INTRODUCTION

Rationale of the study

Apraxia is a disorder of skilled movement. One of the types of apraxia is apraxia of speech (AOS) which is an articulation disorder that results from impairment of the capacity to order the positioning of speech musculature and the sequencing of muscle movements for volitional production of phonemes and sequences of phonemes. [1, 2]

Apraxia of speech is frequently misdiagnosed as Broca's aphasia or dysarthria due to frequent co occurrence of these disorders together. So an accurate diagnosis of apraxia of speech is very important which depends on valid assessment which can be done through accurate evaluation of the patient during automatic speech, spontaneous speech and oral reading. [3, 4]

Test of Apraxia Battery for Adults II (ABA II) was designed to provide clinicians with a measure to assess apraxia of speech, to provide guidelines concerning the severity of the disorder, and to direct therapeutic approaches in the treatment of apraxia of speech.[5]

The current study applied ABA II after its translation and modification on Egyptian patients having AOS in order to detect the diagnostic features of this disorder for planning the proper management and differentiating it from other disorders.

Apraxia

Apraxia is defined as ‘‘a disorder of skilled movement characterized by loss of the ability to execute or carry out learned purposeful movements not caused by weakness, abnormal tone or posture, movement disorders such as tremor or chorea, intellectual deterioration, poor comprehension, or uncooperativeness’’[1]

Types of apraxia

There are several types of apraxia including:

- Ideational/conceptual apraxia: Patients have an inability to conceptualize a task and impaired ability to complete multistep actions so they cannot carry out learned complex tasks in the proper order. It is an inability to select and carry out an appropriate motor program, e.g. the patient may complete actions in incorrect orders, such as buttering bread before putting it in the toaster, or putting on shoes before putting on socks. There is also a loss of ability to voluntarily perform a learned task when given the necessary objects or tools. For instance, if given a screwdriver, the patient may try to write with it as if it was a pen, or try to comb one's hair with a toothbrush. These patients show confusion of the order of steps, omission of one or more of the steps and preserved ability to perform any individual steps in isolation without error. [6,7]

- Ideomotor apraxia: Patients have deficits in their ability to plan or complete motor actions that rely on semantic memory. They are able to explain how to perform an action, but unable to "imagine" or act out a movement such as "pretend to brush your teeth". Apractic patients are able to perform the movements when they are given real objects. The ability to perform an action automatically when cued remains intact, this is known as automatic-voluntary dissociation, e.g. they may not be able to pick up a phone when asked to do so, but can perform the action without thinking when the phone rings. The cause of this disorder is faulty sensorimotor integration for movement leading to faulty or incomplete and inaccurate programming of the movement so that velocity, course and coordination of movements are distorted. [6,7]

There are several types of Ideomotor apraxia which include:

- Buccofacial, orofacial apraxia or Non-verbal oral apraxia: The patient has difficulty in carrying out movements of the face and oral structures on demand e.g. an inability to lick one's lips or whistle.[8]
- Constructional apraxia: I t is the inability to draw or construct simple configurations such as intersecting pentagons, drawing and arrange blocks or matches.[8]
- Gait apraxia is characterized by loss of the normal function of the lower limbs such as walking. This is not due to loss of motor or sensory functions. [8]
- Dressing apraxia: It is the inability to perform the relatively complex task of dressing.[8]
- Limb apraxia: It refers to ideomotor apraxia of the limbs including hands and fingers with difficulty making precise movements with an arm or leg.[8]
- Apraxia of speech: it is characterized by difficulty planning and coordinating the movements necessary for speech.[1]

Etiology of apraxia

Apraxia occurs due to a lesion located in the left hemisphere of the brain, typically in the frontal and parietal lobes. Lesions may be due to stroke, acquired brain injuries, or neurodegenerative diseases such as Alzheimer disease or other dementias, Parkinson disease, or Huntington disease. It is also possible for apraxia to be caused by lesions in other areas of the brain including the right hemisphere. [6]

Ideomotor apraxia can occur due to lesions in parietal and premotor areas. It is frequently seen in patients with corticobasal degeneration. [6]

Constructional apraxia is usually caused by lesions of the inferior right parietal lobe while ideational apraxia can be caused by lesions in the frontal and temporal lobe. [9]

