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DOI: 10.15225/PNN.2025.14.3.6 JNNN 2025;14(3):136–145 **Review**

Neurorehabilitation of Central Speech Disorders in Patients After Stroke and Traumatic Brain Injury — The State of Knowledge and The Potential of Transcranial Direct Current Stimulation

Neurorehabilitacja ośrodkowych zaburzeń mowy u pacjentów po udarze i urazowym uszkodzeniu mózgu — stan wiedzy i potencjał przezczaszkowej stymulacji prądem stałym

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Abstract

Aphasia is a complex neurological disorder resulting from brain damage, leading to significant deficits in speech, comprehension, reading and writing. It significantly reduces the patients' life quality, contributing to social isolation, depression and reduced independence. Despite advances in speech therapy (SLT), many patients experience limited improvement. The aim of this paper is to analyze the current state of knowledge on the rehabilitation of central speech disorders, with particular emphasis on the use of transcranial direct current stimulation (tDCS) as a supplement to traditional speech therapy neurorehabilitation. A literature review was conducted, including studies published between 2014 and 2024 in the PubMed, Scopus, Web of Science, and Cochrane Library databases. Randomized controlled trials (RCTs), systematic reviews, and meta-analyses assessing the efficacy of tDCS in patients with post-stroke aphasia and other speech disorders were analyzed. The review focused on: Mechanisms of tDCS action in the context of neuroplasticity and language recovery, Therapeutic protocols (intensity, duration, electrode location), Clinical outcomes regarding the improvement of functional communication, speech fluency, and other language parameters. Additionally, data on the safety and adverse effects of tDCS were analyzed, based on retrospective reviews and clinical reports. Research shows that intensive speech therapy leads to improved functional communication and reduced aphasia. tDCS has been shown to support neuroplasticity processes, contributing to faster and more effective recovery of speech function. Combined therapy (tDCS and SLT) proved to be more effective than SLT alone. However, differences in tDCS protocols and individual patient response indicate the need for personalization of treatment. tDCS is a promising, non-invasive method for supporting the rehabilitation of speech disorders. Integration of tDCS with traditional neurorehabilitation methods can significantly improve the communication skills of patients with aphasia. Further studies are necessary to optimize stimulation parameters and develop standard clinical guidelines. **(JNNN 2025;14(3):136–145)**

Key Words: aphasia, brain injury, neurorehabilitation, post-stroke rehabilitation, speech therapy, transcranial direct current stimulation (tDCS)

Streszczenie

Afazja jest złożonym zaburzeniem neurologicznym wynikającym z uszkodzenia mózgu, prowadzącym do znacznych deficytów w zakresie mowy, rozumienia, czytania i pisania. Znacząco obniża jakość życia pacjentów, przyczyniając się do izolacji społecznej, depresji i ograniczonej niezależności. Pomimo postępów w terapii mowy (SLT), wielu pacjentów doświadcza ograniczonej poprawy. Celem niniejszego artykułu jest analiza aktualnego stanu wiedzy na temat rehabilitacji ośrodkowych zaburzeń mowy, ze szczególnym uwzględnieniem zastosowania przezczaszkowej stymulacji prądem stałym (tDCS) jako uzupełnienia tradycyjnej neurorehabilitacji logopedycznej. Przeprowadzono przegląd literatury obejmujący badania opublikowane w latach 2014–2024 w bazach PubMed, Scopus, Web of Science i Cochrane Library. Przeanalizowano randomizowane badania kontrolowane (RCT), przeglądy systematyczne i metaanalizy oceniające skuteczność tDCS u pacjentów z afazją poudarową i innymi zaburzeniami mowy. Przegląd koncentrował się na: mechanizmach działania tDCS w kontekście neuroplastyczności i odzyskiwania funkcji mowy, protokołach terapeutycznych (intensywność, czas trwania, lokalizacja elektrod), wynikach klinicznych dotyczących poprawy komunikacji funkcjonalnej, płynności mowy i innych parametrów językowych. Dodatkowo przeanalizowano dane dotyczące bezpieczeństwa i niepożądanych skutków tDCS, w oparciu o retrospektywne przeglądy i raporty kliniczne. Badania wskazują, iż intensywna terapia mowy prowadzi do poprawy komunikacji funkcjonalnej i zmniejszenia afazji. Wykazano, że tDCS wspiera procesy neuroplastyczności, przyczyniając się do szybszego i skuteczniejszego odzyskiwania funkcji mowy. Terapia łączona (tDCS i SLT) okazała się skuteczniejsza niż sama terapia logopedyczna. Jednak różnice w protokołach tDCS i indywidualnej reakcji pacjentów wskazują na potrzebę personalizacji leczenia. tDCS jest obiecującą, nieinwazyjną metodą wspomagającą rehabilitację zaburzeń mowy. Integracja tDCS z tradycyjnymi metodami neurorehabilitacji może znacząco poprawić umiejętności komunikacyjne pacjentów z afazją. Konieczne są dalsze badania w celu optymalizacji parametrów stymulacji i opracowania standardowych wytycznych klinicznych. **(PNN 2025;14(3): 136–145)**

Słowa kluczowe: afazja, uszkodzenie mózgu, neurorehabilitacja, rehabilitacja po udarze, terapia mowy, przezczaszkowa stymulacja prądem stałym (tDCS)

Introduction

Aphasia is a serious set of acquired communication disorders resulting from damage to functional brain areas located in the frontal and temporal lobes, as well as to the language networks associated with them [1]. Aphasia is a disorder resulting from damage to brain centers, usually located in the left hemisphere. Aphasic disorders in the ability to speak, understand, repeat, write and read vary greatly depending on the specific clinical situation of the patient and depend on the type of aphasia.

