CASE STUDY

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EFFECT OF INDIVIDUALLY-TAILORED TDCS AND SYMBOLIC ART THERAPY FOR CHRONIC ASSOCIATIVE PROSOPAGNOSIA AFTER INFECTION BY SARS-COV-2, NEUROCOVID-19 AND ISCHEMIC STROKE

Maria Pąchalska[1[A,B,C,D,E,F]], Jolanta Góral-Półrola[2[A,B,C,D,E,F]], Izabela Chojnowska-Ćwiąkała[3[A,B,D,E,G]]

[1] Chair of Neuropsychology, The Andrzej Frycz-Modrzewski Cracow University, Kraków, Poland
[2] Old Polish University, Kielce, Poland
[3] Collegium Medicum, Jan Kochanowski University, Kielce, Poland

SUMMARY

Background: The rehabilitation of patients with chronic prosopagnosia that occurs following a stroke is a challenge for modern medicine. Dysfunction to the facial processing areas is permanent and standard rehabilitation brings only limited improvement. Therefore, therapists suggest reinforcing the compensatory strategies used by such patients such as voice, figure, and gait recognition to help with the identification of a particular person, which promotes their social functioning. New neurotechnologies, especially QEEG/ERPs, displays of functional brain impairment in prosopagnosia, may be helpful in developing an appropriate neurotherapy protocol and create the conditions for other forms of rehabilitation in such patients. The purpose of our study was twofold: 1) to evaluate QEEG/ ERPs shows of post-stroke functional impairment associated with prosopagnosia, 2) to construct a neurofeedback protocol based on these indices to support the neuropsychological rehabilitation of the case study described herein.

Case report: We present the case of a 23-year-old right-handed student of the Graphics Faculty of the Academy of Fine Arts, with chronic associative prosopagnosia after infection with SARS-CoV-2 followed by Covid-19 and a right hemisphere stroke. He was referred in April 2021 for diagnosis and therapy at the Reintegration and Training Center of the Polish Neuropsychological Society (PTNeur). Six months earlier, in October 2020, the patient had been admitted to the Infectious Disease Hospital. COVID-19 was diagnosed based on coronavirus 2 (SARS-CoV-2) reverse transcription PCR (RT-PCR) on a nasopharyngeal swab. The neurological examination revealed muscle weakness on the left side of the body, slow and aprosodic speech, preserved comprehension, and acute left homonymous hemianopsia, as well as prosopagnosia and mirror symptom. The patient was sedated and mechanically ventilated for six days. The CT-scan showed foci in the posterior part of the superior temporal lobe and hyperintense changes in the blood supply area of the right middle cerebral artery. After 30 days of hospitalization, the patient was discharged from this hospital and referred to an outpatient rehabilitation center for five months. Therapy improved his general condition but did not remove the chronic prosopagnosia: a personal tragedy for the patient which prevented him from continuing his studies. He was diagnosed at the PTNeur Reintegration and Training Center within the next few weeks: (1). ophthalmologic examinations revealed no pathology; (2) neuropsychological testing confirmed the presence of chronic apperceptive prosopagnosia; (3) examination of event-related potentials (ERPs) revealed a large delay of the N170 wave, particularly on the right side, indicating a slowing of the rate of nerve impulses in early face processing and a cause of prosopagnosia. The patient was referred for rehabilitation: he participated in 20 sessions of individually tailored anodal transcranial direct current stimulation (tDCS) twice a week for ten weeks, and in parallel, for individualized Prosopagnosia Symbolic Art Therapy provided once a week for ten weeks. By the end of therapy, the patient was not only recognizing but also painting portraits of faces. He returned to college, finished and defended a master's thesis in Artistic Drawing, in which facial presentation played an important role.

Conclusions: Quantitative EEG (QEEG) and event-related potentials (ERPs) neuromarkers helped to understand the mechanism of prosopagnosia and to choose an individualized protocol, thus the appropriate application of tDCS in our patient, which accelerated the recovery of the ability to perform complex tasks and created the conditions for Symbolic Art Therapy. Modern medicine can successfully use such a management protocol in individuals with chronic prosopagnosia.