Apraxia of speech

Apraxia of Speech (AOS) is defined as a motor speech disorder resulting from the impairment of the capacity to program sensorimotor commands for positioning and movements of muscles for the volitional production of speech. It can occur without significant weakness or neuromuscular slowness, and in the absence of disturbances of thought or language. [10]

Wambaugh et al (2006) defined AOS as a motor speech disorder characterized by increased segment and intersegment durations, segmentation of sounds and syllables, speech sound distortions, and dysprosody. [11]

AOS results in difficult speech production, this impairment in speech production is recently attributed to the level of planning or programming of speech gestures not only programming level. So AOS is considered as a phonetic- motoric disorder rather than a linguistically based one. [12, 13]

Patients with speech apraxia have difficulty in accessing or generating phonetic programs resulting in a series of changes and symptoms which are characteristic of this disorder. [14, 15]

Types of apraxia of speech

There are two types of apraxia of speech:

- Acquired apraxia of speech: It can occur at any age after full development of language and speech, although it most typically occurs in adults. [16]
- Developmental verbal dyspraxia (DVD) also known as childhood apraxia of speech (CAS) and developmental apraxia of speech (DAS): It is an inability to utilize motor planning to perform movements necessary for speech production during a child's language learning process. [17, 18]

Theories of speech motor programming.

Speech motor programming can be defined as the set of processes responsible for transforming an abstract linguistic (phonological) code into spatially and temporally coordinated patterns of muscle contractions that produce speech movements.[19,20]

There are three major theories of motor speech programming which are:

I. Darley et al three-level model of motor speech programming.[21]

II. Levelt psycholinguistic model of speech production.[12]

III. Van der Merwe four-phase model of speech sensorimotor control.[22]

Darley et al three-level model of motor speech programming.

Darley et al (1975) described a three-level model of motor speech programming which consists of a central language processor (CLP), a motor speech programmer (MSP) and the motor cortex.

- CLP: For language, the CLP selects the words and word sequences that transform meaningful content into language. For speech, the CLP identifies the phonemes and sequences these phonemes for meaningful language transmission, and then it converts these phoneme sequences into a neural code that drives the MSP.
- MSP activates the appropriate musculature to produce the desired speech sounds in the proper order. Then it projects directly to the motor cortex.
- Motor cortex: is specified for motor execution.

Darley et al. attributed AOS to a disturbance in the programming of muscle movements needed for speech production. The linguistic plans (syntactic, phonologic) for an intended message are assembled accurately but the speaker is unable to program the musculature for its motor expression. The difficulty of speech production in AOS is not related to deficits in the neuromotor execution of speech but it is related to an inability to transform phonologic plans into a code that is recognized by the speech motor system. (21)

Levelt psycholinguistic model of speech production.

Levelt et al (1999) stated that phonetic plans are built through the activation of syllable-sized gestural scores; the syllabary (the motor programs or gestural scores prescribing the articulation for a syllable) (12)

According to this theory, articulatory syllables are among the most exercised motor patterns we produce hence it can be assumed that representations of these patterns are stored in some motor cortical areas in the brain. (23)

When a word is produced, the motor programs of its constituent syllables are accessed and loaded down to the articulatory apparatus. Access to ready-made motor programs in a syllable lexicon is considered to reduce the load of phonetic processing during spoken language encoding. This is referred to as phonetic encoding process (activation / access to stored syllable-sized motor programs); the pathomechanism of AOS was related to this phonetic encoding component. (12)

The model also postulates that this shortcut is only available for the frequently used syllables of a language. Syllables of less frequent distribution may not become stored to avoid over learned programs. When such an infrequent syllable comes up, its constituents must be assembled from smaller subsyllabic units.

Neurolinguistic studies showed that brain-damaged speakers (patients with speech apraxia) produced more phonetic errors on words composed of low frequency syllables and syllables which are frequently used are better retrieved than infrequently used ones. (24)

Van der Merwe four-phase model of speech sensorimotor control.

