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TREATMENT OF ADYNAMIA IN APHASIA

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1. ABSTRACT

Transcortical motor aphasia (TCMA) is an acquired impairment of language expression that occurs following neurologic damage that affects left frontal cortex and spares perisylvian regions. In some individuals with TCMA, verbal expression is rendered nonfluent due to difficulty spontaneously initiating and elaborating upon verbal messages. Nonfluency arises from impaired activation of intended messages and inhibition of competing verbal expressions. This impairment of the intentional aspects of language expression can be termed 'adynamia.' Because adynamic forms of TCMA occur infrequently, few systematic treatment investigations have been reported for this condition. Behavioral treatments have been proposed to engage intact frontal regions to improve the ability to initiate spontaneous verbal expression. Some data suggest that nonsymbolic limb movements performed in the context of speaking activities, a form of what Luria termed gestural reorganization, may improve the adynamic verbal expression. (1) In addition, the influence of pharmacologic treatment with bromocriptine, a dopaminergic agonist, has been considered for its effects on verbal nonfluency in aphasia. Individuals classified as TCMA are more likely to benefit than those with other forms of nonfluent aphasia, suggesting an influence of bromocriptine on circuits necessary to activate spontaneous language. Additional studies are warranted that contrast behavioral and pharmacologic interventions to determine optimal conditions to improve verbal expression in adynamic forms of aphasia.

2. INTRODUCTION

Language knowledge encompasses a complex system of rules and representations involved in syntax (grammar), lexical-semantics (word meanings), and phonology (speech sounds). Use of language knowledge allows for auditory comprehension, spontaneous verbal expression, and repetition of words and sentences. In the majority of right- and left-handed individuals, the left cerebral hemisphere mediates language abilities.(2) Aphasia is an acquired impairment affecting comprehension and expression of language that may result from damage affecting the left hemisphere.(3) Over 100 years ago, Wernicke and later Lichtheim developed the notion that distinct patterns of language dysfunction may emerge, depending on the location of neurologic damage within the left hemisphere and the aspects of language affected. (4,5)

Assessing language in the primary areas of auditory comprehension, spontaneous verbal expression, repetition, and confrontation naming of pictures, it is possible to identify general patterns or classifications of language breakdown.(6,7) For example, some individuals with aphasia have nonfluent spontaneous verbal expression and repetition due to deficits of grammar and phonology, impaired naming, and better performance for auditory comprehension of language, a pattern referred to as Broca's aphasia. Nonfluent patterns of language breakdown tend to be associated with damage affecting portions of the left frontal cortex and sparing temporal-parietal regions.(8) In contrast, other individuals with aphasia have fluent verbal expression and repetition, and impaired naming, all of which may be fraught with errors in the choice of words and sounds to the extent that verbal utterances may be unintelligible. They also have difficulty with auditory comprehension, even for single words. This pattern, referred to as Wernicke's aphasia, arises due to semantic and phonologic deficits in language. Fluent forms of aphasia tend to be associated with lesions of the left temporal-parietal regions, sparing frontal cortex. (8)

Whereas repetition is impaired in both Broca's and Wernicke's aphasia, some individuals with aphasia have unexpectedly spared repetition abilities relative to

their noticeable difficulties with spontaneous verbal expression. These types of aphasia have been termed the transcortical aphasias.(4,5) Transcortical sensory aphasia, the correlate of Wernicke's aphasia, is characterized by fluent verbal expression, impaired auditory comprehension, and intact repetition. Transcortical motor aphasia parallels the nonfluent verbal expression and relatively preserved auditory comprehension of Broca's aphasia, but with spared repetition. Benson and Ardila called the transcortical aphasias 'extrasylvian' syndromes, as they are associated with left hemisphere lesions that spare the perisylvian cortex responsible for phonologic processing. (2) Transcortical sensory aphasia is characterized by an impairment of semantic processing with retained phonological abilities. Likewise, one might expect that transcortical motor aphasia should represent a grammatical impairment with retained phonological abilities. This is not always the case, however, as impairments underlying TCMA may take a number of forms, as will be described below. (9)

3. NONFLUENCY IN APHASIA

For individuals with nonfluent forms of aphasia, the overall amount of verbal output is reduced such that the number of words spoken per minute is severely limited (less than 50 words per minute compared to the typical 150-200 words per minute).(10) The reduction in verbal fluency in some individuals with aphasia indicates a grammatical problem, as they omit function words (e.g., articles, auxiliaries, prepositions) and word endings (e.g., plural -s, progressive -ing), giving a telegraphic quality to utterances. For others, verbal fluency is disrupted by a phonetic-articulatory deficit, such that the individual struggles to initiate and sequence the complex series of speech sounds that constitute words and sentences. Accompanying grammatical and phonetic deficits can come difficulty in the use of prosody or melody of language, and speech is flat and monotone. (7) Finally, verbal expression can be rendered nonfluent because of difficulty initiating and elaborating verbal messages. The utterances that are finally produced are well-formed sentences in terms of the grammatical and articulatory characteristics, but the nonfluency arises in the inability to get the utterance started.