Key words: SARS-CoV-2, neuroCOVID-19, ischemic stroke, prosopagnosia, painting, art therapy

INTRODUCTION

Since the first confirmed case in Wuhan, China, on December 31, 2019, the novel coronavirus (SARS-CoV-2) has spread quickly, infecting 197 million people in the world as of the end of July 2021. Research, since this first detection, has indicated that people contracting the virus and developing coronavirus disease (COVID-19), may suffer not only respiratory, and other organ challenges but also a wide range of neurological and mental disorders and deficits (Borges do Nascimento et. al 2020; Mao et al. 2020; Paniz-Mondolfi et al. 2020; Paterson et al. 2020; Pinzon et al. 2020; Rogers et al. 2020;Varatharaj et. 2020). Neurological symptoms occur in approximately one-third of hospitalized patients with COVID-19 (Engin et al., 2021). Among these symptoms, ischemic strokes due to thrombotic complications are common in one-third of COVID-19 intensive care patients. A higher incidence of large artery ischemic stroke has also been reported in young patients without cardiovascular risk factors (Oxleyet al., 2020). Brain involvement of severe acute respiratory syndrome-coronavirus-2 (SARS-CoV-2) is eventuated by several routes, including hematogenous spread, transsynaptic entry through infected neurons, the olfactory nerve, ocular epithelium, vascular endothelium, and an impaired blood-brain barrier. Besides the high angiotensin-converting enzyme-2 (ACE2) binding affinity and FURIN preactivation, SARS-CoV-2 maintains efficient neuronal entry while evading immune surveillance by using basic in and neuropilin-1 receptors (DosSantos et al. 2020).

Coronavirus disease 2019 (COVID-19) may increase the risk of an acute ischemic stroke (Merkler et al., 2019), similar to the increased risk of 3.2-fold to 7.8-fold seen within the first three days after other respiratory tract infections (Warren-Gash et al., 2018). A review of the subject literature in April 2020 (Qureshi et al. 2020) showed that the proportion of patients with COVID-19 who have an acute ischemic stroke, was estimated to be 4.9% (95% CI, no continuity correction, 2.8%-8.7%) during the initial hospitalization. Using similar assumptions, an estimated 182 485 and 269 383 patients who have COVID-19 will also have an ischemic stroke; so considering 9 988 254 patients who developed COVID-19 in the world as of June 27, 2020 an estimated 21% to 31% of patients with COVID-19 required hospitalization (Qureshi et al. 2021). Subsequently, several small case series have reported the occurrence of an ischemic stroke in patients with COVID-19.7–12 (CDC Covid-Response Team). The increased risk of ischemic stroke is probably multifactorial, with activation of coagulation and inflammatory pathways as reflected in increased fibrin D-dimer levels, erythrocyte sedimentation rate, lactic acid dehydrogenase, and lymphopenia (Li et al. 2020; Qin et al. 2020; Qureshi et al. 2021).

An international panel of stroke experts from 18 countries recommended further studies to understand whether there are differences in the risk factors, manifestations, response to treatment strategies, and outcomes in acute ischemic stroke patients with COVID-19 (Aknin et al. 2021). Qureshi et al. (2021) identify risk factors, comorbidities, treatment strategies, and outcomes in patients with

an ischemic stroke derived from a large cohort of COVID-19 patients. They found that acute ischemic stroke was infrequent in patients with COVID-19 and usually accompanied by other cardiovascular risk factors. The risk of discharge to a destination other than home or death increased 2-fold with the occurrence of an acute ischemic stroke in patients with COVID-19.

Knowledge about the effects of COVID-19 on the brain is rapidly accumulating (Ellul et al 2020), as reflected in the increasing use of the term "neuro-COVID" (Goldberg et al. 2020). Neurological manifestations are increasingly described, including encephalopathy, neurocognitive symptoms (Helms al 2020; Mao et al. 2020; Hess et al., 2020) and speech/language disorders, such as aphasia (Pensato et al. 2020). However, the pathogenic mechanisms of these symptoms are still being debated in COVID-19 patients. Searching PubMed and Google scholar for the terms' COVID-190, 'long COVID', 'SARS-CoV2', 'cognition', 'brain fog', 'aphasia', 'apraxia', 'agnosia' and 'prosopagnosia' highlights a growing body of studies reporting health changes that persist beyond the acute and sub-acute phases of post-COVID-19 infection, often termed 'long COVID'. Much of this work includes small-scale studies and self-reported cognitive problems with little information regarding whether COVID-19 infection links up to objectively measured cognitive deficits or how it differs from the respiratory symptom severity or hospitalization status of the general population. Furthermore, many previous studies have been limited insofar as they lack a sufficient scope and scale to account for the key socio-demographic variables associated with COVID-19 illness, e.g. age, racial-ethnic group, preexisting medical conditions and symptoms of depression, anxiety or insomnia. However, analysis of the performance of cognitive tests supported the hypothesis that COVID-19 has a multi-domain impact on human cognition comprising the "Long COVID" cognitive symptoms that start at the early-chronic phase (Hampshirea et al. 2021). One of these symptoms is prosopagnosia that coexists with deficits of attention and perception, memory, executive symptoms,-aphasia, apraxia, agnosia, and anosognosia after a stroke associated with SARS-CoV-2 infection and neuroCOVID-19 disease.

