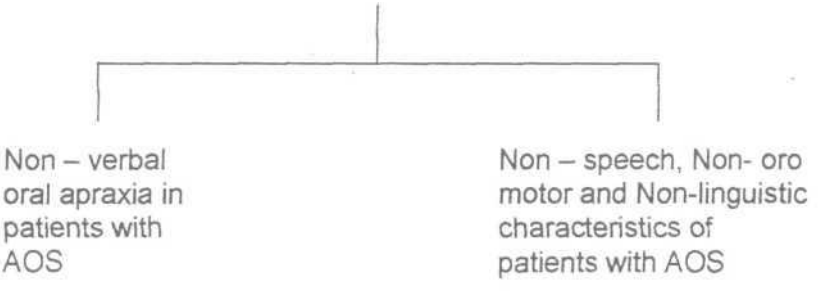
- Phonologic
- Phonetic - motoric
- Phonologic influences on articulation
- Non Phonologic influences on articulation

B) PROSODIC CHARACTERISTICS

C) ACOUSTIC CHARACTERISTICS

D) PHYSIOLOGIC CHARACTERISTICS

E) NON-SPEECH CHARACTERISTICS



A) ARTICULATORY CHARACTERISTICS

Darley (1969) listed the following as the traditional characteristics of AOS.

- Prominent phonemic errors including substitution, omission, distortion, addition and repetition of phonemes.
- Errors which are perseverative or anticipatory.
- Errors which are seemingly off target approximations of the desired production made in an effortful groping for the correct position or sequence of positions.
- Errors which vary with the complexity of the articulatory adjustment.
- Errors which increase as words increase in length.
- Discrepancy between the articulatory accuracy displayed in automatic reactive speech performance and the inaccuracy displayed in volitional purposive performance.
- Imitative responses are particularly poor.
- The speaker is usually aware of his/her errors but is typically unable to anticipate or correct them.

Johns and Darley (1970) report of

- Numerous phonemic errors including substitutions, omissions, additions, repetitions and distortions, with a predominance of substitutions, in the absence of significant weakness, slowness and inco-ordination of the speech musculature. This is also supported by Trost (1970).

- Phonemic error inconsistency : Substitution of a variety of phonemes and phoneme clusters for correct phonemes and inconsistently correct phoneme production, including islands of error free fluency especially during a period of automatic reactive speech. This is also supported by Shankweiler and Harris (1966) and Lapointe (1969).
- Difficulty in initiation of speech: Effortful speech production during purposive volitional speech characterized by hesitant groping movements of the articulators prior to and during speech production and numerous retrievals at correct word production.
- An increasing number of phonemic errors with increasing word length.
- A marked discrepancy between speech perception and speech production. Perception may be good but production is poor.

Rosenbek and Wertz (1976) reported that -

- Substitution errors are more frequent than other error types.
- Error sounds are more likely to differ from target by one phonetic dimension than by two, three or four.
- Errors are most likely those of place followed by errors of manner, voicing and oral/nasal substitution.
- Voiceless for voiced substitutions are more frequent than voiced for voiceless substitutions.

- Some errors are anticipatory, some perseverative and some metathetic, with anticipatory errors probably predominating.
- Errors are more likely on consonant clusters than on singleton consonants.
- Apico-alveolar and bilabial sounds are more often correct than sounds produced at other places.
- Affricates and fricatives as a group tend to be more often erroneous than plosives, laterals, nasals and vowels, although order varies with the position in the utterance.
- Consonant errors are more likely than vowel errors. Some patients may not exhibit more consonant errors than vowel errors.
- Many substitutions appear to be of more difficult combinations for "easier" ones.

Phonetic dimensions

Shankweiler and Harris (1966) found an apparent unrelatedness of many (1/3rd) of the substituted sounds to their targets. The phonetic features of errors may vary widely from target sounds.

Trost and Canter (1974) state that errors are close approximations to their target sounds.

Voiced- Voiceless errors

There is a lot of variability in the literature regarding this feature. Shankweiler and Harris (1966) report more voiced substitutions for voiceless targets than the reverse. Most studies on voicing errors indicate that most apraxic patients' tendency is to substitute a voiceless consonant for a voiced consonant. (DeRenzi et al., 1966; Trost and Canter, 1974; Nespoulous, Joanette. Ska. Caplan and Lecours, 1987). LaPointe and Johns (1976) and Freeman et al. (1978) found no differences in the direction of voicing errors.

Substitution errors

Most researchers (Shankweiler and Harris, 1966; Johns and Darley, 1970; Sasanuma, 1971; Trost and Canter, 1974; LaPointe and Johns, 1975; Klich, Ireland and Weidner, 1979) identified substitution errors in their patients than errors of distortion, addition, repetition and omission.

While most of the reports on substitution errors focussed on consonants, Lebrun. Buyssens and Henneaux (1973) gave special attention to vowels. They observed that vowels were never substituted for consonants or vice versa in their two patients and back vowels were never replaced by front vowels or the converse.

Using narrow phonetic transcription of mono, bi- and tri -syllabic word repetitions, Odell, McNeil, Rosenbek and Hunter (1990) have reported that their four AOS subjects produced more consonant distortions (25%) than substitutions (6%).

Anticipatory, Perservative and Metathetic errors

Trost and Canter (1974) noted a paucity of metathetic errors in their sample of apraxic patients. Sasanuma (1971) found metathetic errors to be maximum in number.

LaPointe and Johns (1975) examined three types of sequential errors: anticipatory/prepositioning ('telo' for 'yellow'), reiterative or post-positioning ('dred' for 'dress') and metathesis ('tefalone' for 'telephone'). Percentage of sequential errors relative to other error types was small. Maximally found errors were anticipatory errors.

Consonant errors

Many authors (Shankweiler and Harris, 1966; Johns and Darley, 1970; Dunlop and Marquardt, 1977; Trost and Canter, 1974; LaPointe and Johns, 1975; Darley, 1982) agree that consonant clusters evoke more errors than singleton consonants and that certain consonants are more difficult than others (affricates and fricatives are more difficult than plosives, laterals and nasals).

LaPointe and Johns (1975) found that most difficult for their sample of apraxic patients were lingua-palatal sounds followed by lingua-dental, lingua-velar, labio-dental, glottal, lingua-alvelolar and bilabial.

Vowel errors

Odell, McNeil, Rosenbek and Hunter (1991) in their narrow phonetic transcriptions demonstrated AOS as having -

- Predominant errors in low, tense and back vowels

- More distortions than other types of vowel errors

- Predominant errors in initial position of words and in monosyllabic words

Consonant vs. vowel errors

Many authors (Trost and Canter, 1974; Keller, 1978; Darley, 1982) observed more errors on consonants than on vowels.

Phonetic disintegration

Shankweiler and Harris (1966) and De Renzi et al., (1966) used the term phonetic disintegration but observed that their patients sometimes ultered difficult strings of consonant clusters as substitutes for easier targets. Johns and Darley (1970) hence reject the term phonetic disintegration.

Coarticulation

Ziegler and Von-Crammon (1985) proposed that if the apraxic speaker had poorly specified information about the upcoming vowel, then this lack of specification would be reflected in the preceding consonant as a lack of co-articulation with the vowel. In their study, normal listeners were asked to judge the upcoming vowel from CV stimuli. The stimuli were such that they had progressively less acoustic information (using selectively gated portions of the acoustic signal ranging from the consonant with a portion of the vowel, to only small portions of the word - initial consonant). The vowels of **the** normal speakers were predicted accurately with only small portions of the consonant (and

no vowel signal). The produaions of the apraxic speakers were poorly identified with an equivalently gated acoustic signal. The authors concluded that the apraxics showed a delayed onset of anticipatory vowel gestures relative to labial occlusion.

INFLUENCES ON ARTICULATION

i) *Phonologic influences* (Rosenbek and Wertz, 1976)

Initial, medial and final positions in a word may or may not have an influence on the speech sound integrity

- Frequently occuring sounds are more likely to less erroneous than infrequently occurring ones.
- Articulatory accuracy is better for meaningful than for nonmeaningful utterances.
- Errors increase as words increase in length but this increase is not linear.
- Errors increase as distance between successive points of articulation increases.

Grammatical class when combined with difficult initial phonemes, longer words and an early position in the utterance, influences the probability of an error.

ii) ***Influences*** *due* ***to stimulus manipulators***

Stimulus manipulators also affect response accuracy in AOS

Visibility, length arid articulatory complexity - strongly affect difficulty for apractic speakers. With increase in visibility, error decreases. With increase in length and complexity errors increase (Shankweiler and Harris, 1966; Johns and Darley, 1970)

- Increase in *rate* increases difficulty (Johns and Darley, 1970)

- *Delay* - When a time delay is introduced between the clinician's stimulus and apractic speakers speech production, the imitation of speech is more difficult for the apraxic (Johns and Darley, 1970)

Sound position - Initial sound is more likely to be produced incorrectly than subsequent sounds (Shankweiler and Harris, 1966; Trost and Canter, 1974). Others who have failed to confirm this effect are Johns and Darley (1970), LaPointe and Johns (1975), Dunlop and Marquardt (1977).

- *Prompting* - Love and Webb (1977) studied the effects of 4 different types of prompts in clients with Broca's aphasia and AOS - the complete target word, the sentence with target word missing, first sound of the target word and printed target word. The complete target word was found to be most successful in eliciting the target word followed by sentence completion and printed words.

- *Stimulus modality* - Most studies show that stimuli presented by multimodality have shown to result in better verbal responses in apraxics (Johns and Darley, 1970).

- *Meaningfulness* - Meaningful material is easier for apraxics(Johns and Darley, 1970). In contrast, another study suggested concentration on production of non-meaningful articulatory sequence in order to teach *the* patient volitional control of speech production before attempting meaningful words.

- *Automation*- - Over-learned sequences like counting etc., may be surprisingly easy for some patients who are severely apractic (Johns and Darley, 1970; Darley, 1982).