Aphasia may also coexist with abnormal speech motor programming in terms of purpose and motor precision called apraxia of speech or verbal apraxia, a disorder characterized by an impaired ability to coordinate the sequential articulatory movements necessary to produce sounds [2].

Impaired functional communication in patients with aphasia can lead to impaired functioning, poor recovery, depression, increased social isolation, as well as a destructive impact on family functioning, impaired partner relationships, and the inability to fulfill social roles [3,4]. It has also been proven that emotional distress, increased aphasia, communication limitations, limited activity levels, other medical problems, and social factors affect the quality of life.

Therapy for Central Speech and Communication Disorders

Priorities and Principles of Speech Therapy Rehabilitation in Aphasia

Due to its high prevalence and increased risk of central speech disorders, neurorehabilitation of aphasia is listed as one of the top 10 research priorities for functioning after brain injury [5]. Seven of the top ten priorities concern stroke-related disabilities, such as cognitive problems, aphasia, mobility problems, visual impairment, fatigue, and sensory impairment.

Currently, the main therapeutic approach to aphasia is a traditional speech therapy program focused on supporting communication skills and speech reeducation, which can to some extent improve the ability to communicate, especially taking into account the aspect of communicating with the environment, which directly affects the improvement of the quality of life. It is worth emphasizing, however, that the degree of regaining communication skills is still limited and often does not bring the expected results [6].

Mechanisms of Speech Function Recovery

Neurological rehabilitation of patients with aphasic speech disorders should be mainly focused on the communication aspect, because the priority is training

the patient's ability to communicate with people from the closest environment, thus giving a sense of confidence. The effectiveness of aphasia neurorehabilitation depends on the extent, nature and depth of CNS damage, but also on the individual characteristics of the patient such as age, psychophysical condition, time and intensity of speech re-education, appropriate selection of methods and forms of therapy, as well as support from the family and environment.

The return of impaired speech functions occurs as a result of the interaction of various compensatory mechanisms occurring through analogous fields of the other hemisphere or through undamaged collateral regions of the same hemisphere of the brain [7–9]. It is important to emphasize that although there is no standard treatment, the main goal is for the patient to regain as much independence as possible. Patients are assessed by speech therapists to determine their strengths and weaknesses in order to individually adjust the treatment plan. However, it has been shown that patients achieve better improvement with short, intensive therapy sessions compared to longer but less intensive sessions.

Factors Influencing the Effectiveness of Therapy

Effective neurological rehabilitation in the case of diagnosed aphasic speech disorders is necessary to minimize the negative impact of these deficits on the daily functioning of patients. Early diagnosis based on screening tests and neuroimaging and neurophysiological diagnostic methods is crucial, which is the basis for implementing comprehensive treatment based on current knowledge of neuroanatomy and pathophysiology of damage to the brain centers that control speech. It should be strongly emphasized that some patients with speech disorders, especially those in a more serious clinical condition initially caused by a more extensive picture of brain damage, experience a number of harmful neglect as a result of improper diagnostics and/or ineffectively conducted therapy. It is necessary to search for increasingly effective neurorehabilitation methods, which are a tool for patients to regain lost functions.

Neurorehabilitation in Aphasia — a Review of Studies (Table 1)

A recent Cochrane review [10] found that high-intensity *speech and language therapy* (SLT) led to reduced aphasia severity and greater improvement in functional communication compared with lower-intensity SLT. Despite these results, there remains uncertainty about the overall intensity of speech and language therapy required to achieve significant improvement in people with aphasia. Evidence was provided for the beneficial effects of SLT for patients with aphasia after stroke, as assessed by functional communication, reading, speech comprehension, expressive speech, and writing. Although there was general consistency in the results across the studies included in these analyses, some of the significant results were dependent on data from a single study with limited information on the nature of the SLT intervention and the methodological quality of that study. Therefore, some caution should be exercised in interpreting these results. It should also be noted that the SLT used in these studies may be considered a high-intensity therapy for variable periods of time. There was also some indication from a smaller number of studies of the benefits of intensive approaches to SLT in improving functional communication and reducing the degree of aphasia. The intensity of intervention varied, as did the duration of therapy, but high-intensity SLT approaches may not have been suitable for all patients. Significantly more patients in the intensive speech therapy groups dropped out of these studies. Similarly, one small study found that social support may be beneficial for some aspects of patients' language skills, but the results were confounded by significantly more participants dropping out of the social support intervention. The review did not provide clear evidence to establish the effectiveness of one treatment approach to SLT over another. There was little evidence of differences between group-based and face-to-face SLT interventions; or computer-based and face-to-face SLT by a therapist. Similarly, there was little evidence of a difference in the effectiveness of SLT facilitated by a trained volunteer compared with SLT delivered by a specialist therapist. This is not surprising, as the volunteers in these studies received specialized training, had access to treatment materials, and in many cases provided treatment interventions designed and supervised by a professional therapist [10].

German research published in the prestigious journal Lancet [11] conducted on a representative sample of 158 patients with aphasia shows that 3 weeks of intensive SLT significantly improves verbal communication in individuals aged 70 years and younger with chronic aphasia following stroke, providing an effective, evidence-based approach to treatment in this group of patients. The authors also conclude that future studies should investigate the minimal intensity of SLT required to achieve measurable therapeutic effects, as well as determine whether the effects of SLT accumulate over repeated periods of speech therapy intervention.

