Transcranial Stimulation, An Adjuvant Therapy for Dysphasia

An Essay

Submitted for the partial fulfillment of the master degree in

Phoniatrics

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2010

List of Contents

Abbreviations…..…… ii
Definitions…….…….……. iv
List of Figures……..…….. v
List of Tables ……..... vi
Introduction…….…….…. 1
Aim of the work……..…….……. 5
Chapter (1): Dysphasia…….……..... 6
Chapter (2): Rehabilitation of dysphasia……..... 25
Chapter (3): Transcranial Magnetic Stimulation ……...… 42
Chapter (4): Role of TMS in Treatment of Dysphasia.….. 68
Chapter (5): Transcranial Direct Current Stimulation …… 80
Chapter (6): Role of tDCS in Management of Dysphasia... 94
Conclusion……..… 99
Summary……..…..……. 102
References…….……..…...… 108
Arabic Summary

List of Figures

Figure (3-1): Transcranial magnetic stimulation equipment and procedure……..… 52

Figure (4-1): Regions of interest..... 70

Figure (5-1): Transcranial direct current stimulation…….... 85

List of Tables

Table (6-1): Comparison between TMS and TDCS……97

INTRODUCTION

Cognitive dysfunction after a brain insult constitutes one of the major causes of disability worldwide. It exerts a major impact on the lives of affected individuals and their families, and represents a major public health and financial burden for society. Therefore, the rehabilitation of disorders of cognitive functions related to language, attention, or memory is clinically important ***(Cappa et al., 2005).***

The central nervous system responds dynamically to degenerative or focal lesion-induced cognitive deficits, this explains the clinical observation of disturbed or lost functions that can be partially or fully restored. With the maturing fields in cognitive neurosciences, the combination of different expertises and methodologies has yielded a new interdisciplinary approach for intervention ***(Cicerone et al., 2005).***

Studies of functional neuroimaging have shown that cerebral reorganization may occur after specific rehabilitation interventions. Moreover, a number of studies indicate that interacting with cortical activity by means of cortical stimulation can positively affect the cognitive performance of patients affected by dysphasia, and other

cognitive disorders ***(Strangman et al., 2005).***

Dysphasia is an acquired language disorder in which there is an impairment of any language modality, after its full acquisition. This may include difficulty in producing or comprehending spoken or written language. The most common cause of dysphasia is cerebrovascular disease, particularly cerebral infarction ***(Inatomi et al., 2008).***Other structural pathologies (infection, trauma, neoplasm) and certain neurodegenerative diseases e.g., Alzheimer's or Parkinson's disease can also cause dysphasia ***(Noppeney et al., 2007).***

It can be divided into three categories: fluent dysphasia, also called receptive dysphasia, nonfluent dysphasia, also called expressive dysphasia, global dysphasia, and "Pure" dysphasia (e.g., alexia, agraphia and pure word deafness) ***(Kolb & Whishaw, 2003).***

The assessment must be broad enough to detect subtle disorders of language in patients in whom dysphasia is suspected. Each component of language should be tested individually and thoroughly. Components of language examination include assessments of spontaneous speech,

naming, repetition, comprehension, reading, and writing ***(Devinsky & D'Esposi, 2004).***

The prognosis of those with dysphasia varies widely, and is dependent upon age of the patient, site and size of lesion, and type of dysphasia. In most individuals with expressive dysphasia, the majority of recovery is seen within the first year following a stroke or injury ***(Bakheit, 2007).***

The following new lines of treatments are currently being studied to determine the best possible method for treating dysphasia:

-Constraint-induced therapy: in which the patient is not allowed to use other forms of communication such as drawing or body language but he is constrained to use spoken words only. It works by the mechanism of increased neuroplasticity. By constraining an individual to use speech only, it is believed that his brain can reestablish old neural pathways and recruit new neural pathways to compensate for lost function ***(Friedemann & Berthier, 2008).***

-Transcranial magnetic stimulation (TMS) and transcranial direct current stimulation (TDCS): which are used to create

electrical currents in specified cortical regions by a painless and noninvasive method. These techniques work by suppressing the inhibition process in certain areas of the brain. By suppressing the inhibition of neurons by external factors, the targeted area of the brain may be reactivated and thereby recruited to compensate for lost function. Research has shown that patients can show increased object naming ability with regular stimulation than patients in therapy without stimulation. Furthermore, this improvement has been proven to be permanent and remains upon the completion of therapy ***(Naeser et al., 2005) & (Monti et al., 2008).***

-Lastly, Stem cells transplantation: results from laboratories studying neural regeneration are promising. They have been using cell implantation into living brain to facilitate recovery from brain damage. Their studies indicate that cell transplantation, which promotes the restoration of brain tissue after brain injury, may benefit patients with dysphasia ***(Korsukewitz et al., 2006).***

AIM OF THE WORK

The aim of this essay is to make a comprehensive review about the role of transcranial stimulation as an adjuvant therapy for dysphasia in order to highlight this therapeutic technique, hoping that it will be beneficial for patients.

