Section A-Research paper

An Insight about Childhood Apraxia of Speech

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Abstract

Background: The literal meaning of the Greek term 'apraxia' is 'without action'. Apraxia in behavioral neurology refers to the loss of the ability to carry out learned, skilled actions (e.g. opening door with a key) in the absence of motor, sensory, coordination or comprehension abnormalities. It is to be differentiated from akinesia, which is defined as a general failure to initiate movement in the absence of weakness. For the desired motor function to occur, it goes through three distinct phases: ideation, planning, and execution. Structures involved in praxis are mainly in the dominant parietal cortex, frontal cortex, basal ganglia, and corresponding white matter connecting these areas. Spatiotemporal movement formulas or visual-kinesthetic motor engrams, also known as praxicons, are essential for skilled learned movements. Activation of these models can be visual, auditory, verbal, or tactile—a model for praxis based on the work of Heilman and Rothi. Neurons in the inferior parietal cortex fire selectively to initiate the process of praxis after recognition of the input provided, especially in left parietal sub-regions. In 1920, Liepmann suggested 3 different types: limb-kinetic apraxia, ideomotor apraxia, and ideational apraxia. Whether all of these are in fact disorders of 'skilled action' is debatable. This controversial nature of apraxia is also reflected in the highly variable rules behind naming of apraxia types and apraxic phenomena. Some are named based upon the body parts involved (e.g. orofacial, ocular motor), some are named after their putative mechanisms (e.g. ideational, constructional), others are named after the tasks that are involved (e.g. writing, speech, eyelid opening), and in rare cases, naming involves the neuroanatomical substrate (e.g. callosal apraxia). CAS is a neurological childhood (pediatric) speech sound disorder in which the precision and consistency of movements underlying speech are impaired in the absence of neuromuscular deficits (e.g. abnormal reflexes, abnormal tone. CAS may occur as a result of known neurological impairment, in association with complex neurobehavioral disorders of known and unknown origin, or as an idiopathic neurogenic speech sound disorder. The impairment in planning and/or programming spatiotemporal parameters of movement sequences results in errors in speech sound production and prosody.

Keywords: Childhood, Apraxia

Introduction

The literal meaning of the Greek term 'apraxia' is 'without action'. Apraxia in behavioral neurology refers to the loss of the ability to carry out learned, skilled actions (e.g. opening door with a key) in the absence of motor, sensory, coordination or comprehension abnormalities. It is to be differentiated from akinesia, which is defined as a general failure to initiate movement in the absence of weakness **(1).**

Apraxia is a helpful localizing sign on the mental status examination and often predicts disability in patients with stroke or dementia. It can affect both sides of the body, even when the underlying lesion is unilateral. Apraxia can occur in the absence of any language deficits, despite the proximity of

cortical areas involved in praxis and language processing. Since the earliest descriptions, the concept of apraxia has expanded to include 'unskilled' and 'novel', as opposed to 'skilled and learned' movements **(2).**

There are several different forms of apraxia, and these have been variously organized based on the body part affected and the specific type of dysfunction. Apraxia is a hallmark feature of corticobasal syndrome (CBS), a progressive condition characterized by frontal, parietal and basal ganglia dysfunction. This syndrome is associated with neurodegenerative diseases such as corticobasal degeneration (CBD), progressive supranuclear palsy (PSP), Alzheimer's disease (AD) and rarely, dementia with Lewy bodies (DLB). Clinically, CBS involves akinesia (lack of movement) and rigidity (stiffness) in combination with cortical signs such as apraxia. As such, CBS is a useful model to study the anatomic correlates of praxis (3).

Etiology

A list of the typical lesions affecting the brain gray and white matter that can cause apraxia are as follows:

Cerebrovascular accident (CVA)
Corticobasal syndrome
Alzheimer disease
Huntington disease
Multiple sclerosis (MS)
Tumors
Creutzfeldt-Jakob disease
Schizophrenia
Traumatic brain injury (TBI)

Risk factors for developing apraxia depend on the risk of developing the etiological disorder **(3).**

Pathophysiology

For the desired motor function to occur, it goes through three distinct phases: ideation, planning, and execution. Structures involved in praxis are mainly in the dominant parietal cortex, frontal cortex, basal ganglia, and corresponding white matter connecting these areas. Spatiotemporal movement formulas or visual-kinesthetic motor engrams, also known as praxicons, are essential for skilled learned movements. Activation of these models can be visual, auditory, verbal, or tactile—a model for praxis based on the work of Heilman and Rothi. **(4).**

Neurons in the inferior parietal cortex fire selectively to initiate the process of praxis after recognition of the input provided, especially in left parietal sub-regions **(4).**

The knowledge of tools and their specific usage, association with other objects, conceptualization, and action semantics reside in the left parietal cortex **(2).**

After conceptualization and semantics, learned motor programs are essential in executing the desired action. The supplementary motor areas in the premotor cortex translate the formulas into specific sequential movements and bilateral coordination. The prefrontal cortex helps sequence fingers, hands, and arms; these are represented functionally in the inferior frontal gyri for the proximal limb and left lateralized for distal limbs **(5).** Visual information is processed and integrated by the bilateral parietal cortex. Other structures in the brain like basal ganglia (putamen, caudate, or globus pallidus), thalamus (pulvinar), and the right (non-dominant) hemisphere, posterior parietal, and temporal cortices are involved in the development of apraxia.