Results of the latest 2021 meta-analysis conducted by the REhabilitation and recovery of peopLE with Aphasia after StrokE collaboration (RELEASE) published in the prestigious Stoke [12] add to the body of knowledge on speech recovery after stroke. They identify the domains that improve the most and describe the degree of

improvement and the time windows in which the greatest improvement occurs. The greatest improvement was observed for patients presenting within 1 month of stroke in all language domains. The relative and absolute improvement in mean scores from baseline decreased with increasing time since stroke but still exceeded the group-level rates of significant improvement in overall speech ability. In cases where early intervention is not feasible, for example due to comorbidity or inability to engage in rehabilitation, it should be considered as soon as possible, as significant improvement was still observed after the acute period.

In summary, various therapeutic methods are used in the treatment of aphasia, and there is evidence of the effectiveness of SLT. It is recommended that all stroke patients with communication disorders undergo comprehensive SLT, individually tailored to their needs resulting from the level of damage [13]. Although comprehensive SLT remains the mainstay of aphasia rehabilitation, the neuromodulation technique tDCS is a promising adjunctive therapy for the improvement of aphasia [14].

Another systematic review with a meta-analysis of randomized controlled trials [15] of tDCS for the improvement of aphasia after stroke, including 21 studies with 421 participants, showed relatively low to moderate quality of evidence from the studies to date. The detailed parameters assessed and the results obtained after the use of speech therapy in combination with tDCS were as follows: (1) functional communication — 3 studies (N=112) showed no effect on functional communication after the intervention (low quality of evidence); and 2 studies (N=80) showed no effect on functional communication after the intervention (very low quality of evidence); (2) language disorders: noun naming accuracy — 11 studies (N=298) showed evidence of a positive effect on noun naming accuracy after the intervention (moderate quality of evidence); and 2 studies (N=80) showed a positive effect on noun naming accuracy after the intervention (low quality of evidence); (3) language impairment: verb naming accuracy — 3 studies (N=21) found no effect on verb naming accuracy after the intervention (very low quality of evidence); however, none of the studies assessed verb naming accuracy after the end of therapy; (4) cognitive impairment — no studies were found to assess the effect of tDCS on cognition in aphasia after stroke; (5) dropout and adverse events — no evidence was found to show a difference in the number of dropouts between the intervention and control groups (low quality of evidence), and no serious adverse events were reported.

Transcranial Electrical Stimulation of The Brain

Biophysical and Neurophysiological Foundations

The tDCS is a noninvasive neuromodulatory procedure for brain stimulation that was first introduced in animal and human experiments in the 1950s. It was only about 20 years ago that tDCS was included in the standard range of neurophysiological methods used in neurological, but also psychological, psychiatric, motor, behavioral, and clinical conditions. Unlike other noninvasive brain stimulation tools, such as transcranial magnetic stimulation (rTMS), it does not directly induce brain activity, but rather changes spontaneous brain activity and excitability through subthreshold modulation of neuronal cell membranes [16].

tDCS stimulation is a noninvasive technique that involves the delivery of a low-amplitude current of values usually not exceeding 2 mA, where the patient's exposure time is not longer than 30 minutes, using rubber electrodes with a positive charge (anode) and a negative charge (cathode) covered with a sponge soaked in physiological saline. At least one of the electrodes is placed on the scalp, and electronic current pulses pass through the skull to the brain to facilitate (activate) or inhibit (inhibit) spontaneous neuronal activity of specific brain areas located below and between the electrodes [17]. In a typical neurophysiological or clinical application of tDCS, two electrodes are placed on the scalp to target a specific brain region. One electrode is placed over the target area. The position of the second electrode is also important and is usually determined empirically or precisely described by the manufacturer of the equipment. The effect of the selected electrode mounting method depends on the spatial distribution of the electric field induced in the gray and white matter of the brain, as well as on many other factors, such as the orientation of the electric field relative to the neurons [18]. Typically, tDCS stimulation includes three stages: (1) fade-in, where the electric field intensity gradually increases, short-stimulation, which is the stage of the actual excitation, and fade-out, where the electric field intensity decreases and stimulation is gradually stopped [19].

Based on preclinical and clinical studies by observing motor evoked potentials, it has been proven that treatments using positively charged electrodes — anodal tDCS (A-tDCS) in the area of the primary motor cortex (M1) cause increased neuronal excitability, while the use of negatively charged electrodes — cathodal tDCS (C-tDCS) results in decreased neuronal excitability [20,21]. However, recently, opposing results have been presented indicating possible excitatory properties of C-tDCS and inhibitory effects of A-tDCS, especially in individuals who do not respond to tDCS [22,23]. Full

stimulation of a neuron is possible only when its cell membrane undergoes a sufficiently large depolarization and reaches the so-called threshold level, i.e. a value of about –50 mV at resting potential values of about –70 mV. Then, the ion channels of the protein-lipid membrane open, which leads to its depolarization reaching a value of +30 mV, which results in the formation of an action potential. If the active electrode is the anode (A-tDCS), the negative charge that forms under it leads to the depolarization of the neuronal cell membrane due to the lack of positive ions, the final effect of which is an increase in the neuronal activity of the cerebral cortex. In turn, if the active electrode is the cathode (C-tDCS), then the positive charge is concentrated under it, which leads to hyperpolarization of the neuronal cell membrane, the consequence of which is a decrease in cortical excitation [24].