The key characteristic of the language deficit in TCMA is the nonfluent verbal expression. Systematic examinations have shown that nonfluency arises for different reasons across individuals whose pattern of language impairment can be characterized as TCMA. (11) Some individuals with TCMA are nonfluent because of a grammatical deficit combined with significant difficulty with word finding during the course of sentence generation. (9) Word retrieval difficulty is usually evident in confrontation picture naming as well. That is, in this subset of individuals with TCMA, nonfluency arises due to a linguistic disturbance. In contrast, nonfluency in other individuals classified as TCMA appears due to difficulty initiating and carrying out a verbal message. (12) The sentences they eventually utter are usually grammatically and phonologically well-formed. These individuals want to communicate, but they cannot seem to gather together the thoughts and words to evoke an utterance. If they can get words or phrases started, they are often succinct and lack elaboration of their messages. That is, the nonfluency in this subset of individuals with TCMA represents an impairment of the intentional dynamic aspects of verbal expression and not to linguistic breakdown, that is, adynamic aphasia. Some individuals who are initially mute quickly resolve to this form of TCMA, demonstrating intact repetition abilities and a tendency for echolalia in which they echo back whole sentences or fragments of sentences as they attempt to respond to queries. (13) Individuals with adynamic aphasia may have no trouble with word retrieval in confrontation picture naming tasks, but they have great difficulty retrieving words in word list generation tasks, such as thinking of all the words that start with a particular letter. (14) Therefore, although some consider TCMA and adynamic aphasia to be synonymous terms, adynamic aphasia constitutes a specific form of aphasia arising from impairment of a non-linguistic system that influences the use of language knowledge in spontaneous speaking situations.

3.1. Mechanisms of Adynamia in Aphasia

Luria described dynamic aphasia as a pattern of intact language abilities for repetition and auditory comprehension in the context of nonfluent spontaneous verbal expression due to difficulty with "speech initiative" (16, p. 212). Language difficulty becomes evident as the individual is required to internally generate or compose a message, rather than to react to a given stimulus. Others have referred to this pattern of language impairment as 'adynamic aphasia.' (17) Researchers have proposed a number of mechanisms for the significant nonfluency in individuals with adynamic forms of TCMA. (18,19) In order to generate an utterance, we must first conceptualize, at a preverbal level, a message to be spoken. (19) Some have proposed that nonfluency in adynamic aphasia represents a failure in the internal generation of ideas and concepts, a breakdown that may be specific to verbal generation and planning. (20) Once an idea is generated, the lexical-semantic and syntactic systems must be activated to develop an intended sentence. Robinson and colleagues noted that their patient with adynamic aphasia actually seemed impaired by the fact that multiple verbal response options were activated in the context of generating an utterance. (18) That is, rather than a lack of concepts generated, their patient's nonfluency was associated with failure to specify and select one appropriate verbal response when many possibilities have become activated. This overactivation and lack of inhibition of unwanted responses impeded the patient's ability to generate an intended utterance. This proposal is similar to that of Gold and colleagues who suggested that their patient with adynamic aphasia failed to develop an effective semantic or lexical search strategy. (17) A search strategy would require the ability to select a specific response and inhibit unwanted alternatives. Although distinct dysfunctions have been espoused to account for adynamia in spontaneous verbal expression, that is, failure to generate ideas and failure to select from and inhibit a number of competing verbal responses, both accounts are encompassed in the model of

frontal lobe functions expounded by Alexander and colleagues. (9) They proposed that the prefrontal cortex provides the drive for cognitive function, meaning the "ability to initiate, modulate or inhibit cerebral activities." (p. 658) Adynamic aphasia appears to result when there is either failure to initiate language processes or failure to inhibit extraneous language information that becomes active when spontaneously generating a verbal expression. In either case, initiation failure or inhibition failure, the individual is prevented from generating sentences in situations in which language must be internally-evoked and verbal output is rendered nonfluent.