- *Context* - Placing a word in a frequently occurring phrase usually makes it easier for the apractic patients.

Situational cues - also may affect success (Trost and Canter, 1974).

(iii) Non — Phonologic influences - Adapted from Rosenbek and Wertz (1976)

- Within narrow limits articulatory accuracy is better for automatic reactive than for volitional purposive speech.

- Articulatory accuracy may be better with auditory visual stimulation than with auditory or visual (reading) alone.

Watching verbal production in a mirror has no effect on the accuracy of simple word production

- Imitative accuracy unless influenced by the test stimuli are better than spontaneous accuracy.

  Some patients improve if given more than one consecutive attempt at a production.

- Motivating instructions within very narrow limits, have no influence on articulatory accuracy.

- Response delay intervals of 0,3 and 6 seconds do not significantly influence articulatory accuracy.

- Binaural masking probably has no facilitating effect on articulation for most patients.

  DAP may have detrimental effect on articulatory accuracy.

B) PROSODIC CHARACTERISTICS

Traditionally observed prosodic characteristics in AOS (Rosenbek and Wertz, 1976) :

Tending toward equal stress.

Use of inappropriate intersyllabic pauses.

Restriction and alteration of normal intonational and loudness contours.

Effortful, groping, repetitive attempts to produce sounds accurately.

- Slow overall rate.

Fundamental frequency variations in Broca Aphasics have been reported as restricted in ranee for sentence level stimuli bv Ryalls (1982). but not for within word level stimuli by Danly and Shapiro (1982). Kent and Rosenbek (1983) report amplitude uniformity, temporal regularity, neutralization of stress pattern and dysrhythmia.

C) ACOUSTIC STUDIES

Earlier studies were based on wide band and narrow band spectrographic analysis. Blumstein (1981) concluded that anterior aphasic patients with AOS demonstrated impairments requiring fine inter-articulatory timing.

Kent and Rosenbek (1983) observed the following -

- Slow speaking rate with prolongation of transitions and steady states as well as inter-syllabic pauses.
- Restricted variations in relative peak intensity across syllables.
- Slow and inaccurate movements of the articulators to spatial targets for both consonants and vowels.
- Frequent mistiming or dysco-ordination of voicing with other articulatory movements.
- Occasional errors of segment selection or sequencing including intrusion, metathesis and omission.
- Imitation difficulties often characterised by false-starts and re-starts.
- Complex sound sequences associated with an apparent search for the intended targets.

Studies on voice onset time(VOT) (Blumstein, Cooper, Goodglass, Statlender and Gottlieb, 1980; Hoit- Dalgaard, Murry and Kopp, 1983) and nasal sounds (Itoh et al., 1979a; Itoh, Sasanuma, Hirose, Yoshioka and Ushijima, 1980) have also revealed abnormalities in apraxics.

McNeil and Kent (1990) summarize that between - group differences in vowel duration were generally not found when stimuli were monosyllables (Bauman, 1978; Duffy and Gawle, 1984, Gandour and Dardarananda, 1984; Ryalls, 1986). Vowels in multisyllabic words or nonsense utterances were shown to be significantly longer for apraxic than normals or Broca's aphasic subjects (Kent and Rosenbek, 1983). Consonant durations were found to be lengthened in AOS (Bauman, 1978; Kent and Rosenbek, 1983). Due to the above features speech rate was inferred to be reduced in apraxics (Kent and Mc Neil, 1987; Kent and Rosenbek, 1983).

D) PHYSIOLOGIC STUDIES

Shankweiler, Harris and Taylor (!968) reported presence of antagonistic muscle co-contraction in AOS. He also reported :

- Presence of continuous undifferentiated EMG activity.
- Instances of a shut down in muscle activity.
- Instances of movement without appropriate voicing.
- Dysco-ordinated, added and groping movements
- Reduced peak expiratory flow in some patients.

The above evidences are obtained from EMG data, movement devices etc.

Recent experiments with sophisticated equipment have evaluated abnormalities in paramaters like force and position control of articulators intra and inter-articulator kinematics (Itoh et al., 1980, Me Neil, Caliguiri and Rosenbek, 1989; Me Neil and Adams, 1990; Me Neil , Weismer, Adams and Mulligan, 1990; Hageman, Robin, Moon and Folkins, 1994)

E) NON SPEECH CHARACTERISTICS

i) Non- Verbal oral Apraxia (NVOA)

A substantial portion of patients with AOS exhibit NVOA (DeRenzi et al., 1966). However there is no one to one correspondence between the two. NVOA and speech errors may be dissociated in some patients. The fact that AOS and NVOA can occur independently argues against the notion that AOS is simp!y &

reflection of a more fundamental disturbance of non verbal oral movement(Wertz et al., 1984)

ii) Non-speech, non-oromotor and non-linguistic characteristics

Duffy (1995) lists these characteristics as - right hemiparesis or/and associated sensory deficits, Babinski' s sign, hyperactive stretch reflexes and limb apraxia which is usually bilateral.

There can however still be a doubt as to whether the errors are generated from motor or linguistic mechanisms, since many of the other features of AOS like dysprosody, effortfulness, limited verbal output, reduced rate, speed, are also characteristics of Broca's aphasia. Hence identification of AOS in a Broca's aphasic is a challenging task as both have many seemingly common features and in the presence of Broca's aphasia and apraxic difficulties, it becomes difficult to extract the apraxic errors.

There is scanty review however, which explains how the apraxic component can be separated from an underlying Broca's aphasia. This can probably be done based on descriptive and detailed qualitative analysis of AOS features in a Broca's aphasic.

The identification of apractic errors in Broca's aphasics is possible based on the qualitative analysis of speech features, and this view is supported by various investigators in the field. (Square-Storer et al., 1995; Duffy, 1995; Ballard et al., 1999)

Some of the characteristics that help in differentiating are as follows -

APRAXIA OF SPEECH	BROCA'S APHASIA
A) EFFORTFULNESS	
• Efforfulness reflects inability to program volitional movements (Duffy, 1995)	• Efforfulness reflects inefficient access to the phonological buffer(Lexical retrieval) (Schuell et al., 1964)
• Groping of articulators (Wertz et. al., 1984)	• Never described as groping but as slow, sluggish (Duffy, 1995)
• Successive attempts at target (McNeil, 1997)	• Few successive attempts towards target (Thompson, 1994).
B) ERROR TYPES	
• Frequent successsive attempts mostly at single sound level or numerous phonemic errors(Johns and Darley, 1970). In repeated trials substitutions may more difficult combinations of earlier ones (Rosenbek and Wertz, 1976)	• Errors/Corrections more at morphemic level (LaPointe, 1990, Thompson, 1994) • More pauses than attempts due to word retneval problem (Thompson, 10041
• Predominantly errors of substitutions or distortion at phonone level (Trost, 1970; Johns and Darley, 1970, Mc Neil et al, 1997;	• Predominantly agrammatic errors eg., omission/substitution of grammatical, morphemes and main verbs, misordering of words in a sentence, more use of only content words and omission of grammatical words. Semantic paraphasias eg.,

He was talking to the farm .....uh....er animals. This is probably due to wrong selection. If produced with motor fluidity, it is not an apractic error (Me Neil et al, 1990)

C) AUTOMATIC vs VOLITIONAL TASKS

- Discrepancy present between automatic and volitional tasks. Good with automatic tasks (counting - Normal forward, slow, backward) even with severe apraxia. Very poor with volitional tasks (Shankweiler and Harris, 1966: Johns and Darley, 1970)
- Discrepancy may not be very large. May be slow with automatic tasks, as well as poor with volitional tasks (LaPointe, 1969)

D) INITIATION OF SPEECH

- Have difficulty in initiation of speech most of the time(Johns and Darley, 1970)
- Spontaneous speech is effortful and halting. They spontaneously initiate communicative interaction (Kearns, 1990)

E) ORAL APRAXIA

If present, always implies AOS, whereas the converse may not be true (De Renzi et al., 1966). Hence presence of oral apraxia can be a feature more supportive of existence of apraxia of speech.

F) PROSODY

- Analysis at single word level indicates syllabic stress errors and more difficulty initiating than completing word(Odell et al., 1991)
- Equal stress placed on all syllables in an utterance (Rosenbek and Wertz, 1976)
- Reduced variations of pitch and loudness (Freed, 2000)
- Dysprosody is a perceptual phenomenon and does not exist in the speech sample as such. Due to halts and pauses there is perception of dysprosody (word + sentence level data) (Danly and Shapiro, 1982)

Based on the available review of literature it may be postulated that differentiating features for AOS and Broca's aphasia can be identified. These features could be sensitive to identify AOS in a given Broca's Aphasia Hov/ever there is no test or protocol available which is sensitive to identify apraxic features in Broca's aphasics. Development of a sensitive protocol could be an answer to resolve this issue. The present study aims to develop a protocol in Kannada and administer the protocol on cases with Broca's aphasia (with or without suspected **AOS)** to establish sensitivity of the protocol in identifying AOS in Broca's aphasics.

METHODOLOGY

Preamble:

Speech apraxia refers to disorders of verbal expression that lie on the border between motor speech disorders or dysarthrias, and language disorders or aphasias. A widely cited definition of Deal and Darley (1972) is, "apraxia is an articulatory disorder resulting from impairment, as a result of brain damage: of the capacity to program the positioning of speech musculature and the sequencing of muscle movements, for the volitional production of phonemes. The speech musculature does not show significant weakness, slowness or inco-ordination when used for reflexive and automatic acts".

The focus on apraxia of speech (AOS) by speech-language pathologists did not occur until the 1960's (Shankweiler and Harris, 1966). However, there was a subsequent increase of interest in the early 1970's (Johns and Darley, 1970; Aten, Johns and Darley, 1971; Deal and Darley, 1972; Halpern, Darley and Brown, 1973). Investigators have continued to demonstrate interests in this disorder to date. (Kent and Rosenbek, 1983; Wertz, LaPointe and Rosenbek, 1984; Kent and McNeil, 1987; McNeil and Kent, 1990: McNeil, 1997; Van der Merwe, 1997).