Epidemiology:

There are no available studies assessing the collective prevalence of the various types of apraxia. Apraxia is prevalent in 25.3% of 'first stroke' patients, 51.3% of left hemispheric strokes and 6% of right hemispheric strokes. Also is often observed in head trauma involving the left parietal lobe. Limb apraxia is highly prevalent in CBS (70–80%) and is a key defining clinical feature of CBS. CBD, its most common neuropathological correlate, is rare, with an estimated prevalence of 2–7 per 100 000 individuals **(6).**

Clinical subtypes:

In 1920, Liepmann suggested 3 different types: limb-kinetic apraxia, ideomotor apraxia, and ideational apraxia. Whether all of these are in fact disorders of 'skilled action' is debatable. This controversial nature of apraxia is also reflected in the highly variable rules behind naming of apraxia types and apraxic phenomena. Some are named based upon the body parts involved (e.g. orofacial, ocular motor), some are named after their putative mechanisms (e.g. ideational, constructional), others are named after the tasks that are involved (e.g. writing, speech, eyelid opening), and in rare cases, naming involves the neuroanatomical substrate (e.g. callosal apraxia) **(7).**

- Idemotor apraxia:

Ideomotor apraxia is defined as an impaired performance of skilled motor acts despite intact sensory, motor and language functions. It is typically demonstrated when a patient is asked verbally to perform a gesture with a limb. Impaired imitating of meaningless gestures, such as made-up hand postures, may suggest a deficit in converting visual information into action, rather than a deficit in retrieving encoded action sequences **(8).**

- Ideational and conceptual apraxia:

In ideational apraxia, patients have difficulty carrying out a sequence of actions in the performance of a complex, multistep task, such as mailing a letter. Ideational apraxia is often seen in patients with extensive left hemisphere damage, dementia and delirium. Problems with ordering actions may be due in part to executive and memory impairments, or to an overall deficit in cognitive resources **(8).**

Gross and Grossman (8) have made a distinction between ideational and conceptual apraxia. In contrast to a disorder of action sequencing, patients with conceptual apraxia demonstrate loss of object or action knowledge. They may misuse objects, have difficulty matching objects and their actions, be unaware of the mechanical advantage provided by tools. Conceptual deficits often can be seen in patients with dementia who have a disorder of semantic memory, and have been associated with lesions of the left temporal lobe. Importantly, both ideational and conceptual apraxias often lead to severe disability in the performance of activities of daily living.

- Limb-kinetic apraxia:

Limb-kinetic apraxia has been used to describe inaccurate or clumsy distal arm or leg movements. It is typically noted in the limb contralateral to the affected hemisphere **(4).** For instance, It was used selective hemisphere anesthesia to demonstrate left hemisphere dominance for motor deftness in right-handed epilepsy patients with typical, left-sided language lateralization. In addition, Heilman and Watson **(1).**have revealed that the left hemisphere influences the ipsilateral left hand more than the right hemisphere influences the right hand.

It is possible that the dominant left hemisphere influences the right hemisphere's motor programme via the corpus callosum, the largest white matter structure connecting the two cerebral hemispheres. Thus, bilateral limb-kinetic apraxia may be observed with lesions limited to the left hemisphere **(1).**

- Orofacial apraxia:

Orofacial apraxia (also called oral or buccofacial apraxia) is characterized by an impairment of skilled volitional movements involving the face, mouth, tongue, larynx and pharynx. It is tested by asking patients to imitate both transitive (e.g. sucking on a straw, blowing out a candle) and intransitive (e.g. whistling) gestures. Orofacial apraxia has been associated with inferior frontal, deep frontal white matter, insula and basal ganglia lesions. Automatic gestures involving the same muscles are often preserved, as is the case with ideomotor limb apraxia. Orofacial apraxia frequently coexists with limb apraxia **(9).**

There are other different types of apraxias: constructional, dressing, writing, gait, gaze, and apraxia of eyelid opening, speech apraxia.

Apraxia and aphasia:

Phoniatricans and neurologists of the late nineteenth century identified the phenomenon of apraxia as part of aphasia, as a deficit in 'recognising the use' of tools, or as a deficit in 'memories of kinesthetic

perception'. Some author considered it as amplification of aphasia **(2).**

In those with concurrent aphasia and apraxia, lesions were noted in the left inferior frontal gyrus, particularly in an anteroventral subarea of Brodmann area 44 (BA 44). It is notable that BA 44 and BA 45 form the traditional 'Broca's area' involved in speech production in the dominant hemisphere. Apraxia was tested in these patients with 3 tasks: pantomiming the use of a tool, imitation of a meaningful gesture and imitation of a meaningless gesture (all shown through pictures). The anteroventral subarea of BA 44 is involved in extracting meaning from sensory information and semantic processing, and could explain the deficits in pantomiming and imitating meaningful gestures in these patients. **(10).**

Apraxia of speech in the acute setting of stroke is commonly misdiagnosed as aphasia. Detailed testing at the bedside can be difficult, but if the patient's writing and reading/auditory comprehension are normal, and speech is notable for phoneme prolongation and inter- syllabic segmentation, then apraxia of speech rather than aphasia should be considered. **(11).**

Apraxia versus Dyspraxia:

The alternative terms—apraxia of speech versus (verbal) dyspraxia—each have established traditions in international literatures. Apraxia of speech is more widely used in the United States following the Mayo Clinic traditions **(11).** , whereas verbal dyspraxia is the preferred term in many other English-speaking countries. Differentiating between these alternatives based solely on etymological distinction (i.e., total [a] vs. partial [dys] absence or lack of function) is problematic when applied to CAS. Clinical experience indicates that although a child suspected to have CAS may have very limited speech, seldom is a child completely without mastery of some speech sounds. Notwithstanding this difference, and to parallel usage for the possible acquired form of this disorder in adults (i.e., AOS), the Committee recommends use of the affix a for this classification term. Rationales for the second and third words in the classification term CAS reflect empirical findings for children suspected to have this disorder.