Prosopagnosia (from Greek *prósōpon*, meaning "face", and *agnōsía*, meaning "non-knowledge"), also called **face blindness**, is a cognitive disorder of face perception in which the ability to recognize familiar faces, including one's own face (self-recognition) is impaired, while other aspects of visual processing (e.g., object discrimination) and intellectual functioning (e.g., decision-making) remain intact. The incidence of prosopagnosia after a stroke is difficult to establish, but in one clinical sample, about half of those who survived a right hemisphere stroke had prosopagnosia (Cousins 2013). There are two types of prosopagnosia: (1) acquired and (2) congenital (developmental). Acquired prosopagnosia results from occipito-temporal lobe damage and is most often found in adults. This is further subdivided into apperceptive and associative prosopagnosia. In congenital prosopagnosia, the individual never adequately develops the ability to recognize faces (Mayer, Rossion 2007).

A specific brain area usually associated with prosopagnosia is the fusiform gyrus,which activates precisely in response to faces. The functionality of the fusiform gyrus allows most people to recognize faces in more detail than similarly complex inanimate objects. The right hemisphere fusiform gyrus is more often involved in familiar faces recognition than the left. It remains unclear whether the fusiform gyrus is only specific for the recognition of human faces or is it also involved in highly trained visual stimuli (Gainotti & Marra 2011). Studies suggest that the face recognition impairments caused by left hemisphere damage are due to semantic defect blocking retrieval processes that are involved in obtaining person-specific semantic information from the visual modality (Biotti et al. 2016).

Prosopagnosia might be associated with other disorders caused by lesions of nearby brain areas and their neuronal connections: left hemianopsia (loss of vision from the left side of space, associated with damage to the right occipital lobe), achromatopsia (a deficit in color perception often linked with unilateral or bilateral lesions of the temporo-occipital junction), and topographical disorientation (a loss of environmental familiarity and difficulties in using landmarks following right hemisphere lesions of the posterior part of the parahippocampal gyrus and anterior part of the lingual gyrus) (Gainotti & Marra 2011; Sanada et al. 2021).

De Renzi et al. (1991) has distinguished two types of acquired prosopagnosia: (1) apperceptive (agnostic), (2) associative (semantic) (Table 1).

Prosopagnosia after infection by SARS-CoV-2, neuroCOVID-19 and stroke effects the human brain for a long term with severe implications for cognition, emotions and behavior (Pąchalska, Bednarek, Kaczmarek 2021). Unfortunately, the articles devoted to the evaluation of the QEEG/ERPs indexes of functional brain impairment after such a disease are scarce. It makes it difficult to construct a proper neurotherapy protocol as well as to evaluate effectiveness of neurotherapy.

The purpose of our study was twofold: (1) to evaluate the QEEG/ERPs neuromarkers of post-stroke functional impairment associated with prosopagnosia, (2) to construct a neurofeedback protocol based on these indices to support the neuropsychological rehabilitation of the case study described here.

Table 1. Two types of prosopagnosia

Type	Brain damage	Symptoms
Apperceptive	Right occipital temporal area The lingua gyrus of the right hemishere	Lack of ability to recognize faces both real and depicted in images as well as to recognize emotions in those faces. Preserved ability to recognize people based on non-face-related cues such as clothing, hairstyle, skin color, or voice.
Associative	Right anterior temporal region; impaired function of the parahippocampal cortex	Preserved face perception, but impaired connections between early face perception processes and the semantic information about people that we store in our memory. Difficulty in identifying the persons seen (who they are, i.e., what name, age, gender, relationship, occupation, etc.).

Source: elaborated by M. Pąchalska

CASE STUDY

We present the case of a 23-year-old right-handed student of Graphics Faculty at the Academy of Fine Arts, with chronic associative prosopagnosia after infection by SARS-CoV-2 followed by Covid-19 disease, and a right hemisphere stroke. He was referred in April 2021 for diagnosis and therapy at the Reintegration and Training Center of the Polish Neuropsychological Society (PTNeur).

Six months earlier, in October 2020, the patient had developed a severe headache, loss of smell (anosmia), loss of taste (ageusia), dizziness, fatigue, imbalance, generalized weakness, a body temperature of 38.9°C, and, consequently, he was admitted to the Infectious Diseases Hospital.

Laboratory tests: COVID-19 was diagnosed based on coronavirus 2 (SARS-CoV-2) reverse transcription PCR (RT-PCR) on a nasopharyngeal swab. Arterial blood gas analysis and blood tests showed hypocapnic hypoxemia (PaO2/FiO2 = 268), an elevated D-dimer level (3.43 mg/L), lymphopenia, and elevated inflammatory markers (CSF-NF-L). Microbiology analysis of CSF was positive RT-PCR for SARS-CoV-2, with unremarkable herpesvirus DNA [herpes simplex virus types 1 and 2 (HSV1, HSV2), cytomegalovirus (CMV), Epstein–Barr virus (EBV), human herpesvirus 6 (HHV6), and varicella zoster virus (VZV)], enterovirus DNA, Gram stain, and bacterial culture.