As apraxia is recognized as a motor speech disorder, the treatment calls for a set of goals in speech therapy which are distinct from the goals set for language treatment of expressive aphasias. The task of identifying the components of neurological impairment in expressive aphasias and verbal

apraxias when they co-occur, which they often do with high frequency in the adult patients, is both necessary and challenging.

Purpose:

There are few aphasia batteries which evaluate apraxia (eg., Western Aphasia Battery, Kertesz, 1982 and the Boston Diagnostic Aphasia Examination, Goodglass and Kaplan, 1983). However, most of these batteries focus on oral, non-verbal apraxia and limb apraxia.

Although there are several tests which are solely meant for the assessment of apraxia, very few of these have adequate psychometric properties and normative data (Haynes and Pindzola, 1998). Some of the tests available for evaluating AOS in adults are

1. *Test of oral and limb apraxia* (DeRenzi, Pieczuro and Vignolo, 1966)

2. *Oral apraxia test*, (includes items for oral apraxia and verbal subtests) (Darley, Aronson and Brown, 1975).

3. *Test of verbal, oral and limb apraxia* (includes a range of verbal tasks) (Rosenbek and Wertz, 1976).

4. *Apraxia Battery for adults* (ABA, Dabul, 1979) This test includes six subtests - DDK rate, increasing word length, limb apraxia, oral apraxia, latency and utterance time for polysyllabic words, repeated trials and an inventory of articulation characteristics of apraxia. This checklist of apraxic features is quantitatively scored to rate the severity of the patient's impairment.

5. *The Motor Speech evaluation* (Wertz, LaPointe and Rosenbelc, 1984). This test is used generally with any motor speech disorder. Tasks include conversation, vowel prolongation, rapid alternating movements, repetition of multi-syllabic words, repetition of words that increase in length, repetition of words that begin and end with the same phoneme, repetition of sentences, counting forward and backward, picture description and oral reading.

6. *Comprehensive Apraxia Test* (CAT, Disimoni, 1989). It is similar to the ABA in organization and standardization. It has a set of non verbal oral volitional tasks and speech tasks.

The above tests have various disadvantages with respect to evaluating AOS in aphasics.

The earlier tests evaluated oral and limb apraxias and not verbal apraxias. Evaluation of verbal performance is important because AOS and oral apraxia may exist independently. Oral apraxia is not always a necessary pre-requisite to AOS. Although a substaintial portion of patients with AOS exhibit oral apraxia, no one to one correspondence between the two has been found (DeRenzi, Pieczuro and Vignolo, 1966; Wertz, LaPointe and Rosenbek, 1984).

The Motor Speech evaluation by Wertz, LaPointe and Rosenbek (1984) does not test for oral apraxia. Moreover the tasks contain only 4 subtests with monosyllables (|a| prolongation and rapid alternating movements of |pa|, |ta|, |ka| singly and |pa-ta-ka| together, and 10-15 monosyllabic words). The other subtest consists of multi-syllabic words (eg., responsibility...etc) which may be

difficult for an aphasic. Hence bisyllabic words could have been an easier section following which words of increasing length or sentences, picture description etc (which form a major section of the test) could have been used.

The Apraxia Battery for Adults (ABA) by Dabul (1979) seems to take into consideration many of the above factors. However in ABA the emphasis has been more on quantitative descriptions rather than qualitative evaluation (eg., measuring latency time in naming and only quantitative scoring). Other short-comings include a lack of normative data and an absence of demonstrated inter-judge and intra-judge test-retest reliability, and measures of validity other than face validity. Similar limitations are also evident in the CAT (Comprehensive Apraxia Test by Disimoni, 1989).

Moreover, only the ABA and the CAT are published tests for AOS in adults. There are no standardized probes available in Kannada or other Indian languages. Specifically, there are no tests to identify AOS when it coexists with expressive aphasias. Hence there is a dire need for a protocol which can be used in clinics to identify the existence of AOS in a given expressive aphasic.

The reason for the lack of emphasis on identifying AOS in Broca's could be the issue of their co-occurrence and /or independent existence. They are not treated as separate clinical entities most of the time. The studies have often attempted to characterize AOS within the category of Broca's aphasia, under the assumption that most of the aphasic speech deficits are reflections of AOS rather than aphasic phonologic difficulties per se. (McNeil et al., 1997). In fact McNeil

and Kent (1990) argue that AOS may be an integral part of the syndrome of Broca's aphasia and that its presence may be required for the diagnosis of Broca's Aphasia.

The studies imply that AOS is synonymous with Broca's aphasia but this may not be true as most definitions of Broca's aphasia do not give overt recognition to the existence of an apraxic deficit nor can AOS explain the aphasic deficits.

The speech sound errors of a Broca's aphasic are not true reflections of speech sound errors of apraxia. As yet, there is no clinical tool available to identify the characteristics of AOS in a given Broca's aphasic case. The reason could be the lack of the clinical designs to study the performance of a large enough group of "pure" AOS subjects and comparison studies with larger group of AOS subjects associated with expressive language disorders. Cluster analysis or similar procedures could be employed to establish the patterns of behaviour that differentiate AOS from its clinical pathological neighbours (McNeil, 1997).

A brief review of the tests for AOS in adults throws light on the fact that there are certain essential features to be kept in mind during assessment of AOS in aphasics. A test to assess AOS in an aphasic needs to be simple enough so that performance is not contaminated by aphasic difficulties.

Instead of having elaborate tests with difficult words and many sentence level tasks, a protocol is essential which has more bisyllabic word level tasks and not only sentence level tasks (considering the aphasic difficulties). Many Broca's

aphasics may not have adequate verbal output to undergo such tests. More qualitative descriptions of error types (eg., groping behavior etc.), within these simpler items would become necessary to support the presence of AOS in cases especially having limited verbal output.

Here, AOS has been specifically discussed with respect to Broca's aphasics. This is because of two main reasons. First, though AOS could be present with any type of aphasia it most commonly occurs with Broca's aphasia. Secondly, identification of AOS when it co-occurs with Broca's aphasia, is very difficult as both these clinical entities affect expressive speech and many of their manifestations are very similar (eg., limited speech out-put, laboured slow speech, abnormal prosody etc.). Hence, identifying AOS in a case of Wernicke's aphasia or Conduction aphasia may be easy unlike that of Broca's aphasia where extracting apraxic features from the already existing expressive difficulty is very challenging.

Identification of AOS in expressive aphasics is possible based on certain qualitative rather than quantitative features. Qualitative observations enable the examiner to describe the apraxic features better and in a more elaborate manner. In this way , typical apraxic features may be differentiated from those errors resulting due to Broca's aphasia. Such an analysis becomes all the more essential in Broca's Aphasics with suspected AOS. This protocol developed in Kannada has qualitative and quantitative probes to identify AOS in Broca's aphasics.

METHOD

Test Background:

The protocol called as " Protocol to identify AOS in Broca's aphasics" is developed at the All India Institute of Speech and Hearing, Mysore.

It consists of five sub-tests :

A. Test for Oral Apraxia

B. Test for Effortfulness

C. Test for Error types

D. Test for Dysprosody

E. Test for Counting numbers (forward vs. backward)

Subject selection criteria

The five sub-tests in this protocol were administered to adult patients with Broca's aphasia. The subject sample included 2 Males and 3 Females. The age of the subjects ranged from 28 years to 59 years, the mean age being 48 years.

The criteria for selection of subjects were as follows :

- Subjects less than 80 years of age were considered for the study.
- Premorbidly all subjects were Kannada speakers and preferably monolinauals.

- The subjects were premorbidly right handed.
- All the subjects had been diagnosed as having Broca's Aphasia based on the performance on a standardized aphasia test battery (WAB-Western Aphasia Battery, by Kertesz, 1982) administered by a qualified Speech and Language Pathologist.
- In all the subjects aphasia was acquired after a single cerebro vascular accident (CVA)
- The testing was done three months post CVA
- The subject sample also included clients who were suspected to be presenting verbal apraxic features based on clinical observation (eg, visible or audible searching/groping, articulatory gestures, frequent attempts toward the target sound, difficulty initiating speech, dysprosody).
- Speech therapy availed earlier or duration of therapy was not considered as a confounding variable.
- The subjects had expressive speech of at least word level.
- The subjects did not have any gross sensori- motor signs such as paralysis or paresis of oral structures.
- Subjects having any other allied speech and language dysfunction or disorder were not included in the study.
- A l l the subjects had adequate hearing and visual acuity.

Instructions

Instructions for the tasks were appropriately framed for each task and are elaborated in the protocol.

Test environment

The test environment was a relatively quiet room, free from distractions or noises. There was adequate lighting and appropriate seating arrangements for the patient as well as the examiner.

Basis for scoring

All the responses of the subtests were video recorded using a video recorder (KEONICS M7 Video camera) for further reference and analysis. For future use of the protocol, audio and video recording of the verbal subtests is recommended.

Scoring system

A three point rating scale (0,1,2) was used throughout the sub-tests in the protocol testing for apraxic features. For each sub-test a descriptive account of 0,1,2 was given. Transcription of the responses on all verbal subtests was carried out for analysis of the errors. Qualitative description was also given by the examiner.

Total score of each subtest as well as the overall score was computed. The cut off criteria based on the test results as well as the qualitative descriptions aided the identification of AOS in the selected cases.