Van der Merwe (1997) described a four-phase model of speech sensorimotor control which includes:

- Linguistic – symbolic planning: Selection and combination of phonemes occur; phonologic representations (phonemes) are activated and transformed to motor code during the motor planning.
- Motor planning: It involves activating and organizing the temporal and spatial specifications for the production of sequences of phonemes (recall of core motor plans).
- Motor programming (selection of articulatory motor programs): The motor plan subroutines are fed-forward to the motor programming system where muscle-specific motor programs for articulatory movements are selected and sequenced. Motor programs specify spatio-temporal and force dimensions such as muscle tone, movement direction, force, range of movement and rate.
- Motor execution: the plans and programs of the preceding phases are transformed to muscle movements. (22)

Accordingly Van der Merwe conceived AOS as a motor planning disorder since:

(a) AOS is not attributable to deficits of muscle tone or reflexes and
(b) Motor programming deficits allow for alterations in muscle tone, force, direction, speed, and timing. [22]

Close inspection of these models reveals an important difference between Darley model and Van der Merwe point of view with regard to AOS. For Darley et al, the linguistic and phonemic planning functions associated with the CLP show little or no impairment while selective activation of the programming routines for the speech musculature is deficient.

While in the Van der Merwe approach, the fundamental deficit of AOS is found in the planning rather than the programming stage of speech motor production. (Between the level of Darley's CLP and MSP) [25]

Despite the above differences, both Darley et al. and Van der Merwe assumed that the specification and programming of motor speech output are mediated by the cortical association areas (the premotor cortex, the supplementary motor area (SMA), the prefrontal areas and the posterior parietal areas)

Both suggest that AOS results from lesions to the cortical association areas while dysarthria results from lesions to the basal ganglia and other lower segmental structures. [26]

Finally AOS is regarded as a problem that occurs at an intermediate level of speech production, between utterance formulation (where aphasic errors occur) and the muscular execution of motor plans in the articulation of speech (where dysarthric errors occur). It occurs as an intermediate stage between phonological encoding and motor execution. [21]

Etiology of Acquired AOS

AOS results from an insult to the left cerebral hemisphere. Vascular lesions are the most common cause of AOS; but this disorder may also result from head trauma, tumor or other neurological diseases. [10]

It can occur with neurodegenerative diseases. [27] Josephs et al (2006) and Ricci et al (2008) stated that in this case AOS can occur in combination with progressive non-fluent aphasia (PNFA) or in isolation. [28, 29]

Site of the lesion

A number of brain areas have been associated with AOS including the following: left inferior frontal (Broca's area), anterior insular cortex, fronto-subcortical white matter, temporoparietal cortex, basal ganglia, [30, 31] and the parietal lobe. [32]

More focal abnormalities in the superior aspects of the premotor cortex have been observed in primary progressive AOS (PPAOS) without any associated aphasia or dysarthria. [33]

Clinical features of acquired AOS

Patients with AOS may present with any or all of the following salient features:

1) Effortful trial and error groping with attempts at self correction.

2) Persistent dysprosody (abnormal rhythm, stress and intonation)

3) Articulatory inconsistency on repeated productions of the same utterance and/or

4) Obvious difficulty initiating utterances. [34]

Both articulatory errors and prosodic abnormalities are considered hallmarks of AOS. Prosodic deficits are thought to be a secondary effect of poor articulation (e.g., patients may speak in a slow, halting manner because they are anticipating difficulty speaking) [21]

These prosodic, rate and fluency abnormalities can include the following:

- Dysprosody
- Slow rate of utterances more than one syllable in length.
- Prolonged consonants and vowels.
- Silent pauses before speech initiation, pauses between syllables or words give them a segregated character.
- Equalized stress across syllables and words.
- Difficult varying stress in spontaneous and imitated sentences.
- Restricted or altered pitch , durational and loudness contour within utterances
- Limited variations in relative peak intensity across syllables resulting in dysprosody with abnormal stress and rhythm patterns. [35]
- False articulatory start and restart and repetitive attempts to produce words.
- Effortful visible and audible trial and error groping for articulatory postures especially at the beginning of the utterances, which may accompanied with facial grimace.
- Sound and syllable repetitions.
- Initiation of utterances particularly difficult. [10]

Articulation can be considered the primary affected speech component in AOS leading to speech abnormalities. These abnormalities are predominantly interpreted as symptomatic disruption in the phasing of movements between subcomponents of the speech production mechanism and disturbed spatial articulatory configurations. [36]

These speech abnormalities include:
- Inconsistent and variable articulatory movements.
- Articulatory errors that increase complexity of articulation across a word rather than simplify.
- Errors that approximate the target within one to two features.
- More substitution than distortion, omission, or addition errors. [11]
- Errors that represent perseveration, anticipation, and transposition of phonemes.
- Schwa insertion in consonant clusters
- Voicing errors may occur.