Neurological examination: muscle weakness on the left side of the body, slow and aprosodic speech, preserved comprehension acute left homonymous hemianopsia and prosopagnosia with mirror syndrome.

Treatment: the patient started on oxygen therapy, hydroxychloroquine, low-molecular-weight heparin, intravenous low-dose steroids, and antibiotics. The next day, the neurological symptoms progressed and his respiratory status worsened, requiring admission to an ICU. The patient was sedated and placed on mechanical ventilation for 6 days. Due to a suspected a stroke, the patient underwent a CT-scan, which confirmed the hyperintense lesions in the blood supply area of the right middle cerebral artery. After 30 days of hospitalization, the patient was discharged and referred to an outpatient rehabilitation center for 5 months. Therapy improved his general condition but did not remove the chronic prosopagnosia, which was a personal tragedy for the patient and prevented him from continuing his studies.

In the next few weeks, he was diagnosed at the PTNeur Reintegration and Training Center. On admission, he complained that he was heartbroken because he still could not see people's faces; he did not go anywhere because he was frightened by faceless people. He also repeatedly complained that a great tragedy had befallen him since he could not see faces which prevented him from continuing and completing his studies at the Faculty of Graphic Arts. The prosopagnosia protocol was used in the diagnostic process: (1). CT-scan; (2). ophthalmologic examinations; (3). neuropsychological testing; (4). examination of quantitative electroencephalography (QEEG) and event-related potentials (ERPs).

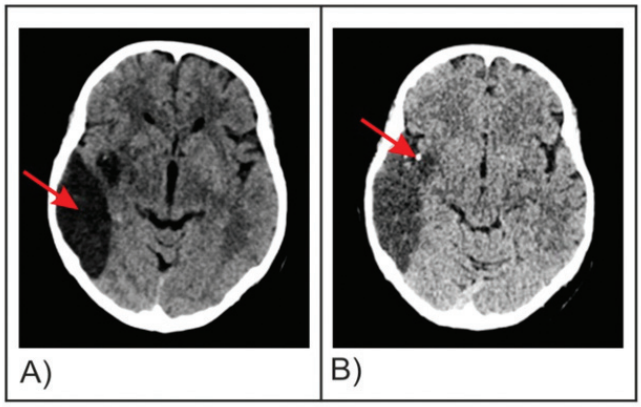

Fig. 1. CT-scan: A) Foci in the posterior part of the superior temporal lobe; (B) hyperintense lesions in the blood supply area of the right middle cerebral artery.
Note: The lesions exist in the right hemisphere

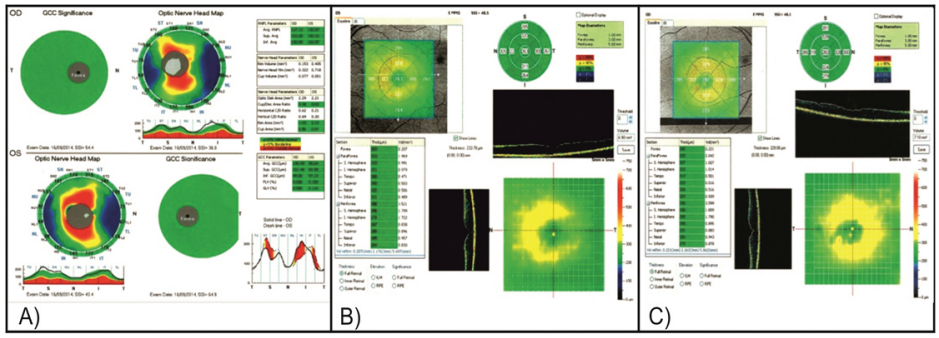

Fig. 2. The ophthalmolgical tests: A). Maps of nerve fibers and GCC complexes, B). Maps of the macula morphology in the right eye, C). Maps of the macula morphology in the left eye

The CT-scan showed visible pathology: the foci in the posterior part of the superior temporal lobe and hyperintense lesions in the blood supply area of the right middle cerebral artery (Fig 1 A & B).

The ophthalmolgical tests did not show any visible pathology in the anterior and posterior segment. The left eye globe was positioned at a slight exophthalmos (the right eye 15 mm; the left 16mm). Maps of nerve fibers and GCC complexes did not show any pathology (Fig.2 A). The examination carried out using 2010 Carl Zeiss Meditec Humphrey Visual Field Analyzer revealed that the visual field was normal for both eyes. Optical coherence tomography (OptovueRTVue OCT) did not show any significant changes in the morphology of the macula and nerve fiber layer (Fig. 2B and 2C).