Task selection

The qualitative descriptions used in the protocol (of apractic errors) have been supported in literature by various investigators (Square-Storer et al, 1990; Duffy, 1995; Ballard et al., 1999). Some of them are as follows:

Oral apraxia if present always implies AOS whereas the converse may not be true (DeRenzi et al., 1966). Effortfulness, groping of articulators and successive attempts at target reflect inability to program volitional movements (Duffy, 1995; McNeil, 1997). The error types in AOS are characterized by frequent successive attempts mostly at single sound level, substitutions which are more difficult combinations of earlier ones and errors are predominantly those of substitution or distortion (Johns and Darley, 1970; Trost, 1970, and McNeil, 1997). Analysis at single word level indicates syllabic stress errors and more difficulty initiating than completing a word (Odell et al., 1991). Equal stress on all syllables in an utterance and reduced variations of pitch and loudness have also been reported (Freed, 2000). Another prominent feature of AOS is discrepancy present between automatic and volitional tasks. The verbal apraxics are good in automatic tasks and very poor in volitional tasks (Shankweiler and Harris, 1966, LaPointe, 1969; Johns and Darley, 1970)

Tasks were framed on the lines of the observations made in various studies. However more scope for qualitative description was allowed and more weightage given to the same. The examiner was required to comment and note all audible, visible or other features observed and elaborate on the type of errors.

Protocol to identify Apraxia of speech in Broca's aphasics

Demographic data of the subjects included in the study:

	S1	S2	S3	S4
Age/Sex				
Pre-morbid Occupation				
Age at CVA				
Type of CVA				
Post CVA duration				
Post morbid change, if any				
Therapy undergone with duration				
Speech profile Phonatlon Resonat lon Articulation Prosody OSME				
Language profile Comprehension Expression WAB results Spontaneous speech Repetition Naming Auditory verbal comprehension Reading Writing Praxis AQ Diagnosis				

The protocol is aimed at identifying AOS in cases of Broca's aphasia. The five subtests of the protocol consist of test items that are sensitive to identify AOS in Broca's aphasics. The test items are on a continuum of simple to complex items. More weightage is given for qualitative descriptions.

The five subtests are :

1. Test for Oral apraxia
2. Test for Effortfulness
3. Test for Error types
4. Test for Dysprosody
5. Test for Counting (forward vs backward)

TEST ADMINISTRATION

A) TEST FOR ORAL APRAXIA :

Instructions -

"I would like you to show me how you do certain activities"

"ನಾನು ಹೇಳಿದ ಕೆಲವು ಚಟುವಟಿಕೆಗಳನ್ನು ನೀವು ಪುನಃ ಮಾಡಿ ತೋರಿಸಿ".

"naanu heeLida kelavu chaTuvaTikegaLannu niivu punaha maaDi torisi".

(Give auditory verbal commands. If the subject fails to understand ask him or her to imitate after you).

Scoring:

0 Subject performs the task correctly. There is absence of any effort, groping or struggle behaviour of articulators. There is ease of production and motor fluidity in the specified task. The transitions between articulatory positions are smooth. Sometimes exhibits word finding difficulties and poor articulatory control.

1 While performing the task the subject demonstrates laboured movement, effortfulness and groping. The appropriate target gesture is reached after groping and searching for the correct gesture with the articulator.

and/or

Subject performs the task on repeated attempts

and/or

Subject performs the task on imitation after the examiner

2 There may be groping, effortful, audible or visible searching behaviour with no success. The subject is unable to perform the gesture.

and / or

Subject is unable to imitate after the examiner

and / or

There is increasing struggle behaviour.

Stimuli	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle behaviour	
1.Take a deep breath and hold ಗಟ್ಟಿಯಾಗಿ ಉಸಿರು ತಗೊಂಡು ಅದನ್ನ ಹಾಗೆ ಇಟ್ಟುಕೊಳ್ಳಿ gaTTiyaagi usiru tagonDu adanna haage iTTukolli						
2. Open your mouth ಬಾಯಿ ತೆಗಯಿರಿ baayi tegiyiri						
3. Clear your throat ಗಂಟಲನ್ನು ಸ್ವಚ್ಚ ಪಡಿಸಿಕೊಳ್ಳಿ ganTaLannu svachcha paDisikoLLi						
4. Smile ಮುಗುಳ್‌ನಗ ಬೀರಿ muguLnage biiri						

5. Puff your cheeks ಕೆನ್ನೆ ಊದಿಸಿ ಬಾಯಿಯಲ್ಲಿ ಗಾಳಿ ತುಂಬಿಕೊಳ್ಳಿ kenne uudisi baaiyalli gaaLi tumbikoLLi						
6. Puff cheeks – release – puff cheeks ಕೆನ್ನೆ ಊದಿಸಿ ಬಾಯಿಯಲ್ಲಿ ಗಾಳಿ ತುಂಬಿಕೊಳ್ಳಿ, ಬಿಡಿ, ತುಂಬಿಕೊಳ್ಳಿ ಬಿಡಿ kenne uudisi baayalli gaaLi tumbikoLLi, biDi,tumbikoLLi, biDi…						
7. Show me your teeth ನಿಮ್ಮ ಹಲ್ಲು ತೋರಿಸಿ nimma hallu torisi						
8. Click teeth together ಹಲ್ಲು ಕಡಿಯಿರಿ hallu kachchi						
9. Lick your lips with the tongue . ತುಟಿಗಳನ್ನು ನಾಲಿಗೆಯಿಂದ ನೆಕ್ಕಿ tuTigaLannu naligeyinda nekki						
10. Bite lower lip with the teeth ಕೆಳ ತುಟಿಯನ್ನು ಹಲ್ಲಿನಿಂದ ಕಡಿಯಿರಿ keLa tuTiyannu hallininda kaDiyiri						
11. Pucker lips ತುಟಿಯನ್ನು ಮುಂದ ಮಾಡಿ tuTiyannu munde maaDi						

12. Retract lips – pucker lips – retract lips ತುಟಿಯನ್ನು ಹಿಗ್ಗಿಸಿ – ತುಟಿಯನ್ನು ಕುಗ್ಗಿಸಿ – ತುಟಿಯನ್ನು ಹಿಗ್ಗಿಸಿ tuTiyannu higgisi – tuTiyannu kuggisi – tuTiyannu higgisi						
13. Move tongue left – right-touch upper lips ನಾಲಿಗೆಯನ್ನು ಎಡಗಡೆ ಬಲಗಡೆ ಅಲ್ಲಾಡಿಸಿ ನಂತರ ಮೇಲಿನ ತುಟಿಯನ್ನು ಮುಟ್ಟಿ naaligeyannu eDagaDe, balagaDe allaaDisi, nantara meelina tuTiyannu muTTi						
14. Retract corner of the lip to left-right-left ತುಟಿಯಂಚನ್ನು ಎಡಕ್ಕೆ ಬಲಕ್ಕ ಎಡಕ್ಕೆ ಮಾಡಿ tuTiyanchannu eDakke ondu saari-balakke ondu saari maaDi						
15. Stick out your tongue ನಾಲಿಗೆಯನ್ನು ಹೊರಗ ತೋರಿಸಿ naaligeyannu horage torisi						
16. Move tongue from side to side ನಾಲಿಗೆಯನ್ನು ಅಕ್ಕ ಪಕ್ಕ ಅಲ್ಲಾಡಿಸಿ naaligeyannu akka pakka allaaDisi						

17. Touch upper lip with tongue ಮೇಲಿನ ತುಟಿಯನ್ನು ನಾಲಿಗೆಯಿಂದ ಮುಟ್ಟಿ meelina tuTiyannu naaligeyinda muTTi						
18. Place tongue on inside surface of cheek ಕೆನ್ನೆಯ ಬಲಬಾಗವನ್ನು ನಾಲಿಗೆಯಿಂದ ಮುಟ್ಟಿ kenneya oLabhaagavannu naaligeyinda muTTi						
19. Touch roof of the mouth with tongue ನಾಲಿಗೆಯಿಂದ ಅಂಗಳನ್ನು ಮುಟ್ಟಿ naaligeyinda angaLannu muTTi						
20. Place tongue between front teeth ನಾಲಿಗೆಯನ್ನು ಮುಂದಿನ ಹಲ್ಲುಗಳ ಮಧ್ಯದಲ್ಲಿ ಇಡಿ naaligeyannu mundina hallugaLa madhyadalli iDi						
Patient's score Maximum error score	40					

Key : If there is no apraxic component all the above tasks will be performed with motor fluidity in all the Broca's aphasics even if there is an initiation delay or slowness. In the presence of apraxia, groping searching behaviour will be evident in the imitation as well as production. Sequencing will also be affected. Subject may also show frustration or react by a head shake or sigh (implying that, though he/she knows what has to be done he/she is unable to do it). Usually , AOS is highly probable if oral apraxia is observed.

Examiner's comments and notes on behavioural observation :

(B) TEST FOR EFFORTFULNESS :

Scoring:

0 Subject performs the task correctly. There is absence of any effort groping or struggle behaviour of articulators. There is ease of production and motor fluidity in the specified task. The transitions between articulatory positions are smooth. Sometimes exhibits word finding difficulties and poor articulatory control.

1 While performing the task the subject demonstrates laboured movement, effortfulness and groping. The appropriate target gesture is reached after groping and searching for the correct gesture with the articulator.

and/or

Subject performs the task on repeated attempts

and/or

Subject performs the task on imitation after the examiner

2 There may be groping, effortful, audible or visible searching behaviour with no success. The subject is unable to perform the gesture.

and/or

Subject is unable to imitate after the examiner

and/ or

There is increasing struggle behaviour.

Types of error patterns in AOS :

- Initiation difficulty
- False-starts/ re-starts for same consonant repetition
- Visible groping or searching behaviour
- Audible searching or groping 5behaviour
- Successive attempts at target
- Off-target responses
- False-starts/re-starts for /paTaka/ combination
- Sequencing error for /paTaka/
- Inability to increase rate while maintaining phonemic integrity
- On multiple trials or as the task proceeds, with overlearning, producions improve in terms of motor fluidity.