The term 'dyspraxia' was not applied to children with developmental speech disorders until the 1950s when Muriel Morley identified a "dyspraxia" as a defect of articulation which occurs when movements of the muscles used for speech appear normal for involuntary and spontaneous movement or even for voluntary imitation of movements, but are inadequate for the complex and rapid movements used for articulation and reproduction of sequences of sounds used in speech. **(12)**

- Oral Motor Skills

It is debatable whether a diagnosis of verbal dyspraxia can be made in the absence of oral dyspraxia. Eisenson (1972) argued that, unlike the acquired condition, developmental verbal dyspraxia will always be accompanied by oral dyspraxia. Some cases had some degree of oral motor incoordination even though no obvious motor weakness was apparent. Crary's (1984)

- The Clinical Perspective

'Hard Neurological Signs Acquired verbal dyspraxia is normally accompanied by a right hemiparesid paralysis with lesions in Broca's area and the sensorimotor cortex. In contrast, hard clinical evidence of a neurological etiology has been difficult to find in its developmental counterpart

'Soft Neurological Signs The incidence of neurological soft signs in developmental dyspraxia includes drooling, early feeding difficulties, and clumsiness on gross and fine motor tasks such as bead threading, tying shoelaces and dressing. **(13).**

There are two main types of AOS: acquired apraxia of speech and childhood apraxia of speech.

Acquired AOS can affect someone at any age, although it most typically occurs in adults. AOS is caused by damage to the parts of the brain that are involved in speaking and involves the loss or impairment

of existing speech abilities. It may result from a stroke, head injury, tumor, or other illness affecting the brain. AOS may occur together with other conditions that are caused by damage to the nervous system. One of these is dysarthria. Another is aphasia **(11).**

Childhood apraxia of speech

Definition of CAS:

CAS is a neurological childhood (pediatric) speech sound disorder in which the precision and consistency of movements underlying speech are impaired in the absence of neuromuscular deficits (e.g. abnormal reflexes, abnormal tone **(14).**

CAS may occur as a result of known neurological impairment, in association with complex neurobehavioral disorders of known and unknown origin, or as an idiopathic neurogenic speech sound disorder. The core impairment in planning and/or programming spatiotemporal parameters of movement sequences results in errors in speech sound production and prosody **(14).**

Some have proposed that CAS is linguistic in nature, others have proposed that it is motoric. Rapin & Allen (1987) do not consider phonological disorder as a separate entity but associate it with sub-types of language disorder e.g. phonological syntactic syndrome, Phonological-programming deficit syndrome and Verbal dyspraxia.

CAS is present from birth. This condition is also known as developmental apraxia of speech, developmental verbal apraxia, or articulatory apraxia. CAS is not the same as developmental delays in speech, in which a child follows the typical path of speech development but does so more slowly than is typical. The causes of CAS are not well understood. Children with AOS often have family members who have a history of a communication disorder or a learning disability. This observation and recent research findings suggest that genetic factors may play a role in the disorder. **(15).**

Neuroanatomical correlates of CAS:

Functional MRI studies in healthy adults suggest that different brain regions contribute to speech planning. the medial frontal cortex, anterior insula, dorsolateral frontal cortex and superior cerebellum. Consistent with this hypothesis, apraxia of speech in adults was found to be strongly related to frank lesions of the left hemisphere, mainly due to brain infarcts involving the posterior part of Broca's region, the frontal insular cortex or its adjacent white matter **(16).**

Much less is known about pediatric motor speech planning disorders. In contrast to what is reported in adults, the majority of children with idiopathic or symptomatic (e.g. related to epilepsy or metabolic disorders) CAS were found to have normal structural brain MRI on conventional imaging, suggesting that brain abnormalities that underly idiopathic CAS might be too subtle to be detected by clinical MRI **(16).**

In children with idiopathic CAS, a thicker left supramarginal gyrus was found compared to controls in the absence of appreciable macroscopic lesions from a neuroanatomical qualitative approach. Overall, these findings have been interpreted as the possible result of an immature or altered development of connectivity, where a thicker cortex may reflect the lack of physiological synaptic pruning that normally occurs after the first year of life **(16).**