Chapter (I)

DYSPHASIA

Neuroanatomy and Language centers:

A large, complex neurocognitive network, usually located in the left hemisphere, subserves the capacity for human language. The language network comprises areas of perisylvian cortex, including the classical language areas of Broca and Wernicke. While these are not anatomically discrete areas, important neural networks subserving critical language function have been identified:

Broca's area or Brodmann's area 44 in the posterior inferior frontal gyrus innervates adjacent motor neurons subserving the mouth and larynx, and controls the output of spoken language. ***Wernicke's area or Brodmann's area 22***, comprising the posterior two-thirds of the superior temporal gyrus, receives information from the auditory cortex and accesses a network of cortical associations to assign word meanings. The angular gyrus in the inferior parietal lobe is adjacent to visual receptive areas and subserves the perception of written language, as well as other language

processing functions. Other regions of the cerebrum contribute importantly to normal language. These include the insula, which is integral to normal articulation, several frontal and temporal lobe regions (including the posterior middle temporal gyrus and underlying white matter, the anterior superior temporal gyrus, the superior temporal sulcus and angular gyrus, mid-frontal cortex in Brodmann's area 46, and Brodmann's area 47 of the inferior frontal gyrus) that support sentence-level processing, and vast regions of temporal, occipital, and parietal cortex that support knowledge of words and their meanings e.g. the middle temporal gyrus ***(Dronkers et al., 2004).***

Although it is likely that subcortical nuclei make a contribution to normal language performance, evidence from perfusion weighted imaging indicates that dysphasic syndromes associated with ischemic subcortical lesions are often accompanied by perfusion defects that involve cortical language regions. The fact that these subcortical dysphasias have been associated with a better prognosis than cortical varieties may be explained by their tendency to resolve with restoration of cortical perfusion ***(de Boissezon et al., 2005).***

Finally, Language disabilities are also seen with cerebellar lesions as the cerebellum contributes in several language

parameters such as lexical retrieval and syntax so there is increasing evidence that cerebellar lesions may result in linguistic deficits, including grammatical disturbances (crossed cerebral diaschisis processes, reflecting a functional depression of supratentorial language areas due to reduced input via cerebellocortical pathways, might represent the relevant pathomechanism for linguistic deficits associated with cerebellar pathology) ***(Karaci et al., 2008).***

Dysphasia:

Dysphasia is an acquired language disorder in which there is an impairment of any language modality, disrupting a formerly normal, pointing to a brain damage of the dominant hemisphere. This may include difficulty in producing or comprehending spoken or written language. Dysphasia may co-occur with speech disorders such as dysarthria or apraxia of speech, which also result from brain damage ***(Devinsky & D'Esposi, 2004).***

Etiology:

-Vascular Causes:

All vascular causes of stroke are characterized by sudden or acute onset of impairment. This is subsequent to reduced blood flow (ischemia) due to thrombosis or embolus, or rupture of a vessel leading to bleeding into the brain or subarachnoid space. Ischemic stroke ensues when a focal area of the brain receives inadequate oxygen and glucose for function, and eventually too little to survive. Intracerebral hemorrhage causes symptoms similar to that of ischemic stroke. Subdural hematoma is a distinctly unusual, but possible, etiology of dysphasia. Subarachnoid hemorrhage rarely causes immediate dysphasia. It can cause delayed focal ischemia due to vasospasm that can lead to dysphasia caused by infarct or poor perfusion ***(Bakheit, 2007).***

Transient episodes of dysphasia may occur with transient cerebral ischemia, and is considered as a risk factor that identifies an individual at a relatively high risk of stroke in the next few days and weeks ***(Ois et al., 2008).***

-Infectious Causes:

Herpes Simplex encephalitis has a predilection for the medial and inferior temporal cortex, and frequently

causes a striking dysphasia with severe impairments in word meaning. The language profile may be very similar to Wernicke's dysphasia, but herpes encephalitis is associated with fever, malaise and other systemic symptoms ***(Noppeney et al., 2007).***

Bacterial infection or cerebral abscess may also cause dysphasia ***(Commondoor et al., 2009).***

-Neurodegenerative Causes:

Primary progressive dysphasia is a disorder of declining language that is a frequent presentation of neurodegenerative diseases such as frontotemporal lobar degeneration. Three variants are recognized: progressive nonfluent dysphasia, semantic dementia and logopenic progressive dysphasia (characterized by slow rate of speech, word-finding difficulties and impaired syntactic comprehension but relatively preserved syntax and phonology) are all considered primary progressive dysphasias ***(Grossman, 2010) .***

Patients with Parkinson disease can develop language deficits along with dementia. Alzheimer disease causes an isolated dysphasia in rare forms where the pathology affects