Tasks :

1) Vowel Prolongation

Instructions	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle behaviour	
a) Say /a/ as long as you can /ಅ/ ಅಂತ ಎಷ್ಟು ಹೊತ್ತು ಆಗುತ್ತೋ ಆಷ್ಟು ಹೊತ್ತು ಹೇಳಿ /a/ anta eshTu hottu aagutto ashTu hottu heeLi						
b) Similarly say /i/ as long as you can /ಇ/ ಅಂತ ಎಷ್ಟು ಹೊತ್ತು ಆಗುತ್ತೋ ಆಷ್ಟು ಹೊತ್ತು ಹೇಳಿ adeetara /i/ anta eshTu hottu aagutto ashTu hottu heeLi						
c) Similarly say /u/ as long as you can /ಉ/ ಅಂತ ಎಷ್ಟು ಹೊತ್ತು ಆಗುತ್ತೋ ಆಷ್ಟು ಹೊತ್ತು ಹೇಳಿ adeetara /u/ anta eshTu hottu aagutto ashTu hottu heeLi						
Patient's score						
Maximum error score	6					

Key : Lack of phonation may imply apraxia of phonation. The lack of voicing may be accompanied by gestures such as oral structure approximations, finger / hand gestures on the throat, silent mouth opening etc.

Examiner's comments and notes on behavioural observation :

2) DDK

Instructions	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle behaviour	
a) Say pa,pa,pa..... ಪ ಪ ಪ.....ಅಂತ ಹೇಳಿ pa, pa, pa ....anta heeLi						
b) Say Ta,Ta,Ta..... ತ ತ ತ .....ಅಂತ ಹೇಳಿ Ta,Ta,Ta.... anta heeLi						
c) Say ka, ka, ka ..... ಕ ಕ ಕ.....ಅಂತ ಹೇಳಿ ka, ka, ka ..... anta heeLi						
d) Say Pa, Ta, Ka, Pa, Ta, Ka,...." ಪ ಟ ಕ ಪ ಟ ಕ.....ಅಂತ ಹೇಳಿ pa Ta ka, pa Ta ka... anta heeLi						
e) Say pa,Ta,ka, Pa,Ta,ka as fast as you can ಪ ಟ ಕ ಪ ಟ ಕ.....ಅಂತ ಎಷ್ಟು ಬೇಗ ಆಗುತ್ತೊ ಆಷ್ಟು ಬೇಗ ಹೇಳಿ paTaka, paTaka....anta yeshTu beega aagutto ashTu beega heeLi						
Patient's score						
Maximum error score	10					

Key : For same consonant repetition, initiation difficulty, false starts, re-starts or groping behaviour may be seen; but once started, the activity may proceed effortlessly in the apraxic due to good abilities at automaticity. With respect to successive attempts audible or inaudible, simple or complex sound sequences associated with an apparent search for intended target may be seen. These may lead to off target responses which are usually in close approximation to the target response. Sequencing of paTaka may be disordered. There may be improved effortless production of paTaka on multiple trials due to automaticity.

In the absence of AOS, the Broca's aphasics should not exhibit any of the above except slowness, delay in initiation or inability to increase rate. They will have more pauses than attempts. Self corrections are not frequent in Broca's aphasics in comparison with that of apraxics. In Broca's aphasia sequencing may not be a problem but there may be substitution errors in the phonemes and hence the order may be incorrect.

Examiner's comments and notes on behavioural observation :

Note: Key of features for the following tasks

3) Words

4) Words increasing in length

5) Sentences

- Initiation difficulty
- Visible groping or searching behaviour
- Audible groping or searching behaviour
- Successive attempts at target, mostly at phoneme level
- Sequencing of phonemes disordered in the word
- More difficulty initiating than completing the task
- Instances of articulatory movement without voicing
- Inconsistency on multiple trials (describe the type of inconsistencies in remarks).
- Improvement with multiple trials or as the task proceeds. With overlearning, productions improve in terms of motor fluidity.

3) Words :

Instructions :

"I am going to show you pictures of some objects which we use daily. Name the object in the picture "

"ನಾನು ಕೆಲವು ವಸ್ತುಗಳ ಚಿತ್ರಗಳನ್ನು ನಿಮಗ ತೋರಿಸುತ್ತೇನೆ. ನೀವು ಚಿತ್ರದಲ್ಲಿರುವ ವಸ್ತುವನ್ನು ಹೆಸರಿಸಿ"

"naanu keLavu vastugaLa chitragaLannu nimage torisuttene. niivu chitradalliruva vastuvannu hesarisi".

(If client is unable to name, ask him/ her to repeat after you. Give multiple trials with the same words)

Note : These words were framed mainly to include the sound classes such as affricates, fricatives and sounds with increasing feature differences. These are generally reported to be difficult for apraxics.

Sounds which are judged easier for apraxics ie., bilabials, alveolars and velars are also included to look for differential performance of the patients. The lexical meaning of these words in English is provided in parenthesis.

Picture size - 15 X 14 cms

Stimuli	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle behaviour	
a) (veena) ವೀಣೆ viiNe						
b) (flag) ಬಾವುಟ baavuTa						
c) (sea-shell) ಶಂಖ shankha						
d) (brush) ಬ್ರಷು brashu						
e) (Spoon) ಚಮಚ chamacha						
f) (Chair) ಕುರ್ಚಿ kurchi						
g) (plait) ಜಡೆ jaDe						
h) (king) ರಾಜ raaja						
i) (bucket) ಬಕೆಟು bakiTu						
j) (lock) ಬೀಗ biiga						
k) (pen) ಪೆನ್ನು pennu						

l) (glass) ಲೋಟ loTa						
m) (flower) ಹೂವು huuvu						
n) (umbrella) ಛತ್ರಿ chatri						
o) (car) ಕಾರು kaaru						
Patient's score						
Maximum error score	30					

Key : Apraxic speakers may state "I know it but I just can't say it", or show the same intention through facial expression. Such comments may be free from many of the articulatory difficulties which may be present in the test items. Many a times awareness of errors also does not appear to help in anticipating or correcting the difficulty.

A Broca's aphasic without AOS will not present with the above difficulties except for a slow, sluggish initiation. Successive attempts are mostly a reflection of word finding difficulty. Hence attempts are more at word level and not phoneme level. Speech sound errors will be consistent across all productions.

Examiner's comments and notes on behavioural observation :

4) Repetition of words that increase in length :

Instructions

" Please repeat the following sequence of words"

"ನಾನು ಹೇಳುವ ಕೆಲವು ಪದಗಳನ್ನು ಪುನಃ ಹೇಳಿ ".

"naanu heeLuva kelavu padagaLannu punaha heeLi".

Note : The meaning of the sentences is provided in parenthesis.

Words	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle	
(see) noDu ನೋಡು (he will see) noDuttaane ನೋಡುತ್ತಾನೆ (he is seeing) noDuttaaiddane ನೋಡುತ್ತಾ ಇದ್ದಾನೆ						
(house) mane ಮನೆ (houses) manegaLu ಮನೆಗಳು (from the houses) manegaLinda ಮನೆಗಳಿಂದ						
(tree) mara ಮರ (of the tree) marada ಮರದ (from the tree) maradinda ಮರದಿಂದ						
(speech) maatu ಮಾತು (of speech) maatina ಮಾತಿನ (from speech) maatininda ಮಾತಿನಿಂದ						
(girl) huDugi ಹುಡುಗಿ (girls) huDugiyaru ಹುಡುಗಿಯರು (from girls) huDugiyarinda ಹುಡುಗಿಯರಿಂದ						
Patient's score						
Maximum error score	30					

Key : A Broca's aphasic without AOS will show errors which are consistent across all these words. An apraxic may exhibit errors only on longer words reflecting difficulty in making too many articulatory adjustments, whereas the same sound may be produced correctly elsewhere or on the shortest word.

Examiner's comments and notes on behavioural observation :

5) Repetition of Sentences

Instructions

"Please repeat the sentences I say"

''ನಾನು ಹೇಳುವ ಕೆಲವು ವಾಕ್ಯಗಳನ್ನು ಪುನಃ ಹೇಳಿ ''.

"naanu heeLuva kelavu vaakyagaLannu punaha heeLi".

(Note ; The meaning of the sentences is provided in parenthesis. The examiner should provide the model of the sentence as a whole and not feed the words in the sentence one by one).

Sentences	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle behaviour	
(the boy is going) ಹುಡುಗ ಹೋಗ್ತಾ ಇದ್ದಾನೆ huDuga hogta iddaane						
(he is eating) ಅವನು ಊಟ ಮಾಡುತ್ತಾ ಇದ್ದಾನೆ avanu uuTa maaDuta iddaane						
(the lock is near the door) ಬೀಗ ಬಾಗಿಲಿನ ಹತ್ತಿರ ಇದೆ biiga baagilina hattira ide						
(she will come home, from the school) ಅವಳು ಶಾಲೆಯಿಂದ ಮನೆಗೆ ಬರುತ್ತಾಳೆ avaLu shaaleyinda manege bartaaLe						
(he is going with an umbralla ಅವನು ಛತ್ರಿ ತಗೊಂಡು ಹೋಗುತ್ತಿದ್ದಾನೆ avanu chatri tagonDu hogta iddaane						
Patient`s score Maximum error score	10					

Key : In Broca's aphasia without AOS, effortfulness is usually in the form of sluggishness in the verbal performance or slowness in the overall articulatory gesture or word finding difficulty. Hence there are self corrections and successive attempts. Successive attempts are more at word level and not at phoneme level. When AOS is present, the typical groping, searching behaviour of the articulation will accompany all the verbalization attempts.

Examiners comments and notes on behavioural observation :

6) General conversation

Instructions :

"I will ask you a few questions please answer for the same".

"ಈಗ ನಾನು ಕೆಲವು ಪ್ರಶ್ನೆಗಳನ್ನು ಕೇಳ್ತೀನಿ, ಅದನ್ನು ಪೂರ್ತಿ ವಾಕ್ಯದಲ್ಲಿ ಉತ್ತರಿಸಿ".

"iiga naanu kelavu prashNegaLanna keeLtiini adakke puurti vaakyadalli uttarisi".