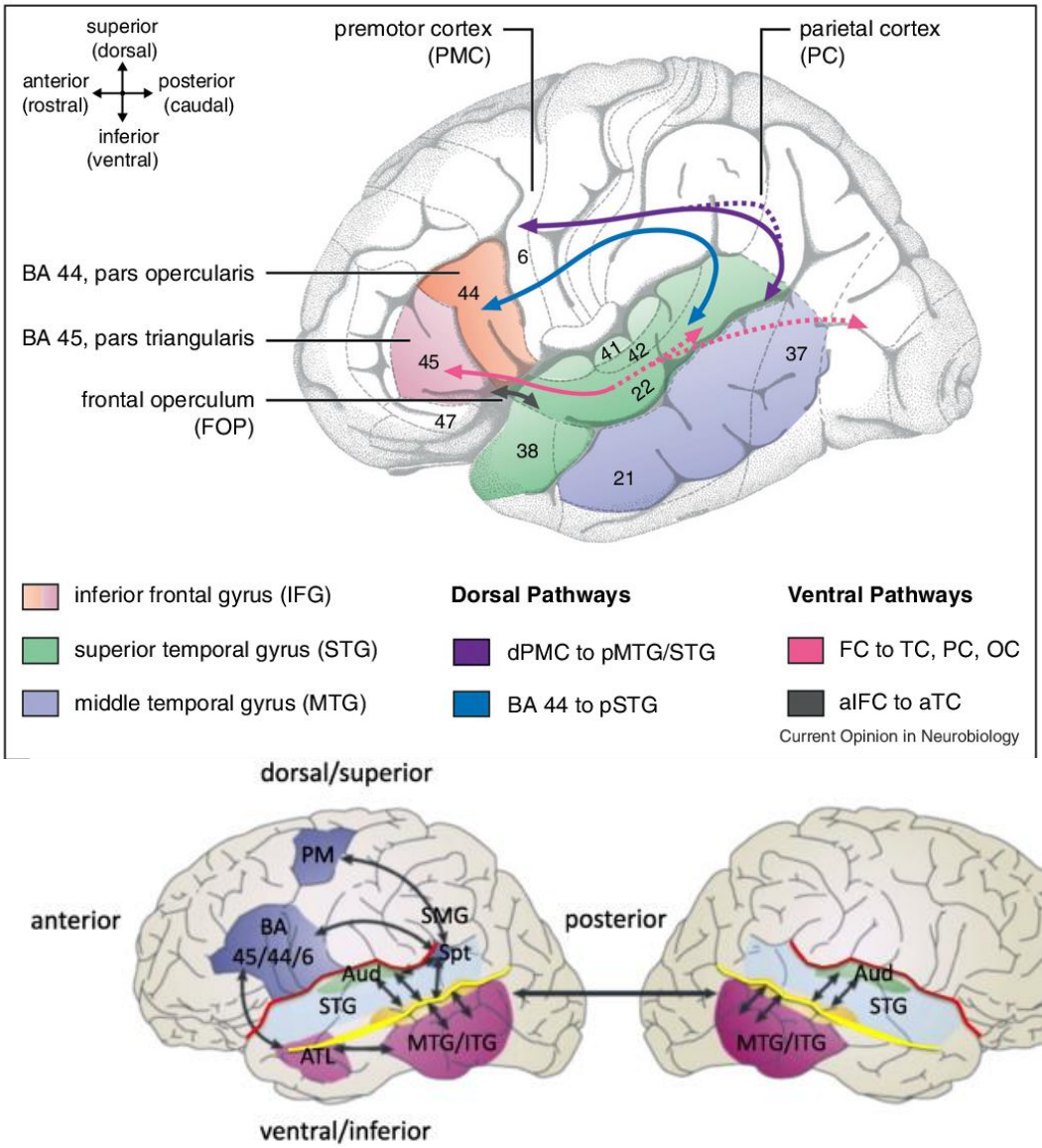

Figure (1): Neuroanatomical correlates of childhood apraxia of speech: A connectomic approach **(17).**

Characteristics of CAS:

The ASHA Committee on Apraxia of Speech in Children **(18).** described three diagnostic segmental and suprasegmental features which are:

1- Inconsistent errors on consonants and vowels in repeated productions of syllables or words:

This feature refers to *token-to-token inconsistency*, which is variability in the way a specific word is produced across multiple attempts. For example, within the same session, a child may produce *banana* as "babana," "bana," and "nana."

2- Lengthened and disrupted coarticulatory transitions between sounds and syllables:

Children with CAS may demonstrate pauses (lengthened coarticulatory transitions) between sounds and syllables (that are not part of the natural rhythm of language), resulting from challenges smooth transitions from phoneme-to-phoneme or syllable-to-syllable. When coarticulatory transitions are disrupted, the child may exhibit articulatory productions that cause speech intelligibility (e.g. frequent phoneme and syllable omissions, sound substitutions, vowel errors, voicing errors, resonance differences, difficulties producing increasingly complex phoneme sequences **(19).**

3- Inappropriate prosody, especially in the realization of lexical orphrasal stress:

Prosody is affected in all its five primary features: stress, intonation, rhythm and tone of voice **(19).**

Additional characteristics associated with CAS:

A large number of additional characteristics have been observed in children with CAS. This additional characteristics frequently cited in (but not specific to) children with CAS according to ASHA **(18).** These characteristics described in the speech and language literature can be divided to five areas as follow:

Speech characteristics:

- ❖ Inconsistency/variability which include token-to-token inconsistency (variations in repeated productions of the same word).
- ❖ Phoneme error variability (phonemes produced differently on different occasions e.g. /s/ produced as /s, t, p, h/) and Positional variability (phonemes produced correctly only in certain positions of words).
- ❖ Contextual limitations (phonemes produced correctly only in certain words or in certain facilitating coarticulatory contexts).
- ❖ Atypical errors (phonetic and phonemic errors not commonly observed in children with articulation or phonological disorders). Syllable segregation (noticeable within-word gaps between syllables).
- ❖ Limited phonetic inventory of consonants and vowels.
- ❖ Higher than expected incidence of vowelerrors (distortions, substitutions, omissions).
- ❖ Predominant use of simple syllable shapes.
- ❖ Increased errors as the complexity of the syllable shape increases.
- ❖ Connected speech is more unintelligible than the child's phonetic inventory results on a single-word articulation test suggest.
- ❖ Limited vocalizations/babbling during infancy.
- ❖ Limited diversity of babbled phonemes before the age of two.
- ❖ Differences in performance of automatic versus volitional speech.
- ❖ Groping/struggling to speak.
- ❖ Atypical levels of regression (loss of sounds or words).
- ❖ Poor diadochokinesis (reduced rates and poor coordination).