Neuropsychological examination

The neuropsychological assessment using the Polish version of the Wechsler Memory Test (WMS-III) showed only disturbances to the working memory. Logi-

Fig. 3. Sample Picture from the Face and Objects Recognition Test (CFORT).
Source: Pąchalska & MacQueen 2000

cal memory and spatial-visual memory both in instant and delayed reproduction were not disturbed. However, examination with the use of the Cracow Face and Objects Recognition Test (CFORT) (Pąchalska and MacQueen 2000) revealed severe apperceptive prosopagnosia. He was neither able to recognize or differentiate even one from 20 famous and unknown faces depicted in images nor able to recognize any emotions in those faces (fig. 3 A,B). But he perfectly managed the part of the test that required the recognition of 20 objects (Fig. 3 C,D).

In real life situations, he could not even recognize the faces of his family and friends. When he wanted to recognize people, he relied on external clues (clothing, hairstyle, scars, glasses, tone of voice, etc.) to identify them.

Neurophysiological testing

EEG recording

The electroencephalogram (EEG) was recorded with the Mitsar 21-channel EEG system, with a 19-channel electrode cap with tin electrodes that included Fz, Cz, Pz, Fp1/2, F3/4, F7/8, T3/4, T5/6, C3/4, P3/4, O1/2. The electro-cap was placed on the scalp according to the standard 10–20 system. Electrodes were referenced to linked earlobes (off-line), and the input signals were sampled at a rate of 250 Hz (bandpass 0.5–30 Hz). The ground electrode was on the forehead. Impedance was kept below 5 kΩ. The patient was sitting in a comfortable chair looking at a computer screen (17 inches) 1.5 meters in front of him. All recordings were made by the first author of this article. The ERP wave forms

were averaged and computed off line, and trials with omission and commission errors were automatically excluded.

Behavioral task

The task consisting of 400 trials were sequentially presented to the subject every three seconds. Three categories of visual stimuli were used: (1). 20 different images of animals – referred to later as A; (2). 20 different images of plants – P; (3). 20 different images of people of different professions (presented together with an artificial "novel" sound), referred to as H. The trials consisted of presentations of pairs of stimuli with inter-stimulus intervals of 1 s. The duration of stimuli presentation was 100 ms. We used four trial categories: A-A, A-P, P-P, and P-H. In the trials with A-A and P-P pairs, the first and the second stimuli were identical (physically the same). The trials were grouped into four sessions, with 100 trials in each. In each session, a unique set of five A stimuli, five P and five H stimuli was selected. Each session consisted of a pseudo-random presentation of 100 pairs of stimuli, with an equal probability for each category and each trial category (Kropotov 2016). The task was to press a button with the right hand to all the A-A pairs as fast as possible, and to stop pressing in response to other pairs. The patient performed 10 trials without recording to see if he understood the instruction. He rested for a few minutes after completing 100 trials. Stimuli occupied about 3.8° of the visual field around the center of the screen. Visual stimuli (and were selected to have) had similar 2D sizes and luminosities.

Artifact correction procedures

Eyeblink artifacts were corrected by zeroing the activation curves of individual independent components corresponding to eye blinks. These components were obtained by the application of Independent Component Analysis (ICA) to the raw EEG fragments as described in Kropotov (2016). Epochs with excessive amplitude of filtered EEG and/or excessive faster and/or slower frequency activity were automatically marked and excluded from further analysis. The exclusion thresholds were set as follows: (1). 100 μV for non-filtered EEG; (2). 50 μV for slow waves in the 0–1 Hz band; and (3). 35 μV for fast waves filtered in the band 20–35 Hz. In addition, we visually inspected the recordings and excluded the remaining artifacts.

EEG spectra

EEG spectra were computed for Eyes Open, Eyes Closed and the GO/NOGO task conditions separately. The artifact free fragments of EEG were divided into 4 sec epochs with a 50% overlap. The Hanning time window was applied. The EEG spectra were computed for each epoch and averaged. The Mean value and standard deviations for each 0.25 Hz bin were computed. For comparison of the EEG spectra pre and before intervention, the t-test was used.

RESULTS

QEEG: Comparison of the EEG spectra of the patient in three conditions (Eyes Open, Eyes Closed, and the cued visual GO/NOGO task) showed **excessive slow activity** over the right posterior temporal electrode. Fig.4 depicts EEG spectra differences between the patient and the grand average spectra for the healthy control group (N=100).The maps of the difference curves are shown at the bottom of Fig.4. at frequencies where the most significant deviations from the control group were observed. It was found that besides the excessive 5 Hz activity over T6 the patient also shows excessive mu-rhythm at C4. The EEG traces of the abnormal patterns are presented at the top.