(Note ; The examiner should try to elicit complete sentences as answers. If patient persists to answer in single words the examiner should accept these and look for the apractic errors, if any within the response given by the client).

Conversation	Rating					Remarks
	0	1		2		
		Repeated attempts	Imitation	Repeated attempts	Increasing struggle	
(What Is your name)? ನಿಮ್ಮ ಹೆಸರು ಏನು ? Nimma hesaru yeenu?						
(Where is your house)? ನಿಮ್ಮ ಮನೆ ಎಲ್ಲಿದೆ ? Nimma mane ellide?						
(Who all are there at your home)? ನಿಮ್ಮ ಮನೆಯಲ್ಲಿ ಯಾರು ಯಾರು ಇದ್ದಾರೆ ? nimma maneyalli yaaru yaaru iddaare?						
(Which is your home- town)? ನಿಮ್ಮ ಊರು ಯಾವುದು ? Nimma uuru yaavudu?						
(What do you do at home)? ನಿಮ್ಮ ಮನೆಯಲ್ಲಿ ಏನು ಮಾಡುತ್ತೀರ ? niivu maneyalli eenu maaDuttira?						
Patient's score Maximum error score	10					

Key : All the apraxic features listed before the section 3) will be evident in Broca's aphasic patients with AOS.

Examiner's comments and notes on behavioural observation :

7) Spontaneous Speech

Instruction :

"Speak on a topic of your interest for sometime"

"ಈಗ ನಿಮಗ ಇಷ್ಟವಾದ ಯಾವುದಾದರು ಒಂದು ವಿಷಯದ ಬಗ್ಗ ಸ್ವಲ್ಪ ಹೊತ್ತು ಮಾತನಾಡಿ".

"iiga nimage ishTavaada yavudaadaru ondu vishayada bagge svalpa hottu maatanaaDi".

(Note : To facilitate spontaneous speech the examiner may prompt with lead questions, provide semantic or phonemic cues, help in topic selection etc. However, while scoring for these spontaneous responses the examiner should look for apraxic errors if any and not for fluent language abilities. Attempt should

be made wherever possible to obtain a speech sample for a minimum of one minute).

Scoring:

0 Client is able to produce thirty fluent words or more in one minute. The words should be fluent and should not have

a) initiation difficulty

b) groping (audible or visible)

c) off-target responses

d) sequencing error within the words

e) inconsistency on the same words when used repeatedly

f) phoneme errors in the words which are inconsistent in nature

g) articulatory movements without voicing.

1 The client has limited verbal output which is approximately less than thirty words per minute and/or the client exhibits the features listed above (a to g) in a mild form on different words in the spontaneous speech sample. Each of these errors should be scored as 1 and the total composite score would consist of the grand total of these errors.

2 The client exhibits the features listed above (a to g) in a severe form or more frequently. This should be judged qualitatively by the examiner keeping in mind the intensity and frequency of the errors.

Note: If the subject does not produce any spontaneous speech this section should not be considered for scoring.

Patient's error score :

Key : In a pure Broca's aphasic though speech is halting they typically spontaneously initiate communication interaction. Their productions will be with motor fluidity and they will have more pauses unlike in apraxics where there are more attempts.

C) ERROR TYPES :

Note : Evaluate the following from recorded tasks in B

Scoring :

0 specified behaviour not observed

1 specified behaviour is observed in a mild form or less frequently

2 specified behaviour is severe or observed more frequently

Features	Rating			Remarks
	0	1	2	
a) In repeated trials substitution may be more difficult combinations of target sound or sounds which were substituted earlier by the patient b) Substitution, omission, distortion or additions present (specify which ones in comments) c) Schwa vowel between syllables and between difficult consonant clusters d) Difficulty on clusters e) Pre-positioning or anticipatory errors f) Post - positioning errors g) Metathetic errors h) Discrepancy in performance between spontaneous speech, conversation vs initiation of words or sentences i) Phonemic perseveration j) Numerous and varied off target attempts of phonemes and words.				
Patient's score				
Maximum error score	20			

Key : If there is no AOS, errors will always be those of simplification. If AOS is present there will be predominantly phoneme level substitutions or distortions which need to be extracted from the already existing agrammatic speech. Discrepancy may be present in performance between spontaneous speech and imitation tasks. Typically errors due to AOS may be inconsistent. An articulatory production or sound may be correct in one context and affected in another (usually correct in informal/spontaneous speech and affect in voluntary imitation or repetition tasks).

D) PROSODY

Note : Evaluate the following from recorded tasks in B.

Scoring :

0 Specified behaviour not observed

1 Specified behaviour is observed in a mild form or less frequently

2 Specified behaviour is severe or observed more frequently.

Features	**Rating**			**Remarks**
	0	**1**	**2**	
a) Slow, even rate b) Equal stress on all syllables. c) Reduced pitch and loudness variations d) Inappropriate inter-syllabic pauses e) Even spacing of speech units (syllables or morphemes).				
Patient's score Maximum error score	10			

Key : Reduced pitch, loudness and stress variations within word are characteristics of AOS. In case of Broca's aphasia without perception of dysprosody at sentence level and not within words.

E) TEST FOR COUNTING NUMBERS (Forward and Backward);

Scoring

0 The client is fluent in forward and backward counting

1 The client is fluent in forward but presents mild to moderate difficulty backward counting

2 The client is fluent in forward and unable to perform backward counting

Instructions :

"Count forward from one to ten, and then backward from ten to one".

"ನೀವು ಒಂದರಿಂದ ಹತ್ತರವರಗೆ ಎಣಿಸಿ. ನಂತರ ಹತ್ತರಿಂದ ಒಂದರವರಗೆ ಎಣಿಸಿ".

"niivu ondarinda hattaravarege eNisi. nantara hattarinda ondaravarege eNisi".

Key : In AOS the automatic abilities may be with articulatory inaccuracy but without groping or effort. In Broca's aphasics with AOS patient performs better in forward counting but demonstrates poor abilities in backward counting. In a pure Broca's aphasic both these may be equally poor.

Patient's score

Maximum error score 2

SCORE SHEET

Subtests	Maximum error score	Patient score
A) Test for Oral apraxia	40	
B) Test for Effortfulness		
1) Vowel prolongation	6	
2) DDK	10	
3) Words	30	
4) Repetition of words that increases in length	30	
5) Repetition of sentences	10	
6) General conversation	10	
7) Spontaneous speech		
C) Test **for Error types**	20	
D) Test for Dysprosody	10	
E) Test for Counting numbers (forward vs backward).	2	
TOTAL	168	

SCORE SHEET

Subtests	Max. error .score	Client 1	Client 2	Client	Client 4	Client 5
A) Test for oral apraxia	40	12	30	24	5	7
B) Test for effortfulness						
1) Vowel prolongation	6	0	1	0	0	0
2) DDK	10	1	0	0	1	1
3) Words	.30	8		13	0	0
4) Repetition of words that increases in . length	30	5	0	0	0	0
5) Repetition of sentences	10	6	0	4	0	0
6) General conversation	10	4	0	0	0	0
7) Spontaneous speech	-	40	0	0	0	0
C) Test for error types	20	9	2	2	2	1
D) Test for dysprosody	10	10	0	0	1	0
E) Test for counting numbers (forward vs. backward.	2	1	1	2	0	1
TOTAL	168	96	37	45	9	10

Demographic data of the subjects included in the study:

	S1	S2
Age/Sex	28 yrs/F	58yrs/M
Pre-morbid Occupation	House wife	Head Master
Age at CVA	25yrs	56 yrs
Type of CVA	Left MCA aneurysm	Left MCA infarct
Post CVA duration	3 yrs 5 months	lyr 6 months
Post morbid change, if any	Left handedness	-
Therapy undergone with duration	- Speech therapy - 9 mo. Physiotherapy - still under going	Speech therapy- 1 week
Speech profile		
Phonation	Normal	Normal
Resonatlon	Normal	Normal
Articulation	Normal	Normal
Prosody	Abnormal	Normal
OSME	Structure - Normal Function - ? oral apraxia	Structure - Normal Function -Reduced range of movement, raising of tongue affected

Language profile		
Comprehension	Good for even complex utterances	Good
Expression	2-3 word sentences	3-4 word sentences
WAB results		
Spontaneous speech	11	: 8
Repetition	5.4	2
Naming	6.5	2.3
Auditory verbal comprehension	7.4	7.3
Reading		
Writing	-	
Praxis		
AQ	60.6	39.2
Diagnosis	Broca's aphasia	Broca's aphasia

Demographic data of the subjects included in the study:

	S3	S4
Age/Sex	52yrs/F	59yrs/M
Pre-morbid Occupation	House wife	Retd. DC. Officer
Age at CVA	52yrs	58yrs
Type of CVA	Left MCA infarct	Left MCA territory and left parietal lobe infarct
Post CVA duration	5 months	5 months
Post morbid change, if any	-	Left handedness
Therapy undergone with duration	Speech therapy - 1 week Physiotherapy - for 2 months after stroke	Speech therapy- once a week since two months - Physiotherapy - for 2 months after stroke
Speech profile		
Phonation	Normal	High pitched hoarse voice
Resonation	Normal	Normal
Articulation	Normal	Distortion/ substitution of all plosives
Prosody	Normal	Slightly affected
OSME	Structure - Normal Function - Lip retraction restricted to the right	Structure - Normal Function -Normal

Language profile		
Comprehension	Adequate	Good
Expression	1-2 word sentences	2-3 word sentences
WAB results		
Spontaneous speech	8	7
Repetition	6.8	4.1
Naming	2.5	6.5
Auditory verbal comprehension	5.9	9.85
Reading		
Writing	–	
Praxis	–	
AQ	46.4	54.90
Diagnosis	Broca's aphasia	Broca's aphasia

Demographic data of the subjects included in the study:

	S5
Age/Sex	43 vrs/F
Pre-morbid Occupation	House wife
Age at CVA	42 yrs
Type of CVA	Left MCA thrombosis
Post CVA duration	6 months
Post morbid change, if any	Left handedness
Therapy undergone with duration	Speech therapy - 1 week Physiotherapy - Undergoing, since 5 months
Speech profile	
Phonation	Normal
Resonat ion	Normal
Articulation	Normal
Prosody	Normal
OSME	Structure - Normal Function - Normal
Language profile	
Comprehension	Good
Expression	3-4 word sentences
WAB results	
Spontaneous speech	9
Repetition	3.8
Naming	4.0
Auditory verbal comprehension	8.9
Reading	-
Writing	-
Praxis	-
AQ	51.4
Diagnosis	Broca's aphasia

RESULTS AND DISCUSSION

The "Protocol to identify AOS in Broca's aphasics" was administered to five subjects with the diagnosis of Broca's aphasia. The quantitative data of the scores obtained by all the subjects on each subtest is shown on the score sheet.