- Awareness of errors with trials of self correction (associated with struggle) is a prominent feature.
- More consonant errors than vowel errors.
- More errors in word initial than word final position.
- More place than manner or voicing errors. [19]
- With regard to phoneme type, consonant clusters are more frequently in error than singletons (e.g., 'strict' will be more difficult than 'sit')
- Affricates and fricatives are more frequently in error than other manners of production (as the 'ch' in 'church' will be more difficult to say for apraxic speakers than a word with bilabial phonemes, such as the 'm' in 'mom')
- Patients with AOS are more likely to produce errors when asked to repeat nonsense words as opposed to meaningful words. [10]
- Increased word and vowel duration patterns. [37]
- Reduced coarticulation has also been observed in speakers with AOS.[38]

In addition to the above speech abnormalities there are other features of speech specific for AOS which include:

- Automatic speech is easier and more error free than volitional speech.[10]
- Imitative responses are characterized by more articulatory errors than spontaneous speech production.
- In oral reading, articulatory errors do not occur at random. They are more frequent on words that carry linguistic or psychological weight and that are more essential for communication. This weight is based on four characteristics: grammatical class (noun, adjective, adverb, and verb), difficulty of initial phoneme (fricative, affricates or consonant cluster), sentence position (one of the first three words of the sentence) and word length (more than five letters long).
- Articulatory errors increase as the complexity of the motor adjustment needed for articulation increases as repetition of single consonants /p/, /t/ or /k/is much more easier than repetition of a syllable like /puh- tuh- kuh/.
- Phonemes occurring with high frequency tend to be more accurately articulated than less frequent phonemes.
- Articulation errors increase with increase in the word length.
- Articulation is affected by mode of stimulus presentation; articulation is more accurate with combined visual and auditory mode than auditory mode only.
- Correct articulation is facilitated more by repeated trials on a word than by increase in the number of stimuli presentations.[11]

AOS may appear isolated in a small number of cases or in combination with other impairments, such as aphasia (usually of the Broca's or conduction aphasia), dysarthria or orofacial apraxia.[39] It may be the first and most prominent symptom in some neurodegenerative conditions, particularly in some variants of primary progressive aphasia and corticobasal degeneration. In this case apraxia of speech can evolve as an isolated impairment during several years without positive signs of aphasia, pointing to a pattern of speech apraxia referred to as primary progressive apraxia of speech (PPAOS). [40, 41]

So AOS can be associated with several disorders and features which include:

1. Limb and oral apraxia: AOS can exist without limb and oral apraxia although there is growing evidence reveals that individuals with AOS consistently demonstrate impairments in nonspeech oral and limb movements (e.g. limb, oral apraxia) [42, 43]
   These nonspeech deficits in AOS are independent from observed speech impairments and merely indicate proximity of underlying neural structures responsible for different tasks as speech and nonspeech movements that are controlled by an overlapping neuromotor control system. [44]
2. Impairment of oral sensation and perception.[10]
3. Aphasia: AOS can occur with aphasia especially Broca's and conduction aphasia. [45, 46]
4. In some cases, a combination of AOS with agraphia has been described. [47,48]
5. Dysarthria: usually unilateral UMN or spastic dysarthria due to the proximity of the site of lesion for these disorders.[10]

Differential diagnosis

1. AOS can be associated with Broca's aphasia, in this case, subjects will present with both disorders, and they display grammatical errors in speech and writing, and impairment in comprehending syntactically complex sentence.[49] It differs from Broca's aphasia in that:
   - Speech errors indicative of AOS are non-linguistic in nature and therefore distinct from those heard in aphasia.
   - Difficulty in articulation of a particular word is not related to word finding difficulty as the patient can write the word or choose the word correctly from a group of presented words.
   - AOS can occur independently of language-related impairments in auditory comprehension, reading comprehension and writing. Patients with speech apraxia do not manifest truly linguistic deficits such as agrammatism and naming deficits.[21]