3.2. Neural Correlates of Adynamia in Transcortical Motor Aphasia

TCMA can be observed in individuals with bilateral frontal/subcortical disease (e.g., progressive supranuclear palsy, encephalitis), as well as those with circumscribed lesions of the left frontal cortex (e.g., following stroke, tumor). (21,22) Distinct left hemisphere lesions may lead to TCMA, not all of which represent the adynamic form of TCMA. (20,23) Although some patients with chronic aphasia can be classified as TCMA, these were typically patients who initially presented with Broca's aphasia. (25) If their left frontal opercular lesions spare critical frontal articulatory regions (e.g., insula), repetition abilities may recover sufficiently to look more like TCMA in chronic stages of the aphasia. TCMA also can occur acutely following dorsolateral frontal lesions anterior and superior to Broca's area.24 This type of lesion is associated with impairments of word retrieval and syntactic deficits in sentence production that are linguistic in nature and do not represent adynamic aphasia. (9).

Lesions associated with adynamic forms of TCMA particularly affect mesial frontal cortex, at times including supplementary motor area (SMA) or subcortical white matter pathways connecting SMA to Broca's area. (9) Less commonly, adynamia may be observed following thalamic infarction. (26) Kertesz noted that SMA plays a critical role in providing limbic input during the initiation of speech activity. (27) SMA lesions disrupt the dopaminergic pathways critical for motor activation, including activation to initiate communication behaviors, leading some to argue that the resulting dynamic dysfunction should not be termed aphasia at all, as the impairment is motoric rather than linguistic. De La Sayette and colleagues also suggested that motor activation may be disrupted by loss of thalamo-cortical interactions. (26) In the event of a large lesion encompassing SMA, language initiation may be impaired not only in motor activation, but also in the formulation stages of language expression. (9) Differences in the extent of the mesial frontal involvement may account for the range of impairments in concept generation and verbal activation and inhibition that have been attributed to adynamic aphasia. Patients with the adynamic form of TCMA in acute phases of their aphasia tend to demonstrate significant recovery as motor activation and language formulation abilities improve. (27,28).

4. TREATMENT OF ADYNAMIA IN APHASIA

Treatment for aphasia is often provided by speech-language pathologists who typically incorporate behavioral methods to improve language and communication skills. (29-31) Within behavioral interventions, Rothi distinguishes between restorative treatments that are intended to re-engage language systems that are usually involved in the process of language functioning, and vicariative treatments that promote language recovery by recruiting other intact neural and cognitive systems (e.g., prosody, right hemisphere) to mediate or enhance aspects of language processing. (1,32) Finally, compensatory behavioral techniques are implemented to promote the use of alternative means to communicate, circumventing impaired language skills. Specific behavioral methods chosen vary in relation to a number of medical, neurological, cognitive and psychosocial factors. Foremost among these factors is the patient's constellation of aphasic symptoms. In the case of adynamic forms of TCMA, treatment needs to target impaired spontaneous verbal expression. In addition to behavioral treatment, clinical researchers have examined effects of pharmacologic interventions to enhance aphasia recovery, including recovery of verbal fluency. (33-45)

4.1. Behavioral Treatments for Adynamia

Relatively few experimental studies have examined behavioral treatments for impaired spontaneous verbal expression in individuals with adynamic forms of TCMA. (12) Anecdotal reports indicate that restorative treatment methods employing repetition, oral reading, or even generation of verbal responses to pictures are less effective for leading to long term changes in spontaneous verbal expression, as these tasks tend to emphasize externally-evoked pathways to verbal production, which are less impaired in adynamic aphasia than internally-based language initiation. (46) However, clinicians often encourage patients with adynamic aphasia to use externally-evoked verbal tasks in compensatory ways as external cues to encourage verbalization and fluency. (16) For example, the echolalic repetition of portions of a question often seen in individuals with adynamic aphasia can be exploited to provide the patient with a "running start" to initiate a novel verbal response. Family members can be trained to provide a partial sentence or carrier phrase (an open-ended phrase that can be completed in a variety of contexts, e.g., "I was thinking that….) for the patient to repeat and then complete with an intended message. Written scripts can be prepared in advance of predictable situations and patients can refer to prepared sentences or phrases to read when verbalizing in these situations. Series of pictures can be used to promote verbalization of a story or longer verbal discourse. Although these strategies are often used clinically, no investigations have evaluated systematically how effective these compensatory measures are for improving verbal fluency in individuals with adynamic aphasia.