In addition, the electric field generated by tDCS during treatments is influenced by several factors, such as the geometry of the sulci or gyri under the electrodes, the central layer of the cerebrospinal fluid and the thickness of the skull and the depth of the gyri, which is associated with individual variability [18,25]. In most studies, two surface-conducting rubber electrodes of size between 25 and 35 cm² are used. The current intensity using these electrodes ranges between 1 and 2 mA, and the treatment time is usually between 10 and 20 minutes. The mechanisms underlying the changes in cortical excitability induced by tDCS differ between the effects observed during stimulation (early effects) and those induced after its cessation (after effects), despite similar neurophysiological effects resulting in the phenomenon of synaptic plasticity due to the generation of long-lasting potentialization [26].

Micropolarization of tDCS can be applied to the prefrontal, visual, somatosensory and motor cortex, influencing the impaired executive, cognitive and perceptual functions, which has an effective effect on the process of motor and speech therapy rehabilitation of patients with brain damage. Micropolarization of the brain has a positive effect on the modulation of the impaired functions of neural circuits, and paves the way for a cascade of self-repair processes based on the activation of the compensatory neuroplasticity phenomenon [27].

Recent studies have highlighted the role of stimulation dosage in tDCS-induced neuroplasticity in the motor cortex of healthy older adults and provide important information for optimizing tDCS protocols in the primary motor cortex. The presented findings may provide a basis for developing optimal stimulation protocols aimed at restoring neuroplasticity in different cortical areas and inducing long-term, functionally relevant plasticity in normal aging and pathological conditions, which would, however, require systematic studies using the tDCS titration technique in appropriate target areas [28].

The acute effect of tDCS is thought to be due to a shift in the membrane potential within neurons [29]. The mechanisms underlying the after-effects of tDCS are still the subject of much research. For example, some studies have shown that the long-lasting effects of tDCS result from changes in synaptic strength through N-methyl-D-aspartate (NMDA) receptors in a polarity-dependent manner [30] or by altering the activity of gamma-aminobutyric acid (GABA) receptors. Other studies suggest that extrasynaptic mechanisms also influence changes in excitability in the brain. The after-effects of tDCS have been shown to have an extrasynaptic mechanism of action, based on changes in the functioning of neuronal membranes. These changes, in addition to reflecting local modifications in ion concentrations, may result from changes in transmembrane proteins and from changes in hydrogen ion levels associated with

Table 1. Summary of selected studies on the effectiveness of tDCS in patients with post-stroke aphasia

Author (year)	Research population	tDCS Protocols	Therapy time	Results	Conclusions
Fridriksson et al. (2018)	Aphasia post-stroke, RCT (N=74)	Anodal tDCS, 2 mA, 20 min	15 sessions (3 weeks)	Increased fluency of speech, improved comprehension	tDCS Therapy as an Effective Support for SLT
Breitenstein et al. (2017)	Aphasia post-stroke, RCT (N=58)	Bipolar tDCS, 1.5 mA, 30 min	21 days	Improving verbal communication, reducing the severity of aphasia	Effectiveness of tDCS in patients 70+
Elsner et al. (2020)	Meta-analysis of RCTs	Different tDCS protocols (1–2 mA)	10–20 sessions	Moderate improvement in naming and spelling	Need for further research on parameter optimization
Russo et al. (2017)	Review literature	Cathodal tDCS, 2 mA	10 sessions	No effect on functional communication	Recommendation for more intensive treatment sessions

electrolysis induced by exposure to a constant electric field [31]. The latest hypothesis is that the effects of tDCS may be induced more by changes occurring at the level of glial cells than neurons, because they actively participate in the functioning of the brain and perform many important functions in it [32]. However, in all cases, these local changes are induced by the applied electric field, directly or indirectly in different cortical areas.

Methodology of Treatments and Treatment Procedures (Table 2)

The standard tDCS procedure uses a target and reference electrode. First, the desired electrode placement sites are determined and are closely related to the patient's medical indications. Before attaching the electrodes to the scalp, the operator should ensure that the patient does not have damaged or broken skin. If saline is used as the conductive substance, the electrodes can be placed in sponge pads that are soaked enough to be sufficiently moist but not dripping. However, increasingly, conductive paste or EEG gel are used to attach the electrodes to the scalp, which can control the current distribution more effectively than saline. The patient's hair should be parted to ensure good contact between the scalp and the electrode. The saline should not run down the scalp or spread on the hair to limit the electric field to the target treatment area. The electrodes are then attached to the stimulator using wires connected to the appropriate anode/cathode ports. Once the electrode is placed over the target area, it should be secured with a cap, rubber bands, or flexible tubular mesh. Once the electrodes are attached, the treatment parameters such as tDCS stimulation duration, current intensity, and pulse rise and fall times should be programmed. Some stimulators allow for pre-programming of stimulation parameters, while others require manual entry of data before each session.

It is important to continuously monitor the participant during tDCS stimulation to ensure complete comfort during the procedure. It is also important to check the impedance levels displayed on the stimulator to ensure that stimulation has not failed. Reliable and consistent use of tDCS requires adequate contact between the electrodes and the scalp to maintain conduction in the circuit. High impedance levels are an indicator of poor conduction and may be the result of poor electrode positioning or insufficient coupling agent. Because impedance levels indicate whether the current can be maintained at a constant level, it is important to monitor these levels displayed on the stimulator throughout the experiment/treatment [33].