2. Beside that AOS is often confused with conduction aphasia, perhaps because sound level errors (substitutions, additions, transpositions or omissions) are prominent in both disorders. However, these disorders are thought to be different as:
   - Patients with conduction aphasia typically speak with near normal prosody, whereas halting effortful speech and struggle with abnormal prosody is considered a hallmark of AOS. [34]
   - Patients with conduction aphasia may lack awareness of their speech errors and therefore may not always make attempts at self-correction, while the opposite is true in cases of AOS. [32]
   - The sound errors in conduction aphasia reflect an underlying deficit in the selection of the phonemes for speech that is a language deficit. Apraxic speakers on the other hand are believed to select the correct phonemes but they have only trouble with their motor execution. [10, 14] Despite this, the differential diagnosis of the speech errors in AOS and conduction aphasia can be difficult due to the similarity in these sound level errors. [32]

3. AOS differs from dysarthria in a variety of features which are described in the following table.

Table 1: Differential diagnosis between AOS and dysarthria.

Acquired apraxia of speech	Dysarthria
Caused by disruption of the message from the motor cortex to the oral muscles (central nervous system lesion)	Caused by disruption in muscular control of the central or peripheral nervous system.
There is change in motor programming for speech secondary to neurologic involvement. Muscle tone not affected. Involuntary motor tasks are not affected.	There is impairment of muscle strength, tone, range of motion and/or coordination secondary to neurologic involvement. Difficult voluntary and involuntary motor tasks such as swallowing, chewing and licking.
The speech process for articulation is primarily affected, prosody may be also abnormal.	All speech processes are affected including respiration, phonation, resonance, articulation and prosody.
Speech errors are inconsistent and unpredictable with islands of clear well articulated speech.	Speech errors are consistent and predictable with no islands of clear speech.
Articulatory errors are primarily substitutions, repetitions, additions, transposition, prolongation, omissions and distortions (which are least common) Most errors are approximations of the targeted phoneme. Errors are often perseveratory or anticipatory.	Articulatory errors are primarily distortion and omissions.
Consonant are more difficult than vowels, initial consonants are more difficult, blends are more difficult than single tones, fricatives and affricates are the most difficult consonant, errors increase as the complexity of the motor pattern increases.	Consonant production are consistently imprecise, vowels may be neutralized.
Prosodic disorder may occur as a result of compensatory behaviors (stopping, restarting and difficulty initiating phonation)	Speech rate is slow and labored, poor breath support may be apparent.
Speech intelligibility sometimes increases as speaking rate increases.	Speech intelligibility is reduced as speaking rate increases.

(Darley et al 1975, Duffy 1995)[10, 21]

Diagnosis

Diagnosis of apraxia of speech tends to be made by clinical judgment with reference to the presence of characteristic apraxic speech behaviors. [14]

Motor speech examination (MSE) has been widely used in the diagnosis of AOS for accurate detection of these speech behaviors. [50, 51]

AOS diagnosis can be done through this systematic approach:

I - Elementary diagnostic procedures

A-Patient and family interview:

Personal history: It includes personal data, handedness, smoking and educational level.

History of the present illness: Which includes the duration, onset and course of the present illness, history of prodromal syndromes, any disturbance in consciousness and its duration, presence of other associated neurological deficits or motor disabilities such as hemiplegia and its severity, the present mean of communication, the previous lines of treatment and any investigation (CT, EEG and MRI…), and the integrity of visual and auditory pathways.