Non speech motor characteristics:

- ✓ Difficulty imitating and sequencing nonspeech oral movements.
- ✓ Possible difficulty (or history of difficulty) with feeding.
- ✓ Possible drooling.
- ✓ Later attainment of motor milestones (e.g. crawling, walking).
- ✓ Poor fine and gross motor coordination.

Suprasegmental characteristics:

- ❖ Prosodic disturbances (atypical lexical and phrasal stress, intonation, and/or rhythm of speech; excessive equal stress).
- ❖ Prolonged pauses or breaks between phonemes, syllables, and words.
- ❖ Vocal pitch differences (lack of variation in vocal pitch).
- ❖ Rate differences (typically slower rate of speech, though rate may be rapid or fluctuating).
- ❖ Loudness differences (lack of variation in vocal loudness).
- ❖ Resonance differences (hypernasality, hyponasality, or fluctuating resonance).

Linguistic characteristics:

- ✓ Slow development of language (late to produce first words).
- ✓ Receptive language (typically) exceeds expressive language, although receptive language also may be delayed.
- ✓ Morpho-syntactic difficulties (word order confusion, incorrect use or omission of grammatical morphemes, telegraphic speech).

- ✓ Social/pragmatic language difficulties.

Educational characteristics:
Greater risk of literacy difficulties (reading, spelling, writing); delayed phonological awareness skills.
Symptoms:

People with either form of CAS may have a number of different speech characteristics, or symptoms:
Distorting sounds. People with CAS may have difficulty pronouncing words correctly. Sounds, especially vowels, are often distorted. Because the speaker may not place the speech structures (e.g., tongue, jaw) quite in the right place. Sound substitutions might also occur when CAS is accompanied by aphasia.

- ♦ Making inconsistent errors in speech. For example, someone with CAS may say a difficult word correctly but then have trouble repeating it, or may be able to say a particular sound one day and have trouble with the same sound the next day.
- ♦ Groping for sounds. People with CAS often appear to be groping for the right sound or word, and may try saying a word several times before they say it correctly.
- ♦ Making errors in tone, stress, or rhythm. Another common characteristic of CAS is the incorrect use of prosody. Prosody is the rhythm and inflection of speech that we use to help express meaning. Someone who has trouble with prosody might use equal stress, segment syllables in a word, omit syllables in words and phrases, or pause inappropriately while speaking.

Children with CAS generally understand language much better than they are able to use it. Some children with the disorder may also have other speech problems, expressive language problems, or motor- skill problems **(20).**

Assessment of children with suspected CAS:

Formal tests for assessment of CAS:

1. Mansoura Arabic Test for Childhood Apraxia of speech (MATCAS)

The Arabic test battery for childhood apraxia of speech passed through the following stages, namely, design stage, pilot study and test application stage, and data analysis stage **(21).**

2. The Dynamic Evaluation of Motor Speech Skill (DEMSS):

It assesses, from imitation, the word and vowel articulation accuracy, prosody and consistency of utterance with 9 subtests, totaling 66 items **(21).**

3. Madison Speech Assessment Protocol (MSAP):

There are 4 age-based protocols; preschool, school age, adolescent and adult. The Protocol includes 15 tasks assessing speech **(14).**

4. Verbal Motor Production Assessment for Children (VMPAC) **(22):**

Designed to assess the motor functions of speech and oral structures (including tasks related to feeding) and aims to assess children aged 3–12 years.

5. The apraxia profile **(23):**

Designed to identify and describe the apraxic characteristics present in a child with speech intelligibility deficits. Normed for children aged 3 to 13. Used to assess automatic and volitional oral movements, diadochokinesis, imitation of increasingly complex words and sentences, and connected spontaneous speech. It provides a checklist of CAS characteristics.

Severity of CAS:

The following factors may use in determining severity:

1. Intelligibility. Children with more severe CAS will struggle to be intelligible even to immediate family.
2. Speech inventory (number of sounds and syllable structures) in comparison to other people of the same chronological or language age.
3. Number of features of CAS present and severity of features. These lists of features come from two sources.
4. In older children, adolescents, and adults: Difficulty saying new or longer words, avoiding speaking tasks such as using the phone, social isolation, or reduced quality of life.

(14).

Differential diagnosis of CAS:

Although significantly reduced speech intelligibility is a common characteristic of children with CAS, dysarthria, and severe phonological disorders. Disorders of neuromotor implementation would result in a dysarthria with potential involvement of respiration, phonation, resonation, and articulation. Impairment in encoding and memory processes causes phonological disorder. Transcoding problems are thought to be responsible for the deficits identified in the speech of children diagnosed with CAS. That is, there are problems in the planning and programming of the motor gestures used to produce speech **(14).**

Treatment approaches for CAS:

Therapy for apraxia is still experimental, and much of the evidence for targeted rehabilitation comes from studies on stroke patients. Compared to conventional rehabilitation for aphasia, a behavioural training program of gestural exercises has been shown to improve limb apraxia specifically and functional independence generally **(24).**

There is some evidence that using communicative gestures alongside rehabilitation for aphasia in stroke patients may improve not only the practised gestures but also unpractised gestures. Anodal stimulation using transcranial direct current (tDCS) over the left parietal cortex improved ideomotor upper limb apraxia in small samples of CBS patients and left hemispheric stroke patients **(24).**

Cholinesterase inhibitors shown to improve cognition in dementia have not been specifically studied for subcomponents of impairments such as apraxia. There is no pharmacologic therapy with evidence for improving apraxia currently available **(24).**

There are two main components of any therapy programs for CAS:

1-Because CAS is a disorder in carrying out or learning complex speech movements, so motor learning principles must be incorporated.