Behavior

The behavioral parameters of the patient in the cued GO/NOGO task are presented in Table 2. It can be observed that the patient is inconsistent in his responses. In line with behavioral abnormalities all executive components of ERPs in response to GO and NOGO stimuli are reduced significantly in amplitude. Be-

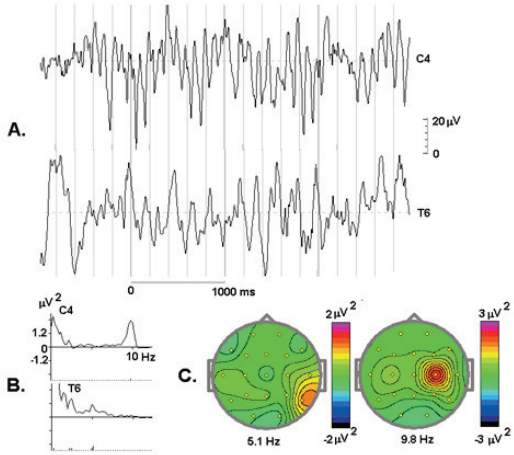

Fig. 4. The patient's EEG spectra deviations from the normative data. A: traces of EEG recorded at C4 and T6 in the eyes open condition. B: EEG spectra difference curves (patient – healthy controls) for C4 and T6 electrodes. C: Maps of EEG spectra differences (patient-grand average for the healthy control group) for the two frequencies at which the most significant differences are found. The small vertical bars below the curve indicate $p<0.05$ for the statistical significance level

Table 2. Comparison of the patient's behavioral parameters in the cued GO/NOGO task with the normative data

Tested persons	Omission errors	Commission errors	Reaction time	Error in standard deviation response time
Patient	**88**	10	614	**42.1 ms**
Norms	**6.3%**	2.6%	480ms	**13.2 ms**
p-value difference	**0.00**	**0.16**	**0.12**	**0.00**

sides the executive components, the related visual components are found to be significantly deviant from the normative data.

Fig. 5 shows the patient's ERPs in response to the continue (animal) and discontinue (plant) cues in the trials. One can see a **large delay of the N170 wave, especially on the right side**.

From prosopagnosia to new life

Taking into account the magnitude of this student's tragedy, and here caused by his inability to continue his studies in the Department of Graphic Arts, a therapy aimed at reducing his prosopagnosia was proposed. He took part in 20 sessions of individually tailored anodal transcranial direct current stimulation (tDCS) over the supplementary motor area (SMA) but not pre-SMA to promote short-term visuomotor learning, as suggested by recent studies (Vollmann 2013; Parasuraman, McKinley 2014). The neuromarkers obtained with the use of QEEG/ERPs were helpful in choosing the appropriate protocol of tDCS: neurostimulation with the use of this method was administered twice a week for 10 weeks.

He also took part in sessions of individually conducted Symbolic Art Therapy (Pąchalska 1986) directed toward the reduction of prosopagnosia through the vividness of the visual image of the faces (Kaczmarek 1991). This program was administered once a week for 10 weeks, and the entire treatment lasted 10 weeks.

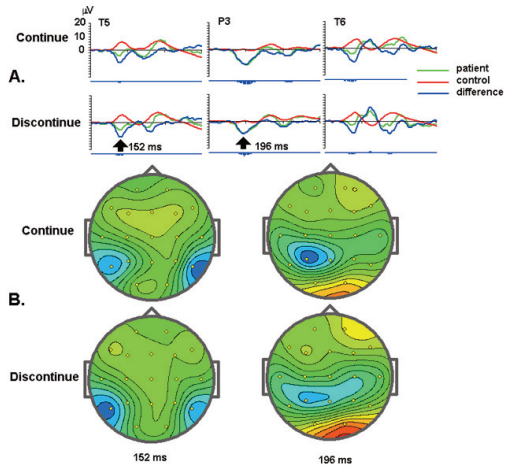

Fig. 5. Delay of the visual related waves in the ERPs of the subject in comparison to healthy controls. A: ERP wave forms in response to continuing cues (an image of an animal at the first place in the trial) and discontinue cues (an image of a plant at the first place in the trial) for the patient (green line) in comparison to the grand average for the healthy control group (red line) and the difference waves (blue line) with indexes of statistical difference (patient-norm). The large vertical bars below the curves indicate $p<0.001$ for the statistical significance level. B: The maps of the difference waves computed for two different moments (162 and 196 ms) after the first stimulus presentation