Using the protocol one of the five clients (Client no.l in the score sheet) is identified as definitely having apraxia of speech with an accompanying oral apraxia. Two of the remaining four clients (Client no, 2 and 3 in the score sheet) present features of oral apraxia. The remaining two clients (Client no. 4 and 5 in score sheet) reveal no observable features of apraxia on the protocol. No attempt is made to establish a quantitative cut-off score which aids in diagnostic conclusions because the protocol needs to be administered on more number of clients to establish the cut-off score.

This pilot study is aimed to find out whether the protocol items are sensitive in identifying apraxic features in a given case of Broca's aphasia. The inference as to the performance of the clients are based to some extent on the quantitative scores obtained on the protocol but by and large on qualitative descriptions of each of the cases. Based on the three categories under which the clients evaluated lie, descriptive and inferential methods have been used to discuss whv and how each of the clients have been classified.

CATEGORY 1. Broca's aphasia with AOS and oral apraxia

Client 1 (a 26 year old female) was identified as having AOS as well as oral apraxia.

On the oral apraxia subtask the client scored 12/40. Apraxia of the oral structures was evident based on the features such as performance by trial and error, inaudible groping of the tongue or occassionally the lips. There was lack of motor fluidity on at least 50% of the tasks in this section. Task number 19 (touch roof of the mouth with tongue) was highly sensitive in eliciting the typical apraxic groping behaviour. These features have been described as key features of oral apraxia by various investigators (Wertz, et al., 1984; Duffy, 1995).

The speech sub-tasks ranged from vowel prolongations to conversational tasks. The performance of client 1 on vowel prolongation was smooth, effortless and continuous with an error score of 0/6. On the DDK task, initially the client was unable to perform rhythmic repetitions of /pa/. Inspite of realizing that her productions were wrong, she was unable to repeat correctly. Once automaticity improved, productions also improved. These features of inability to correct self (with awareness of errors) and improved abilities at automaticity are characteristic of AOS (Johns and Darley, 1970, Kent and Rosenbek 1983). Overall productions on the DDK were slow. There were however no sequencing errors. On faster DDK performance the client was able to increase rate only to a certain extent. The DDK error score was 1/10.

Features of AOS were evident on the word level tasks. There were many

errors of silent posturing for forthcoming words, groping of articulators for correct position, unnecessary pauses within words and vowel prolongations. Inconsistencies of errors were present from trial to trial. These features are also reported by Rosenbek and Wenz (1976) and McNeil (1997) as characteristics of AOS. The sound /ch/ was more difficult in terms of production than other sounds. This coincides with the finding in a study by Shankweiler and Harris (1966). Word finding difficulties were few and when present, occurred on words with low frequency of occurrence like "shankha". The difficulty experienced in expressing the other words could not be attributed to word finding difficulty as she was attempting all the words and not just pausing to recall them.

On words increasing in length apraxics usually exhibit increase in struggle behaviours (Johns and Darley, 1970; Deal and Darley, 1972, Dabul, 1979; Nespoulous, Lecours and Deloche, 1981). In the client being discussed there was difficulty on longer words along with increasing difficulty in articulatory adjustments. As the examiner presented the word, the client was simultaneously executing appropriate smooth (silent) articulatory gestures. However, when the client actually tried to produce the words, articulatory breakdown was noticed. Numerous intersyllabic pauses which were observed in the client's speech is another characteristic feature of AOS reported by DeRenzi et al. (1966), Rosenbek and Wertz (1976) and Odell et al. (1991).

On repetition of meaningful sentences the client had more difficulty with longer sentences. Articulatory groping and silent posturing were the prominent

features. There were numerous intersyllabic pauses. Motor fluidity was strikingly better in this task compared to the task of repetition of words with increasing length. The improved semanticity in the stimuli could have facilitated better performance. The task on repetition of words with increasing length involved repeating each word after the examiner. However, when the three words were linked together the client could not produce the string correctly unlike the sentences which were produced correctly. It is interesting to note that sentences with more linguistic and semantic load were uttered more easily than words increasing in length, inspite of the fact that the words were phonetically similar. Often motor programming is expected to be easier for words which are phonetically similar. In the client being discussed the similarity effect on words increasing in length (noDu-noDuttaane-noDutiddaane) did not facilitate production as compared to production of sentences. This is in contradiction to the phonological output buffer theory by Rogers and Storkel (1998) which states that AOS patients cannot program two different words or syllables at a time (which as characteristic of the sentences).

Spontaneous speech was very poor in the client. It may be presumed that asking the client to "speak on any topic" presented itself as a more volitional task and hence, was found to be more difficult. Leading questions were required for all tasks in the general conversation. Ideally speaking, a spontaneous sample should never be an "elicited" one, but should be recorded as and when uttered by a client. This client had few spontaneous utterances. Hence, elicited speech had to be obtained. Imitation is believed to be more difficult for apraxics than

spontaneous speech according to Darley (1969) and Johns and Darley (1970). However, in the present study imitation and repetition were better than spontaneous speech which is in accordance with studies by Shankweiler and Harris (1966) and Trost and Canter (1974). According to Rosenbek and Wertz (1976) the presence and severity of co-existing aphasia as well as a patient's avoidance of sounds and combinations of difficult sounds influence comparisons of imitation and spontaneous accuracy.

Error types predominantly included difficulty on clusters, pre-positioning or anticipatory errors, phoneme perseveration and numerous and varied off target attempts at phonemes and words (Trost and Canter, 1974; Dabul, 1979; McNeil et aL 1997). The error scores on words were 8/30, on words increasing in length were 5/30, on sentences 6/10, general conversation 4/10 and 40 on spontaneous speech. These scores were higher than all the other four clients. Even though the case qualitatively shows apraxic features the scores only reflect the presence of AOS, and at this point of time it is difficult to comment on the severity of the disorder. The cut-off score to reveal the presence or absence of AOS can be only established once the protocol is administered on a large group of subjects.

Prosody was highly abnormal in Client 1. This is one section where the quantitative error score correlates well with the qualitative description i.e., 10/10. Hence prosody can be a sensitive measure which can be assessed both qualitatively and quantitatively. Prosodic disturbances are a key feature of apraxics due to their numerous intersyllabic pauses, equal stress on all syllables.

and reduced pitch and loudness variations (Odell et al., 1991; Freed, 2000). A pure Broca's will not have such severe prosodic disturbances as they have only pauses and halts and their dysprosody is more at sentence level and not at syllable level.

Counting forward being automatic was very easy for the case. The case was however unable to count backward (score 1/2). This forward backward discrepancy is a reflection of discrepancy between automatic and volitional tasks respectively and is found to be present in AOS (Darley, 1969).

Hence, based on the qualitative as well as the quantitative assessment it was concluded that the case presented AOS and oral apraxia in association with the Broca's aphasia.

CATEGORY 2. Broca's aphasia with oral apraxia

Two clients ; Client 2 (a 57 year old male) and Client 3, (a 52 year old female) were classified as having only oral apraxia and no AOS.

On the oral apraxia subtest Client 2 scored 30/40 whereas Client 3 scored 24/40. These scores were higher than all the other three clients. Though Client 1 had oral apraxia and AOS, the severity of oral apraxia was less than clients 2 and 3. It is important to note that inspite of a severe oral apraxia, AOS may be absent. This is in accordance with studies which state that though oral apraxia is highly indicative of AOS it is not always necessary that AOS should accompany oral apraxia (Tognola and Vignolo, 1966). Severe oral apraxia according to the

present study was not correlated with AOS.

Client 2 verbally expressed that he knew what had to be done but he was unable to do it. The presence of oral apraxia was highly evident in the case especially revealed by the finer tasks involving the articulators (Task nos. 12,17,18,19 and 20). Behaviours like using hand to facilitate articulator placement, ability to make oral movements only on imitation and struggle and spontaneous verbal expression "I can't say it", "I knew it, but I can't do it" are typical of apraxics. Such utterances may not be present in automatic or spontaneous speech (Schuell, et a1, 1964; Johns and Darley, 1970). Client 2 also showed delay, groping, trial and error performance and lack of motor fluidity for many tasks.

Inspite of having adequate comprehension. Client 3 was unable to carry out simple activities. With effort, she was able to perform the tasks on imitation but voluntarily could not do the same when asked. All the tasks could be elicited on imitation only. This is supported by the experience of authors like Trost and Canter, (1974) Rosenbek and Wertz, (1976), who state imitation to be generally better than spontaneous activity.

On vowel prolongation Client 2 scored 1/6 whereas Client 3 scored 0/6 On DDK tasks both the clients had 0/6. Bot the clients were good at these two tasks. Mild imitation difficulty was present but once automaticity was attained, phonemic integrity could be maintained with few breakdowns. Off target responses if any were in close approximations of target responses eg., /ta/ for /Ta/.

These few breakdowns cannot be called truly AOS errors. They could have been mild reflections of the existing oral apraxia.

On the word level tasks both Clients 2 and 3 had productions (spontaneous and on imitation) which were fluent in terms of motor fluidity. Error scores were 3/30 and 13/30 respectively. They required imitation mainly because of the aphasic word finding difficulty. Since there were no characteristic apraxic like features such as groping, repeated attempts at target or other articulatory difficulties the errors were attributable only to the word finding difficulty which is a component of Broca's aphasias and not AOS (McNeil et al., 1997).