2-Because CAS is a disruption of central sensorimotor processes, multisensory cueing system is needed **(25).**

I- Principles of Motor Learning and Their Application to Treatment of CAS:

The process of motor learning is essential for either learning new skills (e.g. a baby learning to walk) or re-learning the lost skill(s) (e.g.an adult re-learning to walk after a stroke). Motor learning refers to a set

of processes associated with practice or experience leading to relatively permanent changes in the capacity for movement **(26).**

II- Multi-Sensory cueing:

It is hypothesized that children with CAS may have poor feed forward programs (anticipatory motor plans), and thus, rely on auditory feedback but auditory feedback is inefficient in the process of speech, it is essential that clinicians provide cueing (including tactile and proprioceptive) that facilitates greater internal representations of the motor speech plans so they are not reliant on auditory feedback for accurate productions of target utterances **(27).**

A wide variety of cues that help children gain greater motor control of speech are described below:

1- Hand cues: representing a specific articulatory placement, lip shape, or manner of production of a phoneme.

2- Cognitive cues (verbal cues): either specific verbal instructions (explanation) e.g. bite your lip and blow (for /f/) or metaphors e.g. yummy food sound for /m/.

3- Tactile cues: using touch or manipulation of the head, lips, jaw during production.

4- Visual cues: oral posturing, graphic cues, flashcards and mirror.

5- Body awareness and movement cues: like rising hands up for / **(18).**

Figure (2): picture on the left touch cue for /d/ by tapping the upper lip

(Left) ***(29)*** touch cue for /w/ (right) **(*28*).**

Treatment Programs for Children with CAS:

1. Integral Stimulation and Dynamic Temporal and Tactile Cuing (DTTC):

Integral stimulation refers to intervention approach originally developed for apraxia of speech and involves imitation "watch me, listen, and do what I do". Dynamic Temporal and Tactile Cueing (DTTC) was developed by **Strand and Skinder, (30)** as a form of integral stimulation for non-verbal children.

Description of DTTC steps:

Treatment begins with direct imitation at a slightly slower than normal rate. If the child is not producing the correct response, choral or simultaneous productions are introduced. Failure at this level would mean the clinician should slow the rate of the production and (if necessary) add tactile cues.

The tactile cues (if used) involve the clinician physically helping the child achieve the correct lip and jaw positions to begin the movements. Once the child achieves consistently correct versions of the targets, the clinician reverts to direct imitation. The rate is then slowly increased to normal. DTTC use a hierarchy of temporal delay between the clinician's model and child response (31).