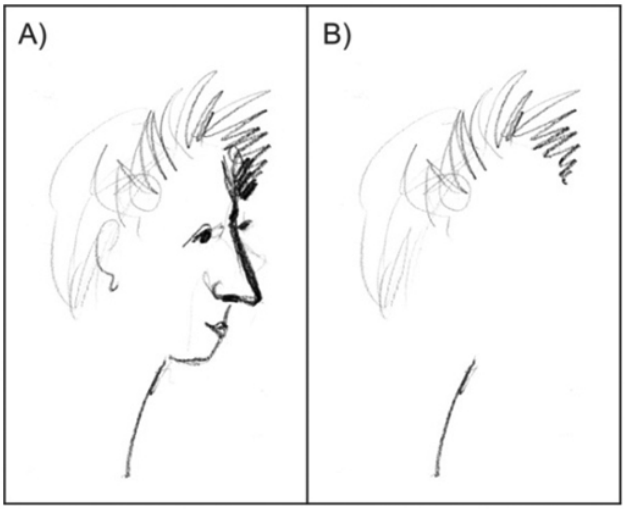

Fig. 6. Art therapy: first copy attempts (A) a self-portrait taken before the disease; B) a copy of a self-portrait made after the illness

Initially, the patient copied his previously drawn graphics of famous people and a self-portrait (see: Fig. 6).

Despite many subsequent attempts at copying, the patient did not manage to accomplish this task, and he became very frustrated. He could not understand what was happening to him and why he had lost his previous abilities. On the next copy of the portrait, he expressed this frustration with the words: *I don't know what on earth is here because I can't see it. Help me!* (Fig. 7).

Fig. 7. Art therapy: Subsequent copy attempts (A) a self-portrait taken before the disease; B) a second copy of a self-portrait made after the illness with commentary "I don't know what on earth is here because I can't see it. Help me!

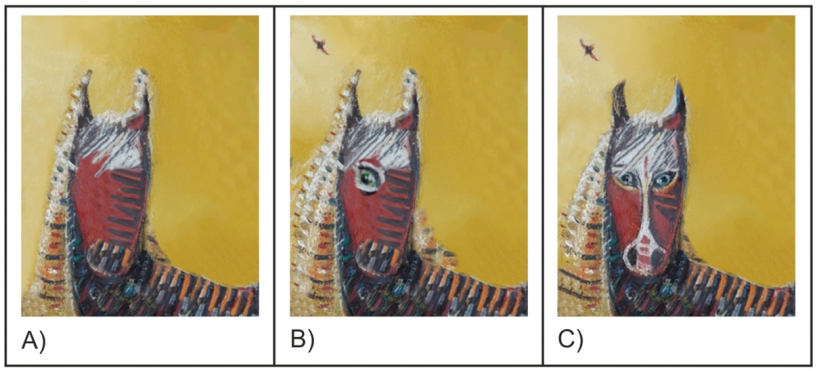

Fig. 8. Symbolic Art Therapy: Subsequent copy attempts. A) Patient's painting of the horse (after 2 weeks of treatment) note the perfection in painting and the absence of facial elements. B) Patient's painting of the horse (after 4 weeks of treatment): note the one eye that appears in the image. C) Patient's painting of the horse (after 6 weeks of treatment): this almost exact copy of the work draws one's attention

Fig. 9. Symbolic Art Therapy: Sketch for a portrait of the patient's doctor. Note that the patient has marked all the facial elements

As a second step, the subject was asked to analyze the pictures of famous painters, especially the pictures of animals. The faces of horses were chosen by the patient. He had to introduce the elements in the face. The results of six weeks of such therapy are illustrated in Fig. 8.

In the third step, the patient was asked to draw a portrait of his own choice. He chose to draw one of his favorite persons, who had helped in the process of his diagnosis and therapy. Note that the patient marked all of the facial elements (Fig. 9).

To sum up: The case study described above shows that appropriate neurostimulation and activities consistent with the patient's needs and interests have resulted in recovery. Neurons and their connections, as we know, can make the world or break the world of perception. In the case of our patient, it was possible to create his world again, that is to restore his disturbed process of seeing and, consequently, make it possible to paint portraits of faces. The patient's public test came when he returned to his studies. His recovery enabled him to finish and defend his master's thesis in Artistic Drawing, in which facial presentation played an important role.