Several methods can be used to localize the electrode; however, the most common method is the 10:20 system, in which the EEG, tDCS and TMS measuring electrodes are placed on the surface of the head according to a strictly defined pattern. When using this method, the participant's head is first measured to precisely localize the areas of interest corresponding to individual brain regions. This is usually done by measuring in the sagittal plane, i.e. from the depression at the top of the nose (Latin: *nasion*) to the convexity lying in the midline of the base of the skull (Latin: *inion*) and in the coronal plane, i.e. from the left preauricular part (Latin: *preaurical*) to the right, passing through the top of the head (Latin: *vertex*) [34].

The measurements can then be used in conjunction with the 10:20 system to locate regions of interest. Target regions can be marked with a washable marker. When locating electrodes to measure electrical activity in the brain, their topography is described in three planes: sagittal, coronal, and horizontal. The 10:20 system names contain odd numbers for electrodes placed on the left side of the head and even numbers for electrodes placed on the right side. Alternatively, neuronavigation software can be used, which can be more accurate than the 10:20 system. However, this method is dependent on whether the participant undergoes MRI. Access to previous MRI scans may be possible, but if this is not possible, scanning each participant before undergoing tDCS can be expensive. Physiology-based positioning can also be used; for example, if the region of interest is the motor cortex, motor evoked potentials can be stimulated first to identify this region [21]. However, physiological electrode placement is currently limited to a few core cortical regions, which means that not all electrode locations may be affected by this measurement [16].

The region of interest is stimulated with a target electrode, the location of which depends on the application. The treatments should recruit neurons in the target region so that functional changes associated with stimulation can be observed. Instead, dual-plate montages (also known as "dual" stimulation) can be used, in which the position of both target electrodes is important for regulating down-regulation of one region (cathodal current) and up-regulation of the region in the opposite hemisphere (anodal current). For example, if the goal is to improve motor function, dual stimulation can be focused on both motor cortices in the precentral gyrus [35]. The target region should be located on the cortical surface because scalp electrodes do not penetrate deep brain regions. Modeling studies have shown that current distribution and therefore current density can vary between individuals, even when the electrode montage is consistent, due to anatomical features such as skull thickness and composition [25]. Current direction may also be influenced by pathological changes that

may be common among study participants and/or patients [36]. The use of neuronavigation software allows the investigator and/or therapist to more precisely place electrodes over a specific cortical location while taking into account anatomical differences between patients.

The location of the reference electrode should primarily take into account factors influencing the effect of its location on the purpose of the procedure, the direction of current flow, patient comfort and safety. Some therapists use a montage in which two reference electrodes are placed on the scalp (ensuring the same polarity), and one reference electrode is used (ensuring a different polarity), which gives a total of three electrodes, not two. To ensure proper stimulation in which most of the current reaches the target area, the reference electrode is usually placed on the opposite side to the target electrode [37].

In some cases, the electrodes are placed much closer together, but this should be avoided because it can lead to the current flowing through the cerebrospinal fluid between the electrodes, without stimulating the cerebral cortex. This is because the cerebrospinal fluid has a higher electrical conductivity than the brain tissue [38]. On the other hand, the current can dissipate across the scalp, meaning that a reduced concentration reaches the brain region. Recent studies have shown that only 10% of the current penetrates the stimulated area of the cerebral cortex, with the remaining 90% dissipating during propagation through the scalp, bone tissue, gray matter, and white matter. It has been proposed that if the distance between the electrodes is 5 cm or less, the current will be very susceptible to the shunting effect [39].

Table 2. tDCS treatment protocols for aphasia therapy — a review of meta-analyses

Parameter	Suggested settings	Most common settings
Current	1–2 mA	1.5 mA
Time duration session	10–30 minutes	20 minutes
Number session therapeutic	5–30	15
Location Electrodes	Broca's area, left hemisphere, field M1	Broca's area, F3
Type stimulation	Anodal, cathodal, bipolar	Anodal
Effects side effects	Minimal and temporary: skin redness, tingling	

In the case of impaired speech expression, therapy focuses on improving speech articulation using various types of auxiliary equipment (microphone, headphones, tablets, laptops, speech therapy labiograms). In the case of patients with speech impression disorders, everyday objects, photos, equipment, objects, as well as sets of ready-made exercises published by Komlogo are also used. WiR and Harmonia dedicated speech and cognitive therapy for adults with CNS damage based on a wide range of speech therapy, pedagogical accessories and diagnostic tools. An auditory processing method is also used, which involves the patient listening, processing and articulating syllables and words spoken by a lecturer, listened to on headphones.

Scientific Evidence and Practical Recommendations

Evidence suggests that tDCS can effectively modulate cortical excitability, suggesting its potential therapeutic value in treating a variety of conditions, including cognitive and motor impairments following stroke, but also neuropathic pain, migraine headaches, and epileptic seizures [41]. Meta-analysis data suggest that tDCS improves walking ability, with the exception of walking speed and endurance in stroke patients. Both anodal and bihemispheric tDCS have a positive effect on promoting gait retraining skills, although there are some difficulties in improving balance using tDCS [42]. Micropolarization tDCS appears to be an effective adjunct to conventional rehabilitation techniques. When applied in the acute phase of stroke, functional recovery is not only accelerated but also improved, and the results are maintained for up to 1 year after stroke [43].

Evidence-based recommendations from an international expert panel indicate that significant research efforts have been made to establish the clinical potential of tDCS in humans. Data from numerous studies conducted by global teams have repeatedly shown that tDCS can provide clinical benefits in many neurological conditions, such as post-stroke motor disorders, aphasic speech disorders, cerebellar ataxia, Alzheimer's and Parkinson's disease, chronic pain, and psychiatric disorders such as schizophrenia and major depression. Furthermore, the utility of tDCS in scientific research has proven valuable in elucidating the function of neuronal circuits in the brain by providing a tool capable of safely modulating neurophysiological functions in humans [44,45].