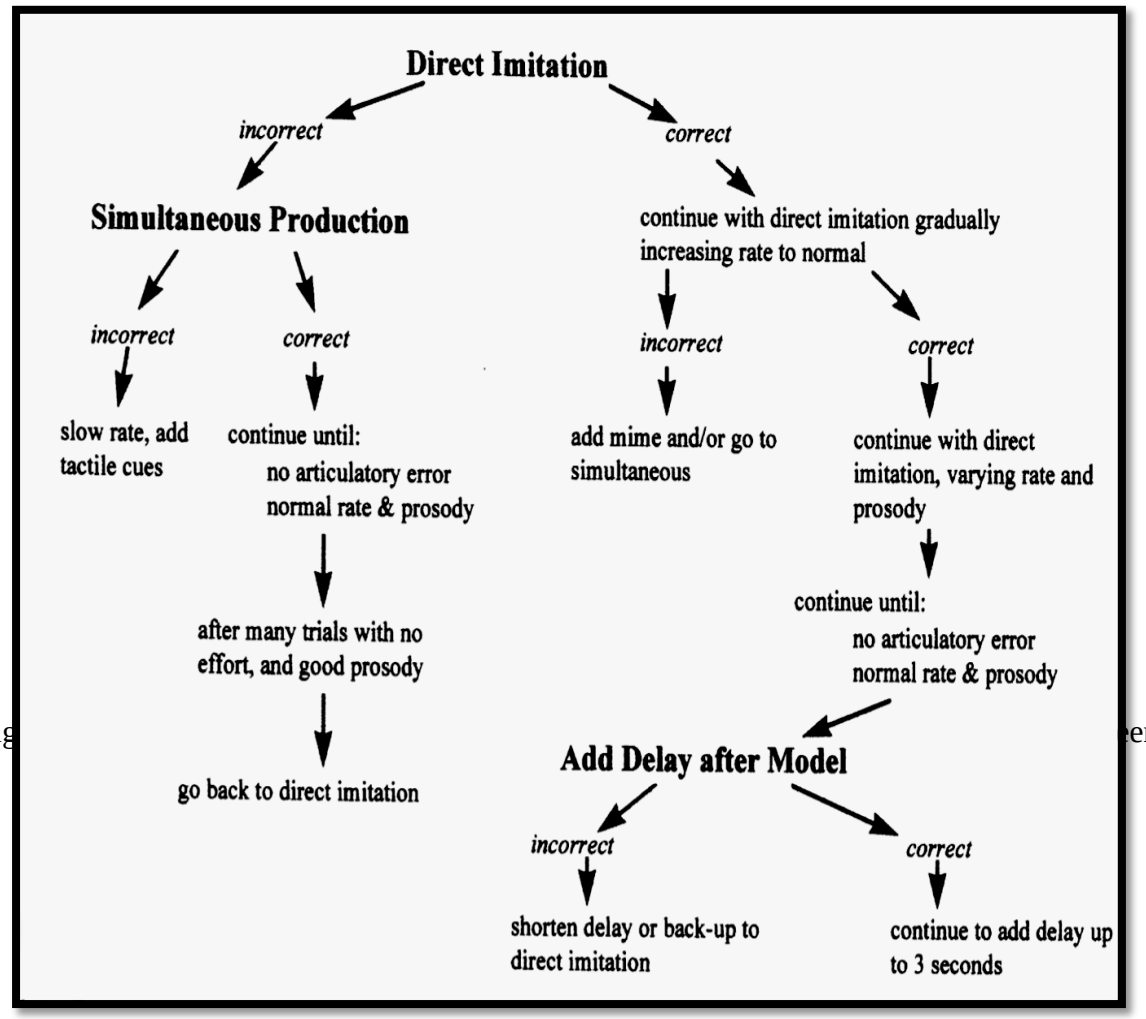

Fig en the clinician's

2. Rapid Syllable Transitions (ReST) ***(31):***

ReST is a treatment approach designed to target the core features of CAS It addresses:

(a) Segmental (sound) consistency through improving accuracy.

(b) Rapid and fluent transitions from one segment and syllable to the next.

(c) Accurate production of lexical stress.

ReST is used successfully with children aged 4-13 and it is designed to be used with children who can produce a reasonable number of sounds and use CV structured syllables. The minimum repertoire is 4 consonants and 4 vowels. So, because of the nature of the program it is recommended for children with

It is a program designed to address the motor planning and programming challenges of children with CAS. NDP3 is a “bottom up” approach to treatment that begins by helping children establish accurate motor programs for individual vowel and consonant phonemes, and then systematically build from simple to increasingly complex syllable shapes, phrases, sentences, and connected speech. The program is built on a hierarchical framework in which individual phonemes form the base and increasingly complex sequences are built on each subsequent layer. Phonological awareness skills are incorporated naturally throughout the stages of the program by including activities to address blending of syllables and

phonemes, segmentation of syllables in CVCV and multisyllabic words ***(32).***

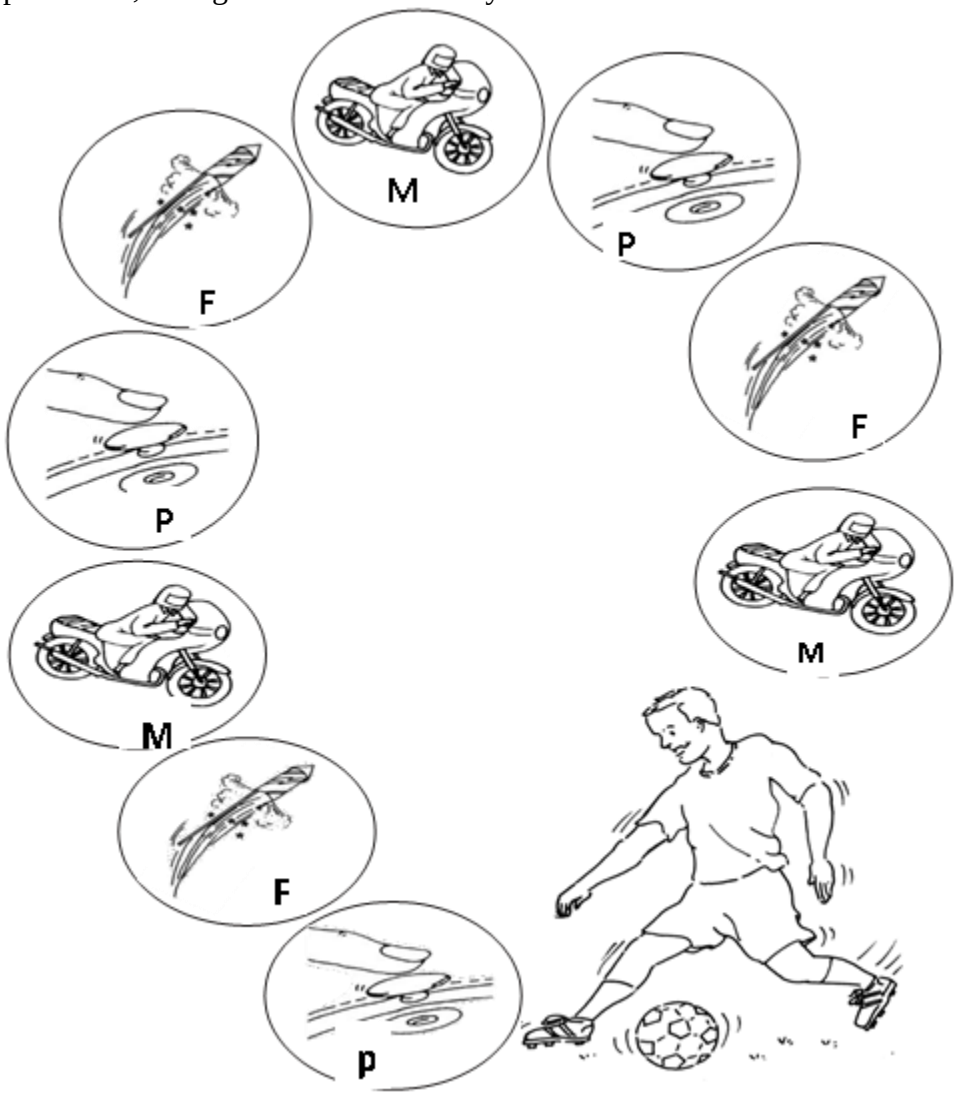

Figure (4): Work sheets of NDP3 program for consonant sequencing pictures for (/p/, /m/, and f/) to facilitate transitioning between sounds and encourage repetitive practice ***(32).***