Not all verbal situations can be predicted and prompted. Therefore other treatment methods have been explored to engage language initiation mechanisms

internally and invoke long-term changes in language expression abilities for individuals with adynamic aphasia. Luria suggested that motor techniques, such as moving the limbs and touching pieces of paper during the course of speaking, may be useful to mediate verbal initiation, a method known as intersystemic gestural reorganization. (1,47) Limb movements implemented during speaking tasks may include simple repetitive tapping movements or complex sequences of movements produced in relation to the rhythm of an utterance. Hanlon and colleagues evaluated the influence of right and left limb pointing movements during picture naming in individuals with various forms of aphasia, including some classified as TCMA. (48) They found that movement of the right proximal limb was associated with greater picture naming accuracy than was left proximal limb movement in individuals with nonfluent forms of aphasia, including at least one participant with TCMA. The long-term effects of limb movements on word retrieval were not evaluated.

Picard and Strick reported that complex limb movements engage prefrontal cortex, whereas simple repetitive tapping movements engage primary motor and premotor cortex. (49) With these notions in mind, Crosson proposed that complex movements performed with the left hand in left space may activate right hemisphere mechanisms for movement initiation and encourage language initiation with intact right hemisphere frontal cortex. (50) In what Crosson and colleagues have termed 'intentional' language treatment, they have reported that training using complex nonsymbolic limb movements during picture naming practice led to increased accuracy of picture naming in some individuals with nonfluent aphasia. (50,51)They have not reported data for individuals with TCMA, however.

Individuals with adynamic forms of TCMA are likely to have less trouble with word retrieval in picture naming than they do with spontaneous generation of sentence-length material. Therefore treatment for adynamic aphasia needs to incorporate tasks requiring sentence generation. Rothi, recognizing the notions of Luria, advocated the use of rhythmic limb movements to improve verbal fluency in patients with TCMA, including adynamic aphasia. (1,12) One method that comes to mind in this regard is Melodic Intonation Therapy (MIT), a sentence production treatment technique used commonly in individuals with nonfluent aphasia. (29,52) MIT involves a systematic protocol in which patients practice use of highly intoned verbal sentences as they tap with the left hand in one location in space. A recent investigation reported that the tapping movements alone are as effective as the intonational component of MIT for improving verbal production in individuals with nonfluent aphasia. (53) However, MIT is reported to be less effective in individuals with transcortical aphasias who can repeat well, but have difficulty spontaneously generating utterances. (52)

In keeping with the notions of Crosson and colleagues, facilitation of verbal expression in adynamic aphasia may require treatment incorporating complex limb movements rather than the simple tapping movements employed in MIT. (50,51) In one experimental treatment study with a patient with the adynamic form of TCMA, Raymer and colleagues examined the effectiveness of a treatment incorporating complex nonsymbolic limb tapping movements to improve sentence generation. (54) Unlike MIT, in which tapping occurs repeatedly in one place, Raymer et al. required their patient to alternate tapping movements back and forth between two colored blocks during sentence generation practice. The patient made these movements with the left hand in left space in an effort to engage prefrontal cortex in the sentence generation process. (48) With practice, the patient with TCMA improved in generation of timely, well-formed sentences as well as in the number of words per minute generated in longer narratives; no improvements were evident in a control word list generation task. The positive results of intentional movement training on verbal expression await replication in other similar participants with aphasia. These preliminary data suggest that use of nonsymbolic limb movements may be a useful behavioral intervention strategy for improving spontaneous verbal expression in individuals with adynamic forms of TCMA.

4.2. Pharmacologic Treatment

In addition to behavioral methods, researchers have explored the effectiveness of pharmacologic treatments in the restitution of language in some patients with aphasia. (33-35) The nonfluency or reduced verbal generativity often seen in patients with adynamic forms of TCMA arise due to reduced dopaminergic input to the frontal lobe following strategically-placed left frontal lesions. (42) Therefore, the use of dopaminergic agents may be indicated to augment disrupted neural circuits and induce improved verbal fluency in some patients with nonfluent aphasia, particularly adynamic aphasia. (36,41) One dopaminergic drug that has been explored for its effects on aphasia recovery is bromocriptine. Table 1 summarizes findings of a number of investigations that have examined the effects of bromocriptine in individuals with aphasia. Following administration of bromocriptine, some studies have reported significant improvements in verbal fluency measures. (36-38,41-42) Those studies reporting positive effects of bromocriptine have documented increased naming accuracy with decreased naming latency, reduction in pauses and hesitations in connected speech, and increased initiation of verbalization. (36-37,41-42,44) Other studies have found no reduction in hesitancy or naming improvement, however. (38) One double-blind placebo-controlled investigation reported limited or no improvements in verbal fluency measures associated with bromocriptine use. (39,43,45) In general, these pharmacologic studies have demonstrated little improvement in standardized aphasia tests and other language measures such as length of utterances and grammatical complexity.(38-40,44)