On the increasing word length task, both Clients 2 and 3 had 0/30 errors scores. Their productions were good and motor fluidity was present. However, on longer words few distortions and articulatory difficulties were present. Simplification of the longer words were also present. These could have been due to the effect of the mild oral apraxic component or aphasic language difficulties on the more complex utterances (Lapointe, 1990, Thompson, 1994).

On sentence repetition, Client 2 hardly showed any apraxic error score, while Client 3 showed 4/10 error scores. The speech of both the clients was agrammatic, with poor memory for words or word sequences and word finding difficulties. Attempts were due to word finding problems and were hence mostly at word level. Few phoneme level difficulties or articulatory difficulties were present. There were more pauses than attempts which reflects a word retrieval problem and not a programming error. Investigators like LaPointe (1990) and

Thompson (1994) attribute such difficulties to the aphasic problem.

In general conversation, and spontaneous speech abilities, both the clients 2 and 3 obtained zero apraxic error scores. Client 2 spontaneously initiated communicative interactions, though his speech was halting Kearns (1990) reported that aphasics may initiate communicative interactions more easily, unlike the apraxics in whom initiation difficulty is a key feature of speech as reported by Johns and Darley, (1970). In Client 3, spontaneous speech was limited. Lead questions were required to elicit responses. Though both the clients had agrammatic speech and word finding problems no apraxic errors were observable.

In both the clients again imitation and repetition was better than spontaneous utterances. The few error types present were those of distortions and presence of schwa vowels between consonants.

Both the clients had good prosody. Dysprosody was not present at syllable level. Due to word finding difficulty and pauses, speech rate was reduced which lead to a perception of overall dysprosody. In fluent parts of speech, prosody was intact.

Forward counting was accurate in both the Clients 2 and 3, whereas backward counting was affected in both. Based on this finding it can be concluded that counting involves cognitive abilities and this cognitive ability is affected in aphasics. So counting forward and backward may not be a sensitive task to tap apraxia unless the case is non aphasic or a pure apraxic, which is rare. Hence discrepancy between forward (automatic) or backward counting

(volitional) may not be shown even if the client had apraxia due to the overlaid aphasic dysfunction.

From the quantitative and qualitative assessment it is probable, that the Clients 2 and 3 have oral apraxia but no AOS as word level difficulties are mainly due to word finding difficulty and not articulatory or apraxic difficulties.

CATEGORY 3. Broca's aphasia without apraxia

Clients 4 and 5 obtained few error scores overall. They did not exhibit any of the apraxic features.

On the oral apraxia subtests they obtained low error scores of 5/40 and 7/40. These errors mainly reflected restricted mobility of structures or mild comprehension deficits which could be overcome on imitation or giving a model by the clinician. One of the features which was strikingly apparent was the inability of Client 4 to clear his throat. This feature was seen across four out of the five clients. The reason could not be attributed to any other known cause except laryngeal apraxia (Marshall, Gandour and Windsor, 1988). However with reference to verbal tasks this should be viewed skeptically as the clients were able to perform the verbal tasks but were unable to approximate the vocal cords only for the act of throat clearing.

The clients performed well on all the speech tasks. Client 5 had a few word finding difficulties. Performance of both the clients was good in terms of initiation of speech, range and precision of movements and excellent prosody.

Both the clients showed phoneme substitutions and distortions. In correcting these, the speech of Client 4 became more prone to improper stress and pauses leading to mild dysprosody.

Both the clients were able to perform forward and backward counting unlike the other three clients. Client 4 was exceptionally good at the task.

Both qualitatively and quantitatively the clients show few apraxic features. Both are well recovering from the insult of Broca's aphasia with no AOS. This finding supports the concept of the dissociation of apraxia and aphasia i.e., apraxia and aphasia can exist independently (Shankweiler and Harris, 1966; Heilman et al.. 1979; Kertesz et al., 1984; Ojemann, 1984; Square Storer et aL 1990).

To summarize, the results of the present pilot study reveal that Broca's aphasia may or may not be associated with AOS and oral apraxia. Presence of oral apraxia need not have a high positive correlation with AOS as evident by the results on two clients (Client 2 and 3) who had oral apraxia but no AOS. The protocol was found to be sensitive and could differentiate and identify the presence or absence of AOS in the five clients studied. Quantitative scores and more of qualitative descriptions aided the identification of AOS. Quantitative measures are not as helpful in identifying the AOS as qualitative descriptions, as correlation between quantitative and qualitative results is not very high i.e., scores/digits do not describe the magnitude of the problem effectively. Eg., In Client 1 a score of 12/40 gives an impression that there is no oral apraxia.

However, qualitatively one can assert that the case has oral apraxia along with AOS.

Counting cannot be considered a very sensitive task (to test for discrepancy between automatic and volitional tasks) to test aphasics as cognitive deficits may be a variable that could affect performance and mimic an apraxic feature.

Commenting on the spontaneous speech task it can be said that spontaneous speech sample should be one that consists of the client's self initiated utterances. They should not be elicited following a command eg., "speak on a topic of your choice" as this may become a volitional task for some. Hence the difference between spontaneous vs volitional cannot be demonstrated in the real sense.

However, most of the other sections of the protocol mainly the oral apraxia tasks, the vowel prolongation task, DDK, words that increase in length, sentences and conversation tasks are sensitive in identifying AOS in Broca's aphasics. The error types and dysprosody when observed gave valuable information regarding the speech sample, as to whether the errors are those of apraxia or merely those due to Broca's aphasia.

To establish the cut-off scores for the quantitative data the protocol must be administered on a large number of subjects. This could also give us important information as to which, features or tasks are relatively more common, easily observable or more sensitive in tapping apraxic behaviour.

SUMMARY AND CONCLUSIONS

The aim of the present study was to develop a protocol with features sensitive to identify AOS in Broca's aphasics. The attempt can be called a preliminary step in aiding the provisional diagnosis of AOS in Broca's aphasics. The identification of AOS in a Broca's aphasic has consistently, theoretically been pointed out to be a difficult task as both the disorders have overlapping features (Duffy-, 1995; McNeil et al., 1997; Ballard et al., 1999).

The presumption under which this study was undertaken was that, practically there are ways and means available which would enable a clinician to identify' AOS in Broca's aphasics. This can be best achieved by relying on qualitative analysis more than quantitative analysis of features of AOS in Broca's aphasics. Such an attempt is relevant in the present clinical status as it would enable the clinician to plan for differential intervention when the two disorders co-occur. Another reason for the need for the development of such a protocol is the dearth of available tools for identifying AOS in Broca's aphasics especially in Kannada. The available batteries do not emphasize on qualitative evaluation.

The five sub-tests in the protocol take approximately 30 to 45 minutes to be administered. The protocol has both nonverbal and verbal tasks, the five subtests being:

(A) Test for oral apraxia

(B) Test for effortfulness

(C) Test for error types

(D) Test for dysprosody

(E) Test for counting (forward vs backward)

The scoring was based on a 3 point rating scale (0,1,2) wherein, a higher score obtained indicated the presence of an apractic error. Each sub-test also had qualitative feature probes for the AOS errors. The cumulative of quantitative results and the examiner's observation substantiated the identification i.e., presence or absence of AOS in Broca's aphasics.

Considering the aphasic difficulties the protocol has been made simple enough, with, for example-options for responding on imitation if client fails to comprehend verbal commands, and use of simple common vocabulary etc. This was acceptable because AOS features can be evident even on simple tasks. In all the tasks, assessment focussed on "how" the patient performed the task rather than on the speech output. For example, in the verbal subtasks it was not the verbal output that was assessed, but the effort in the performance and the error types.

The protocol was administered on five clients who were diagnosed as Broca's aphasia on the WAB test (Kertesz, 1982). The results of the assessment revealed one client as having AOS, with oral apraxia, two clients as having oral apraxia and no AOS and the remaining two as having no AOS or oral apraxia.

Certain items of oral apraxia were more sensitive than the others in

eliciting apraxic behaviours eg., the tasks such as touching roof of mouth with tongue, placing tongue between front teeth, puckering lips and clearing throat.

Among the other features, dysprosody was a major feature typically found to be more pronounced in the cases presenting AOS. In other words, using the subtests of the protocol the dysprosody resulting due to an apraxic error and that of an aphasic error could be differentiated.

Based on the results obtained on administration of the protocol, it would be premature to comment on the true "sensitivity" of the protocol in identifying AOS in a Broca's aphasic. However, the protocol can be considered as a useful clinical probe as there is a lack of clinical tools available to identify AOS in Broca's aphasics. The drawbacks of the tools available is that they do not focus on qualitative assessment. In this sense, the formulation of the protocol based on clinical observations over time and detailed study of reports in literature has been a preliminary attempt. Moreover there are no tools available in Kannada for identification of AOS in Broca's aphasics.

The protocol needs to be administered on a larger population to validate and standardize the results. Administration of the protocol on a larger groups of Broca's aphasics especially those with suspected AOS is required to :

- establish the cut-off criteria (for the quantitative scoring of apraxic errors) for the diagnosis of AOS.
- elaborate on the specific distinguishing characteristics of error types of

AOS vs Broca's aphasia especially on the verbal sub-tasks.

- find out which of the sub-tests and items are more sensitive in eliciting AOS behaviours. Such tasks could be expanded on, while the less important or least sensitive tasks could be eliminated or given lesser weightage in scoring.

- find out any difficulties which may be faced during testing and how they could be overcome by modifications.

- modify task items or instructions if they are being affected by aphasic difficulties (eg., cognitive load on items, linguistic load on items or instructions etc).

Hence, the protocol has been presented as a preliminary attempt to aid identification of AOS in Broca's aphasics and needs to be substantiated with more case studies in order to be utilized in clinics in the future.

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