In the case of conducting therapeutic sessions, but also when performing tDCS procedures during scientific research, it is very important to observe, monitor and report any adverse effects or side effects, as well as any non-standard reactions of patients. It is worth emphasizing that there is no data indicating the occurrence of serious adverse effects when using tDCS with a standard intensity of 1–2 mA [46]. Nevertheless, there may be mild, transient side effects, such as headache, itching and tingling

sensation of the scalp in the areas subjected to stimulation, redness of the scalp under the electrode, but also moderate fatigue, difficulty concentrating, short-term mood changes and nausea [47,48]. It has been documented that these effects are spontaneously reported by less than 20% of the studied people. However, it is worth adding that symptoms such as moderate fatigue and weariness may be related to participation in the research project itself, and not to tDCS procedures. The most commonly reported side effect is a skin sensation, which tends to subside once the current waveform stabilizes [49]. Side effects can also be reduced by using a moderate NaCl solution on the fixation bag, using more gel to ensure better conductivity, reducing the current intensity, and using smaller electrode sizes [50,51].

Moreover, it has been proven, based on the example of patients with neurological disorders, that most patients undergoing regular tDCS sessions did not report any serious adverse effects. Based on the data obtained from the MEDLINE/PubMed database review in the years 1998–2015 describing the use of tDCS in stroke patients, only 11.62% of published papers reported the occurrence of adverse effects of the tDCS procedure. The most common of them was itching of the scalp (70%), followed by a burning sensation of the scalp (40%), headache (40%), tingling (30%), drowsiness (20%), difficulty in concentration, mild fatigue, skin redness and dizziness (10%). It is worth adding that significant differences were found between studies "reporting" and "not reporting" adverse effects in terms of tDCS parameters (intensity, current density, stimulation duration and number of sessions) [52].

It should be emphasized here that the basic procedural parameters of tDCS in the cited study [52] were usually used, with a current intensity between 1–2 mA, with a median of 1 mA (52.95% of articles used ≤1 mA); the median current density, defined as the mean stimulation current divided by the stimulating electrode area, was 0.04 mA/cm² (50.63% of articles used ≤0.04 mA/cm²). The median stimulation duration was 20 min (80% of articles used a shorter or equal duration); it was used for a median of 5 therapeutic sessions in 56.47% of articles [52]. Current evidence indicates that micropolarization tDCS performed in accordance with current safety guidelines is well tolerated by patients with brain injuries; Adverse events have been reported in rare cases, but they were mostly mild and transient, which was also observed in healthy control subjects [52].

An important consideration in the design and conduct of clinical trials using DC micropolarization is the use of sham tDCS interventions within placebo-controlled interventions to ensure blinding. In order to determine the neurophysiological, cognitive, or clinical effects of tDCS, most studies compare the effects of active tDCS with those of sham tDCS. In most cases, sham tDCS involves the delivery of active stimulation for a few seconds to mimic the sensations observed during active tDCS, and participants remain blinded to the intervention. However, to date, sham tDCS-controlled studies have yielded inconsistent results, which may be due in part to inconsistencies in the use of sham tDCS. In fact, tDCS studies employ a variety of sham stimulation protocols that may have different biological effects beyond the intended transient experiences [53,54]. Most clinical and non-clinical studies using tDCS include in their experimental design some form of placebo treatment as a control group, e.g., a "*fade-in, short-simulation, fade-out*" sham protocol. This type of sham protocol is considered to be indistinguishable from longer, active stimulation periods (e.g. 10–30 min) and is assumed to blind participants to the applied stimulus [55].

Summary

Neurorehabilitation of aphasia and other central speech disorders is a key element in improving the quality of life of patients after brain damage. These disorders not only limit the ability to communicate, but also lead to social isolation and deterioration of the mental health of patients. The literature review indicates that intensive speech therapy can bring significant benefits, but the effectiveness of rehabilitation still depends on many factors, such as the time of starting therapy, its intensity, and social and family support. tDCS is a promising method supporting speech therapy. The mechanism of tDCS is based on the modulation of neuronal excitability, which promotes neuroplasticity processes, allowing the brain to adapt to new conditions.

Numerous studies and meta-analyses confirm that tDCS combined with speech therapy rehabilitation can accelerate the recovery of language and motor functions in patients after stroke. Despite promising results, the tDCS technique requires further research on optimal stimulation parameters, duration of therapy and its effectiveness in the long term. In particular, it is necessary to establish minimum and maximum doses and protocols adapted to the individual needs of patients. Integration of modern technologies with classical speech therapy methods can contribute to improving treatment outcomes and reducing the burden associated with communication disorders.

References

[1] Worrall L., Foster A. Does intensity matter in aphasia rehabilitation? *Lancet*. 2017;389(10078):1494–1495.