The divergent findings regarding the effectiveness of bromocriptine for ameliorating nonfluency in aphasia may relate in part to the distinct neural and cognitive bases for nonfluent verbal expression among individuals with aphasia. Bromocriptine is likely to have a less powerful treatment effect when critical language

Table 1. Summary of investigations of bromocriptine in individuals with nonfluent aphasia

N/Aphasia	Design/Dosage	Findings	Reference
1 TCMA	Open-label 6 wks/30 mg	Decreased pauses in conversation; increased picture naming	36
2 Broca 2 TCMA	Open-label 10 wks/15 mg	Picture naming: Improved word retrieval in 4/4; Decreased response time in 3/4	37
1 Broca's 1 TCMA	Open-label 5 wks/30 mg	#1: Improved repetition, naming; Increased utterance length #2: Increased utterance length Improved content of utterances	38
1 TCMA	Single-blind 7 wks/15 mg	Increased number of words; No change in naming; fluency	39
2 Broca's 1 global 1 TCMA	4 wks/25 mg	No changes in speech samples	40
1 TCMA	Open-label 3 wks/6 wks 20 mg	Improved letter category fluency; Increased words per minute No change in control measures	41
4 TCMA (1 severe) 1 Global (severe) 2 Broca's (severe) 2 Broca's	Open-label 14 wks/15-60 mg	Increased words in picture description; Decreased pauses in 3 moderately-impaired TCMAs; not in severe patients	42
2 Broca's 3 TCMA 2 Anomic	Double-blind 16 wks/3/75-60 mg	Grouped data: No significant increases in fluency or naming measures	43
1 TCMA 1 mixed 1 Broca's	Open-label 30-40 mg	#1 reported in Albert et al. #2&#3: somewhat increased initiations; and naming	44
1 TCMA	Open-label	Reported unsuccessful	45

regions are damaged or destroyed, as is often seen in individuals with nonfluent Broca's aphasia. Additional dopamine in the brain would not be expected to have a modulating effect when regions responsible for storage of language knowledge are lost. In contrast, bromocriptine would be anticipated to have more robust effects in individuals whose nonfluent aphasia arises from loss of dopaminergic input to intact frontal language regions, as is the case in individuals with adynamic forms of TCMA. (37,41) Administration of bromocriptine addresses the underlying pathophysiology of the impaired verbal expression system in adynamic TCMA. Gold and colleagues proposed that bromocriptine acts somewhat like a spotlight in the language system, facilitating the activation of intended words while suppressing activation of extraneous words outside of the spotlight. (37) When studies group participants only according to level of fluency, without examining the overall aphasia pattern and lesion locations more closely, effects of bromocriptine potentially are masked. Alexander noted anecdotally that patients who benefit most from bromocriptine are almost exclusively those with TCMA. (13) Although caution may be in order, it appears that bromocriptine, if it has positive effects for recovery of aphasia, is most likely to be effective in individuals with adynamic forms of TCMA, and less so in other forms of nonfluent aphasia.

5. PERSPECTIVE

Adynamic aphasia is a relatively uncommon occurrence among individuals with acquired language impairments. It is usually reported in individuals with left mesial frontal cortex lesions. Treatment for the adynamia in aphasia has been only infrequently examined in experimental investigations. Intentional nonsymbolic movements paired with verbal production provides a promising direction for behavioral intervention for clinicians addressing the decreased spontaneous verbal expression in individuals with adynamic forms of TCMA. In addition, the pharmacologic agent bromocriptine may be an appropriate adjutant treatment for physicians to consider in the treatment for individuals with adynamic aphasia. Future studies need to contrast behavioral, pharmacologic, and combined behavioral plus pharmacologic interventions to determine the optimum conditions for maximizing gains in spontaneous verbal expression in individuals with adynamic aphasia. Clinicians must also explore novel treatment approaches to enhance the ability of these individuals to internally generate language to communicate.

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