Past history: It includes relevant diseases such as hypertension, heart diseases and diabetes mellitus and similar or related episodes as transient ischemic attacks**.**

B-Clinical Examination:

- **Auditory perceptual assessment:**

- **Contextual speech assessment:**

Diagnosis of AOS depends on tasks placing demands on the volitional sequencing of a variety of sounds and syllables which are most likely to elicit the salient and distinguishing features of AOS. Conversational and narrative speech and reading can be used for this purpose, particularly if language and reading skills are good and the patient can give more than brief and unelaborated conversational or narrative responses. [10]

In MSE speech samples has to be elicited during various tasks and it is to be determined the presence or absence of AOS and/or dysarthria by analyzing the patients' performance on these tasks with assessment of articulation, resonance, voice, prosody, breathing and speech intelligibility to detect any abnormalities in these speech components. [50]

These tasks include:

- Vowel prolongation.
- Repetition of syllables:
    - Rapid repetitions of a monosyllable usually one of /pa/, /ta/, /ka/ or /ba/, /da/, /ga/ is considered a sensitive test to measure motor performance of the muscular system required for speaking. This task which is often referred to by the terms syllable

alternating motion rate (AMR), rapid repetitive articulation (RRA), or oral diadochokinesis (DDK) is included in standard assessment protocol for speech disorders. [50, 51] This examination is considered speechlike since it is based on real syllables and it is seen as a sensitive index of motor speech impairment because it requires maximum performance and coordination of articulators. [52, 53] Oral DDK rate is correlated with age and is viewed as an index of oral motor development.[54,55]

- Sequential motion rate (SMR): In which the patient is asked to repeat 'puh-tuh-kuh' over and over again; it is useful to measure the ability to move quickly from one articulatory position to another.

- Words and phrases: Sequential motion rate and imitation of complex multisyllabic words and sentences are considered among the tasks most sensitive to AOS.

- Oral reading, conversational and narrative speech and picture description.

Performance on these tasks can be compared with easier tasks like singing a familiar tune, counting and saying the days of the week. A mismatch between speech adequacy on complex voluntary tasks and simpler automatic tasks increase the likelihood of AOS. [10]

- **Assessment of language**: To exclude any associated language impairment (aphasia), it can be done through assessment of :
  - o Receptive language (word comprehension, understanding simple and complex sentences)
  - o Expressive language(assessment of nominative and narrative function of language)
  - o Reading and writing assessment.

- **ENT examination:** This includes examination of oral cavity, nasal cavity, ears, pharynx and indirect laryngoscope.

- **Neurological examination:** complete neurological examination includes: State of consciousness, cranial nerves examination including oral motor examination to detect any abnormalities in the speech muscles, examination of motor system (muscle state, power, tone and involuntary movement), testing superficial and deep tendon reflexes, examination of superficial and deep sensation, coordination and description of patient gait.

II-Clinical Diagnostic aids

- **Formal testing:**

  - Dysphasia test: using modified scoring system for testing language disability in dysphasic patients.[56]
  - Psychometric tests such as:
    - Progressive Matrices test of Raven.
    - Snijders – Oomen, Non Verbal Intelligence scale.
    - Taylor Test of Anxiety.
  - Articulation test and intelligibility test.
  - Apraxia Battery for Adults II.

It is the standardized test that is widely used as a formal measure of apraxia characteristics in adult speech. (5)

There is other test for assessment of AOS called Comprehensive Apraxia Test (CAT). But it has no validity and reliability data which are available in ABAII. The advantage of ABAII that is it represents tasks that can be administrated in a standard fashion. Scores can be used to describe patient performance, compare performance over time and quantify the diagnosis and the severity of the problem. (10)

Apraxia Battery for adult consists of six subtests which are:

Subtest 1: Diadochokinetic Rate: This measures volitional control over the articulators.

Subtest 2: Increasing word length: This measures the ability to sequence the correct number of syllables in the proper order.

Subtest 3: limb apraxia and oral apraxia: This subtest measures the ability to produce a movement based on an oral direction.

Subtest 4: latency time and utterance time for polysyllabic words: This measures the amount of time it takes to begin initiation of a word and the time taken to produce a word.

Subtest 5: Repeated trials: This subtest measures change in production of words over successive trials.