DISCUSSION

Our patient revealed prosopagnosia after an acute stroke lasting >24 hours. The stroke was linked to symptoms of SARS-CoV-2 infection, and COVID-19. He had a markedly elevated D-dimer level, which is associated with an increased incidence of ischemic stroke (Mao et al. 2021; Markus &Brainin 2020; Frisulloet al. 2020; Rubin et al. 2019; Rostami &Mansouritorghabeh 2020). His brain MRI confirmed stroke and structural lesions nearby the areas of the visual cortex that respond to images of faces in healthy human subjects (see: Rossion et al. 2003). Neuroimaging studies have identified at least two bilateral areas of the extrastriate visual cortex that respond to the images of faces in healthy human subjects. These are the middle fusiform gyrus [the 'fusiform face area' (FFA)] and, more posteriorly, the inferior occipital cortex ['occipital face area' (OFA)], with a right hemisphere dominance (Rossion et al. 2003). However, it is not yet clear how these regions interact with each other and whether they are all necessary for normal face perception. Also the mechanisms underlying prosopagnosia are still not fully recognized (Biotti et al. 2016).

A great help in understanding these mechanisms are studies using functional neuromarkers that allow for the assessment of cerebral performance in milliseconds, such as qEEG/ERPs (Pąchalska, Kaczmarek, Kropotov 2014; Kropotov 2016; Kopańska et al 2021). The ERP data show that the primary dysfunction resides in the sensory system, in the way the patient's brain perceives the visual world concerned with extracting information from the semantic memory. This process takes place in the ventral visual stream, in the inferior temporal cortex in particular. The information processing in the inferior temporal area is reflected in the N170 wave of the visual-related ERPs. The delay in this process is a key to understand prosopagnosia, and therefore the dysfunction observed in the patient described here. It should be also stressed that previous research revealed that **the brain and mind work in time,** and any acceleration, as well as deceleration of this process, will cause dysfunction of a particular area of the web of neuronal connection patterns.

Microgenetic Theory: Brain and Mind in Time

Apperceptive prosopagnosia can be explained as an error in the perception process in terms of microgenetic theory that is, difficulties in processing the face

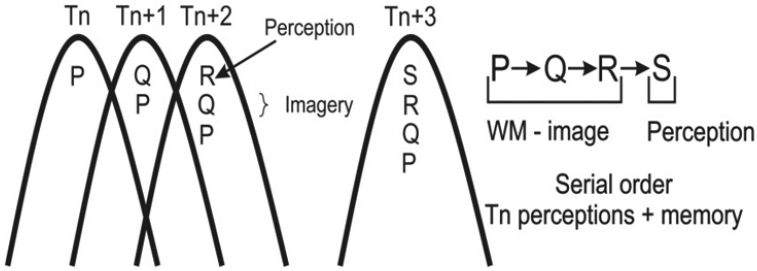

Fig. 10. The perception (P) at Tn is replaced at Tn+1 by another perception (Q), which may resemble or differ from that at Tn. Perceptual stability depends on resemblance; change depends on difference. Within the perception (arrow, R), the mind/brain state at Tn+2 revives Tn+1 almost completely, so that the image of P at Tn+2 is prior to the object (Q), and so on. Over a brief succession of mental states, P, Q, and R represent images of past perceptions revived to a decreasing extent in the oncoming present and graded according to this revival. An eidetic image is a near-complete revival. A memory image is a vague recurrence at some psychic distance from a present object. At Tn+3, the series of images, P, Q, and R, form an order antecedent to the perception (S). The perception and memory of serial order depend on the perception developing out of the long-term memory. Serial order occurs within the present but depends on succession for the layering of prior experience
Source: Pąchalska, MacQueen, Brown (2012)

in milliseconds and delays in this processing. The process of face perception in a healthy person takes place in milliseconds, gradually recurring over time and developing the perception of individual elements of the face because this is how the brain works (Fig. 10).

To improve the disrupted process of perception, especially face perception in our patient, we used an individually tailored program:

1. tDCS directed to improve the delayed information processing in the inferior temporal area, as reflected in the N170 wave of the visual-related ERPs, in accordance with the procedure elaborated by Kropotov 2016);
2. Symbolic Art Therapy, assuming that creativity is enormously adaptive for individuals and society (Pąchalska 1986).

To our satisfaction and the patient's joy, the neurostimulation and activities consistent with the patient's needs and interests have resulted in recovery.

CONCLUSIONS

Individually-tailored tDCS given simultaneously with Symbolic Art therapy resulted in the recovery of our patient with chronic prosopagnosia. The neuromarkers in Quantitative EEG (QEEG) and Event related potentials (ERPs) made it possible to delineate the mechanism of prosopagnosia and choose the tDCS protocol. Noninvasive brain stimulation accelerated skill acquisition in complex tasks and provided the conditions for Symbolic Art Therapy. After the combined therapy, the patient took up his degree studies again and passed the master thesis in Artistic Drawing, of which a significant component was the presentation of the face.

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Address for correspondence:
Maria Pąchalska
Chair of Neuropsychology and Neurorehabilitation
Andrzej Frycz Modrzewski Cracow University, Cracow, Poland
Herlinga-Grudzinskiego 1
30-705 Krakow
e-mail: neuropsychologia23@go2.pl