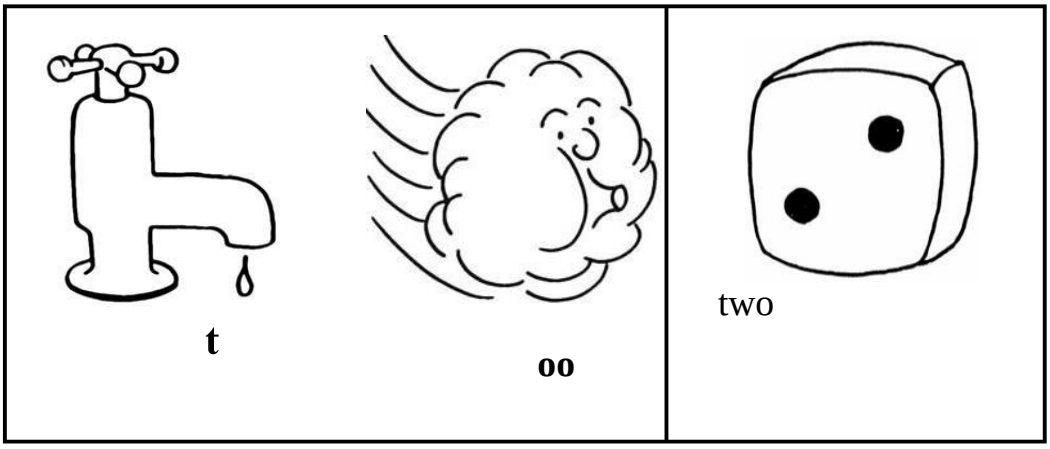

Figure (5): work sheet of NDP3 program for CV syllable

formation by transition (onset-rime) too= two ***(32).***

4-Prompts for Restructuring Oral Muscular Phonetic Targets (PROMPT):

The PROMPT system is a treatment that does not rely on imitation for success. IT is a motor- based intervention where in place, manner, movement transitions and timing of speech movements are facilitated by the precise use of tactile–kinesthetic–proprioceptive (TKP) cues or prompts on the child's articulators to facilitate speech production. This TKP input help guide accurate development of the single sound (e.g., construct a motor-phoneme template) or provide motor planning for syllables or phrases. The PROMPT clinician serves as an external programmer of speech movements **(33).**

5-Integrated Phonological Awareness Intervention **(33).**

Based on the view that CAS is a multi-level problem and drawing on evidence suggesting connections between poor phonological representations, poor speech production, poor phoneme and syllable awareness, and persisting challenges in literacy. Gillon and Moriarty **(34).**

Integrated phonological awareness incorporates targeted speech production practice into phonological awareness activities and uses letters and phonological cues to prompt speech production. It includes activities like:

- Identification of phonemes in isolation.
- Identification of initial and final phonemes in words.
- Phoneme segmentation and phoneme blending ***(35).***

6- Augmentative and alternative communication (AAC):

Alternative communication is referred to as any communication techniques other than natural speech **(18).** AAC enhances general communication effectiveness, speech production, and overall intelligibility for children with CAS. The purpose of AAC for children with CAS is to supplement the child's limited verbal expression, providing an alternative method of communication, while facilitating communication interactions ***(35).***

Types of (ACC):

Unaided ACC: includes methods of communication that do not require additional equipment, such as manual sign, body language, finger spelling, gesturing, and facial expressions.

Aided ACC: includes methods of communication that require additional equipment aids or tools, such as picture exchange, communication books and voice output.

7-The Kaufman Speech to Language Protocol (K-SLP) **(36).:**

It is a set of techniques that involves reinforcement of *successive approximation*, and employs phonological processes to simplify words. Any word can be broken down to the point of success. The task of verbal imitation is simplified into smaller units that can be more easily mastered and utilized immediately and functionally.

K-SLP uses multisensory cues to assist the child with initial success, and then fade the cues so that the ultimate response is as spontaneous as possible.

Word approximations are temporary and functional and can be done by two mechanisms: intentional phonological process and word chaining **(36).**

ReST (Rapid Syllable Transition Treatment) and IPA (Integrated Phonological Awareness) are suitable for less severe and/or older children. DTTC (Dynamic Temporal and Tactile Cueing) and NDP3 (Nuffield Dyspraxia Programme 3rd edition) are more suitable for younger and/or more severe children. Resources and clinician training for ReST, IPA and DTTC are freely available on the internet and NDP3 is a kit which can be purchased from the UK **(36).**

It Was found that ultrasound biofeedback may be viable treatment options for school-age children with CAS. Ultrasound biofeedback involves training articulatory patterns using real-time visual displays of the tongue. ReST and ultrasound biofeedback were equally effective. Either ReST or ultrasound biofeedback may be viable treatment options for school-age children with CAS.

The CAS treatment evidence shows that therapy 4 times a week in blocks of 12-16 sessions followed by a 4-6 week break from therapy is optimal **(37). Namasivayam et al. (38**) have showed that the greater the treatment intensity the more effective the therapy and the more efficient the progress. A minimum of two sessions a week has been shown to work clinically. Session length ideally should be 45-60 minutes but will depend on both the child and the treatment selected.

Prognosis of CAS:

Because clear results of CAS prognosis are not handy in the literature, the oral reports of therapists on the considerable challenge of treatment have been used as the last resort. In a Swedish survey, the majority of the respondents believed that children with CAS improve rather slowly. The majority of participants voted for the constancy of speech results at school age; more than half of them, as well, reported the children resisted treatment and had a backslide of the symptoms after a remission period **(39).**

Conflict of Interest: None

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