Subtest 6: Inventory of articulation characteristics of apraxia: This subtest measures the presence of apraxic speech behaviors as the various types of articulatory errors that may be perceived by a listener (e.g., phonemic anticipatory errors, perseverative errors, transposition errors, etc.).(5)

III-Additional Instrumental measures

- **Acoustic measures:**

This includes measurement of speaking rate, vowel and syllable duration, voice onset time. The common characteristics of AOS involve spatio-temporal disruptions with articulatory discoordination manifesting as impaired spatial targeting, reduced coarticulation, reduced control of voice onset time (VOT) and increased variability on durational measures over repeated trials with impairment of articulators control.(13, 57) With increased word and vowel duration is also characteristic of AOS (37)

- **Physiological measures:**

These physiological measures have two applications:

- Examination of motor control of single articulator(kinematic characteristics)
- Examination of interarticulator coordination.(13)

These measures include direct measures of movement amplitude, duration, velocity and force within the articulators (single or multiple muscles) to define spatial and temporal parameters of articulator movement.

These instruments include:

- Electromagnetic articulography (EMA)
- Electromyography (EMG)
- Electropalatography (EPG) which is used to provide information on tongue –palate contact during production of speech sounds. [58,59]

It can also be used as a tool to provide direct feed back to the patient during training to evaluate movement patterns as the treatment progresses. [13]

Treatment

Most available treatment approaches for AOS have limited data to support their efficacy. [60] Over the last few decades a variety of treatment approaches have been studied with no one approach proving to be effective for all patients.

AOS is generally believed to primarily disturb articulation and prosody; many approaches have focused on remediating these specific deficits. [61]

Therapy goals are typically designed to improve communicative effectiveness. For the mildly apraxic patients poor prosody may be the primary speech deficit therefore goals designed to improve intonation and stress may be the most appropriate. For the moderately or severely apraxic patient, therapy might focus on relearning oral postures for individual speech sounds. [32]

Treatment approaches that are frequently suggested for apraxia of speech fall into three categories:

1. Medical intervention: Pharmacologic intervention may be used for people with AOS to treat the underlying etiology or prevent further impairment (for example, antibiotics for infection, anticoagulant to prevent stroke, anticonvulsant to prevent seizures) and may indirectly result in speech improvement or prevent deterioration.

2. Alternative or augmentative communication devices have also been prescribed for patients with severe AOS. (Although augmentative communication is not a treatment approach for apraxia, it is sometimes necessary to facilitate overall communication) These devices include the use of compensatory strategies to replace speech (as gesturing, writing, drawing, communication books, etc.)[10, 61]

3. Behavioral management: It is at the heart of managing AOS, it includes several approaches which include:

   a. Those that target articulatory movement patterns and sound production in the form of traditional articulation therapy (repetitive exercises involving imitation of speech sounds and words)

   b. Those that focus primarily on prosodic aspects of speech production.[61]

   c. Those that use tactile and gestural cues as the primary facilitator.

The following section present various treatment approaches used in treatment of AOS which include:

I. Articulatory approaches

Many of the approaches used to treat apraxia of speech focus on improving the ability of the individual to achieve initial articulatory configurations then transition the movement throughout the syllable structure. The approaches and techniques that focus on articulatory movement usually start with phonetically simple shorter utterances and progress to longer more phonetically complex phrases and sentences.

These treatment approaches primarily targeted motor performance for sound production and/or integral stimulation for improvement of speech production. [61]

These articulatory approaches include:

A. Integral Stimulation

For moderate or severe apraxia, the most commonly used method to improve speech production is imitation. The clinician provides an auditory and visual model after asking the speaker to "watch me and listen to me."

Wambaugh et al 2006 stated that integral stimulation is imitative method and emphasizes multiple input modes; it works well as a motor approach to treatment, because the focus is on the movement patterns. Repetitive motor practice is incorporated to facilitate motor learning. [11]

There is systematic Eight Step Continuum for treatment of AOS (using integral stimulation) which include:

Step1: Integral stimulation; the clinician presents a target stimulus which the patient then imitates while watching and listening to the clinician's simultaneous production.

Step2: As step 1 but the patient response is delayed and the clinician mimes the response (without sound) during the patient's response; so the simultaneous auditory cue is faded.

Step3: Integral stimulation followed by imitation without any simultaneous cues from the clinician.

Step4: Integral stimulation with several successive productions without any intervening stimuli and without any simultaneous cues.