[2] Saur D., Hartwigsen G. Neurobiology of language recovery after stroke: lessons from neuroimaging studies. *Arch Phys Med Rehabil*. 2012;93(1 Suppl):S15–25.
[3] Cichon N., Wlodarczyk L., Saluk-Bijak J. et al. Novel Advances to Post-Stroke Aphasia Pharmacology and Rehabilitation. *J Clin Med*. 2021;10(17):3778.
[4] Rasmus A., Orłowska E. Marriage and Post-stroke Aphasia: The Long-Time Effects of Group Therapy of Fluent and Non-fluent Aphasic Patients and Their Spouses. *Front Psychol*. 2020;11:1574.
[5] Pollock A., St George B., Fenton M., Firkins L. Top ten research priorities relating to life after stroke. *Lancet Neurol*. 2012;11(3):209.
[6] Fridriksson J., Rorden C., Elm J., Sen S., George M.S., Bonilha L. Transcranial Direct Current Stimulation vs Sham Stimulation to Treat Aphasia After Stroke: A Randomized Clinical Trial. *JAMA Neurol*. 2018;75(12): 1470–1476.
[7] Lewicka T., Stompel D., Nowakowska-Kempna I. Zaburzenia językowe w chorobach neurodegeneracyjnych — aspekty diagnostyczne i terapeutyczne. *Logopedia Silesiana*. 2014;3:76–94.
[8] Maruszewski M. *Chory z afazją i jego usprawnianie*. Nasza Księgarnia, Warszawa 1974.
[9] Szumska J. *Metody rehabilitacji afazji*. PZWL, Warszawa 1980.
[10] Brady M.C., Kelly H., Godwin J., Enderby P. Speech and language therapy for aphasia following stroke. *Cochrane Database Syst Rev*. 2012;5:CD000425.
[11] Breitenstein C., Grewe T., Flöel A. et al. Intensive speech and language therapy in patients with chronic aphasia after stroke: a randomised, open-label, blinded-endpoint, controlled trial in a health-care setting. *Lancet*. 2017; 389(10078):1528–1538.
[12] REhabilitation and recovery of peopLE with Aphasia after StrokE (RELEASE) Collaborators. Predictors of Poststroke Aphasia Recovery: A Systematic Review-Informed Individual Participant Data Meta-Analysis. *Stroke*. 2021;52(5):1778–1787.
[13] Küçükdeveci A.A., Stibrant Sunnerhagen K., Golyk V. et al. Evidence-based position paper on Physical and Rehabilitation Medicine professional practice for persons with stroke. The European PRM position (UEMS PRM Section). *Eur J Phys Rehabil Med*. 2018;54(6):957–970.
[14] Tippett D.C. Update in Aphasia Research. *Curr Neurol Neurosci Rep*. 2015;15(8):49.
[15] Elsner B., Kugler J., Mehrholz J. Transcranial direct current stimulation (tDCS) for improving aphasia after stroke: a systematic review with network meta-analysis of randomized controlled trials. *J Neuroeng Rehabil*. 2020;17(1):88.
[16] Woods A.J., Antal A., Bikson M. et al. A technical guide to tDCS, and related non-invasive brain stimulation tools. *Clin Neurophysiol*. 2016;127(2):1031–1048.
[17] Yokoi Y., Narita Z., Sumiyoshi T. Transcranial Direct Current Stimulation in Depression and Psychosis: A Systematic Review. *Clin EEG Neurosci*. 2018;49(2):93–102.
[18] Miranda P.C., Mekonnen A., Salvador R., Ruffini G. The electric field in the cortex during transcranial current stimulation. *Neuroimage*. 2013;70:48–58.
[19] Ambrus G.G., Al-Moyed H., Chaieb L., Sarp L., Antal A., Paulus W. The fade-in--short stimulation--fade out approach to sham tDCS--reliable at 1 mA for naïve and experienced subjects, but not investigators. *Brain Stimul*. 2012;5(4):499–504.
[20] Pellicciari M.C., Brignani D., Miniussi C. Excitability modulation of the motor system induced by transcranial direct current stimulation: a multimodal approach. *Neuroimage*. 2013;83:569–580.
[21] Nitsche M.A., Paulus W. Excitability changes induced in the human motor cortex by weak transcranial direct current stimulation. *J Physiol*. 2000;527(3):633–639.
[22] López-Alonso V., Cheeran B., Río-Rodríguez D., Fernández-Del-Olmo M. Inter-individual variability in response to non-invasive brain stimulation paradigms. *Brain Stimul*. 2014;7(3):372–380.
[23] Wiethoff S., Hamada M., Rothwell J.C. Variability in response to transcranial direct current stimulation of the motor cortex. *Brain Stimul*. 2014;7(3):468–475.
[24] Pirulli C., Fertonani A., Miniussi C. Is neural hyperpolarization by cathodal stimulation always detrimental at the behavioral level? *Front Behav Neurosci*. 2014;8:226.
[25] Opitz A., Paulus W., Will S., Antunes A., Thielscher A. Determinants of the electric field during transcranial direct current stimulation. *Neuroimage*. 2015;109:140 –150.
[26] Stagg C.J., Nitsche M.A. Physiological basis of transcranial direct current stimulation. *Neuroscientist*. 2011;17(1):37 –53.
[27] Frase L., Mertens L., Krahl A. Transcranial direct current stimulation induces long-term potentiation-like plasticity in the human visual cortex. *Transl Psychiatry*. 2021;11(1): 17.
[28] Farnad L., Ghasemian-Shirvan E., Mosayebi-Samani M., Kuo M.F., Nitsche M.A. Exploring and optimizing the neuroplastic effects of anodal transcranial direct current stimulation over the primary motor cortex of older humans. *Brain Stimul*. 2021;14(3):622–634.
[29] Liebetanz D., Nitsche M.A., Tergau F., Paulus W. Pharmacological approach to the mechanisms of transcranial DC-stimulation-induced after-effects of human motor cortex excitability. *Brain*. 2002;125 (Pt 10):2238–2247.
[30] Nitsche M.A., Fricke K., Henschke U. et al. Pharmacological modulation of cortical excitability shifts induced by transcranial direct current stimulation in humans. *J Physiol*. 2003;553(Pt 1):293–301.
[31] Ardolino G., Bossi B., Barbieri S., Priori A. Non-synaptic mechanisms underlie the after-effects of cathodal transcutaneous direct current stimulation of the human brain. *J Physiol*. 2005;568(Pt 2):653–663.
[32] Ruohonen J., Karhu J. tDCS possibly stimulates glial cells. *Clin Neurophysiol*. 2012;123(10):2006–2009.
[33] Thair H., Holloway A.L., Newport R., Smith A.D. Transcranial Direct Current Stimulation (tDCS): A Beginner's Guide for Design and Implementation. *Front Neurosci*. 2017;11:641.
[34] Klem G.H., Lüders H.O., Jasper H.H., Elger C. The ten-twenty electrode system of the International Federation.