Step5: Written stimuli without auditory or visual cues followed by the patient response while looking at the written word.

Step6: Written stimuli with delayed production following removal of the written stimuli.

Step7: A response is elicited with an appropriate question.

Step8: The response is elicited in an appropriate role play situation. [62]

B. Multiple Input Phoneme Therapy (MIPT):

This approach is similar to integral stimulation. It is appropriate for those individuals with severe apraxia who have only a few verbal stereotypies.

Frequent stereotypic utterances are used as initial stimuli; the clinician models the utterance for the client.

A simultaneous gestural/prosodic cue (tapping of the ipsilateral arm) is performed by both the client and the clinician.

After about 10 productions, the clinician fades the voice but mimes the movement and taps while the client continues to produce the target.

Then the clinician introduces new words using the initial phoneme of the stereotypic utterance.

Progressively the clinician would increase the number of targets as well as the length and phonetic complexity of targets. (63)

C. Sound production treatment (SPT)

It is used to improve production of consonants that are problematic for a specific speaker with apraxia of speech. (61)

It is a treatment hierarchy composed of modeling and repetition of minimally contrastive words with the use of graphemic cues, integral stimulation and phonetic placement cueing. (64)

A number of studies have shown positive effects of this treatment for sound acquisition and response generalization to untrained exemplars of trained sounds. (65)

II. Prosodic approaches

Rhythm, rate and intonational contours are used as the primary targets of treatment. The clinician can change rate or incorporates rate, rhythm and/or contrast stress in order to improve the accuracy of speech production. A number of prosodic treatment approaches have been reported including:

- Melodic intonation therapy (MIT): Its distinctive feature is its reliance on singing and a variant of it in which intonated utterances are based on melody, rhythm, and patterns of stress in a spoken model provided to the patient. Repetition forms the core of MIT with the use of a variety of high probability utterances with semantic value to the patient. It includes the use of verbal and gestural cues with twice daily treatment sessions(10, 66)

- Use of a metronome or finger tapping and other rate control strategies: Rate modification plays a significant role at the multisyllabic word, phrase, or sentence level in treatment of AOS. (67, 68)
  Pacing board, metronome, hand or finger tapping, and other gestural rate control strategies may be helpful, as well as slowing rate without cues from other modalities. (69, 70)

- Contrastive Stress: It is helpful at the multiple syllable utterance level, it is very applicable to patients with AOS because it slows rate. It includes imitation, question and answer dialogue with stress on a target word, and more complex utterances with different locations for target words or multiple stressed target words. (34)

III. Tactile/Gestural approaches.

Gesture is commonly used as a facilitator to augment the primary procedures used in treatment; but tactile cues and/or gesture can be used as the primary focus of intervention as in these methods:

- Cueing Strategies: These are relevant for sound, syllable, and word level activities. In addition to biofeedback, cues that facilitate accurate responses at the word level include word imitation (watch and listen), sentence completion, first sound of the target word, description of function, the printed target word and presentation of associated words. (71)

- Prompts for Restructuring Oral Muscular Phonetic Targets (PROMPT): Its distinctive feature is its use of tactile cues to provide touch, pressure, kinesthetic, and proprioceptive cues to facilitate speech production. It uses highly structured finger placements on the patient face and neck to signal articulatory target positions as well as cues about other movement characteristics such as manner of articulation, degree of jaw movement and syllable duration. It is appropriate for patients with severe AOS whose spontaneous verbal output is very limited and for whom traditional methods of treatment have failed. (72, 73)

Biofeedback techniques in treatment of AOS.

- One recently reported treatment technique suggested that on-line visual feedback using Electromagnetic Articulography (EMA) improved articulatory accuracy in one individual with AOS. That study used feedback, a condition reported to increase rate of skill acquisition but diminish generalization and maintenance effects. (74)

- Other study described the use of Electropalatography (EPG) to treat a patient with severe acquired apraxia of speech. EPG is a computer-based tool for assessment and treatment of speech motor disorders. The program allows patients to see the placement of articulators during speech production thus aiding them in attempting to correct errors. This study showed that EPG therapy gave the patient valuable visual feedback to clarify speech movements that had been difficult for the patient to complete when given only auditory feedback. (59)