The International Federation of Clinical Neurophysiology. *Electroencephalogr Clin Neurophysiol Suppl*. 1999;52:3–6.
[35] Lindenberg R., Renga V., Zhu L.L., Nair D., Schlaug G. Bihemispheric brain stimulation facilitates motor recovery in chronic stroke patients. *Neurology*. 2010;75(24):2176–2184.
[36] Datta A., Baker J.M., Bikson M., Fridriksson J. Individualized model predicts brain current flow during transcranial direct-current stimulation treatment in responsive stroke patient. *Brain Stimul*. 2011;4(3):169–174.
[37] Nasseri P., Nitsche M.A., Ekhtiari H. A framework for categorizing electrode montages in transcranial direct current stimulation. *Front Hum Neurosci*. 2015;9:54.
[38] Moliadze V., Antal A., Paulus W. Electrode-distance dependent after-effects of transcranial direct and random noise stimulation with extracephalic reference electrodes. *Clin Neurophysiol*. 2010;121(12):2165–2171.
[39] Rush S., Driscoll D.A. Current distribution in the brain from surface electrodes. *Anesth Analg*. 1968;47(6):717–723.
[40] Miyagishi Y., Ikeda T., Takahashi T. et al. Gamma-band auditory steady-state response after frontal tDCS: A double-blind, randomized, crossover study. *PLoS One*. 2018;13(2):e0193422.
[41] Budzisz J., Szczepanowski R., Kruk P. Przezczaszkowa stymulacja stałoprądowa tDCS w badaniach naukowych mózgu człowieka. *Prz Elektrotech*. 2017;4:42–45.
[42] Tien H.H., Liu W.Y., Chen Y.L., Wu Y.C., Lien H.Y. Transcranial direct current stimulation for improving ambulation after stroke: a systematic review and meta-analysis. *Int J Rehabil Res*. 2020;43(4):299–309.
[43] Bornheim S., Croisier J.L., Maquet P., Kaux J.F. Transcranial direct current stimulation associated with physical-therapy in acute stroke patients — A randomized, triple blind, sham-controlled study. *Brain Stimul*. 2020;13(2):329–336.
[44] Fregni F., Nitsche M.A., Loo C.K. et al. Regulatory Considerations for the Clinical and Research Use of Transcranial Direct Current Stimulation (tDCS): review and recommendations from an expert panel. *Clin Res Regul Aff*. 2015;32(1):22–35.
[45] Orrù G., Cesari V., Conversano C., Gemignani A. The clinical application of transcranial direct current stimulation in patients with cerebellar ataxia: a systematic review. *Int J Neurosci*. 2021;131(7):681–688.
[46] Arul-Anandam A.P., Loo C., Sachdev P. Transcranial direct current stimulation — what is the evidence for its efficacy and safety? *F1000 Med Rep*. 2009;1:58.
[47] Poreisz C., Boros K., Antal A., Paulus W. Safety aspects of transcranial direct current stimulation concerning healthy subjects and patients. *Brain Res Bull*. 2007;72(4–6):208–214.
[48] Brunoni A.R., Amadera J., Berbel B., Volz M.S., Rizzerio B.G., Fregni F. A systematic review on reporting and assessment of adverse effects associated with transcranial direct current stimulation. *Int J Neuropsychopharmacol*. 2011;14(8):1133–1145.
[49] Nitsche M.A., Cohen L.G., Wassermann E.M. et al. Transcranial direct current stimulation: State of the art 2008. *Brain Stimul*. 2008;1(3):206–223.
[50] DaSilva A.F., Volz M.S., Bikson M., Fregni F. Electrode positioning and montage in transcranial direct current stimulation. *J Vis Exp*. 2011;(51):2744.
[51] Turi Z., Ambrus G.G., Ho K.A., Sengupta T., Paulus W., Antal A. When size matters: large electrodes induce greater stimulation-related cutaneous discomfort than smaller electrodes at equivalent current density. *Brain Stimul*. 2014;7(3):460–467.
[52] Russo C., Souza Carneiro M.I., Bolognini N., Fregni F. Safety Review of Transcranial Direct Current Stimulation in Stroke. *Neuromodulation*. 2017;20(3):215–222.
[53] Fonteneau C., Mondino M., Arns M. et al. Sham tDCS: A hidden source of variability? Reflections for further blinded, controlled trials. *Brain Stimul*. 2019;12(3):668–673.
[54] Palm U., Reisinger E., Keeser D. et al. Evaluation of sham transcranial direct current stimulation for randomized, placebo-controlled clinical trials. *Brain Stimul*. 2013;6(4):690–695.
[55] Turner C., Jackson C., Learmonth G. Is the "end-of-study guess" a valid measure of sham blinding during transcranial direct current stimulation? *Eur J Neurosci*. 2021;53(5):1592–1604.

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