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APPLICATION OF FUNCTIONAL NEUROMARKERS FOR CONSTRUCTING PROTOCOLS OF NEUROTHERAPY AND MONITORING THE SUCCESS OF TREATMENT IN CHRONIC CROSSED TRANSCORTICAL SENSORY APHASIA: A CASE STUDY

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SUMMARY

Background:

The goal of the study was threefold: 1) to evaluate QEEG/ERPs indexes of functional brain impairment after a stroke associated with chronic crossed transcortical sensory aphasia, 2) to construct a neurotherapy protocol to compensate for this functional damage, and 3) to assess the changes in the functional neuromarkers induced by the neurotherapy sessions.

Case study:

A 72-year-old, strongly right-handed woman with atrial fibrillation suddenly developed cerebral embolism of the right middle cerebral artery. She was treated conservatively, and the left hemiparesis, and aphasia – in a moderate degree, consequently existed. A CT-scan showed a large infarct lesion partially parallel to Wernicke's area. After one year of ineffective aphasia therapy we constructed an experimental neurotherapy protocol (TMS combined with comprehensive aphasia therapy) on the basis of an assessment of the spontaneous QEEG and event-related potentials (ERPs) in the cued GO/NOGO. The patient was assessed before and after the neurotherapy sessions by the same methodology.

Conclusions:

It was found that before the TMS treatment the temporal area (T6) generates a strong P2 wave in response to visual stimulus indicating a hyper-sensitivity of the neurons located at temporal areas of the right hemisphere. This was connected with crossed transcortical sensory aphasia found within the aphasia profile in the Polish version of the Western Aphasia Battery (K-WAB). The TMS sessions reduced this hyper-sensitivity substantially. The patient speech returned to the norm, she was to return to social life.

Keywords: stroke, language disturbances, TMS

INTRODUCTION

Clinical research on crossed transcortical sensory aphasia (CTSA) is limited because it occurs so infrequently in patients after a stroke that it is very difficult to perform systematic studies (Ardilla 2014). An even worse situation occurs when one would like to evaluate the effectiveness of the treatment for these patients (Pachalska 2011).

Transcortical (extrasyllabic) sensory aphasia (TSA) has been a polemic syndrome; frequently it is considered as a subtype of Wernicke's aphasia. Seemingly, the polemic is related to the way TSA is defined and the elements included in its definition. Two integrative revisions of TSA are available (Berthier, 1999; Boatman et al., 2000). In general, it is considered that extrasyllabic (transcortical) sensory aphasia includes the following elements:

1. Good repetition (the patient repeats words and sentences presented by the examiner, regardless if they are incorrect and even in a foreign language);
2. Fluent conversational language;
3. Significant amount of verbal paraphasias and neologisms;
4. Empty speech.

TSA presents similar deficits as in Wernicke's aphasia, but repetition ability is spared and phoneme discrimination impairments are not found (Ardilla 2014). Some authors also include a semantic jargon in the definition of TSA (Bub & Kertesz 1982). But jargon is not a required symptom for the diagnosis of TSA (Benson & Ardilla 1996; Ardila 2013). By the same token, other language impairments can also be found, such as poor naming, and preserved oral reading with impaired reading comprehension, but their presence is not essential to establish the diagnosis of TSA (Berthier, 1999).

Recent reports support the assumption that transcortical sensory aphasia (TSA) is usually found associated with extensive lesions of the left hemisphere (Warabi et al., 2006), generally involving large portions of the temporal-parietal-occipital areas. According to Alexander, Hiltbrunner, and Fischer (1989), the critical lesion for transcortical sensory aphasia in these patients involved pathways in the posterior periventricular white matter adjacent to the posterior temporal isthmus; pathways that are most likely converging on the inferolateral temporo-occipital cortex. Dronkers and Larsen (2001:29) state that:

“transcortical sensory aphasia always resolves into mild anomia aphasia”

TSA is a fluent aphasia similar to Wernicke's aphasia, with the exception of a strong ability to repeat words and phrases (Rho et al. 2007; Kim et al. 2013; Ardilla 2014). The person may repeat questions rather than answer them („echo-lalia”). However, frequently the variability in the lesions responsible for TSA account for the variability observed in its clinical manifestations, suggesting that TSA does not necessarily represent a single aphasic syndrome (Ardilla 2014). These finding might be also important for CTSA.

Crossed aphasia, including crossed transcortical sensory aphasia (CTSA), is common in left-handed people, and this term is now used exclusively to describe aphasia following a right hemisphere lesion in a right-handed person (Warabi et al. 2006). The prevalence of CA in right-handed patients is reported to be between 0.38 and 3% of all aphasic syndromes. It is a kind of aphasia that involves a damage lesion in the right unilateral hemisphere, specifically in the areas of the temporal lobe of the brain (Coppens et al. 2002). Atypical functional lateralization and specialization for language have been proposed to interpret this type of aphasia (Brown 1975; Dronkers et al. 2000).

The diagnostic criteria for CTSA are similar to those for TSA (Pachalska 2011). The patients should have:

1. language deficit (e.g. poor auditory comprehension, relatively intact repetition, and fluent speech with semantic paraphasias);
2. a lesion in the right unilateral hemisphere;
3. a strong preference for right-hand use without a familial history of left handedness;
4. structural integrity of the left hemisphere;
5. absence of brain damage in childhood.

There have been only a few descriptions of the neuropsychological status of such patients (Warabi et al. 2006; Ardilla 2014; Pachalska 2011).

We report one patient with a right anterior cerebral artery infarction who demonstrated CTSA. Our study is aimed at an evaluation of the reduction of the cognitive disturbances, including language problem, after the neurotherapy program.

CASE STUDY

We report a case of chronic crossed transcortical sensory aphasia (CTSA) that occurred after infarction of the right cerebral hemisphere. A 72-year-old, right-handed woman with atrial fibrillation suddenly developed cerebral embolism of the right middle cerebral artery. She developed the left hemiparesis and moderate aphasia. It should be stressed that she had a strong preference for right-hand use without a familial history of left handedness. She had an absence of brain damage in childhood.

A CT scan, made directly after admission to hospital, showed a hipodense area in the right temporo-parietal region, involving internal capsule and partially lenticular nucleus corresponding to a subacute infarct from the right medial cerebral artery supply region. Hiperdensity of the insular part of the right medial cerebral artery suggests the presence of arterial thrombus (embolisation). A CT scan showed a discrete shift of the cerebral structures to the left from midline, mild cerebellar atrophy (Fig. 1).

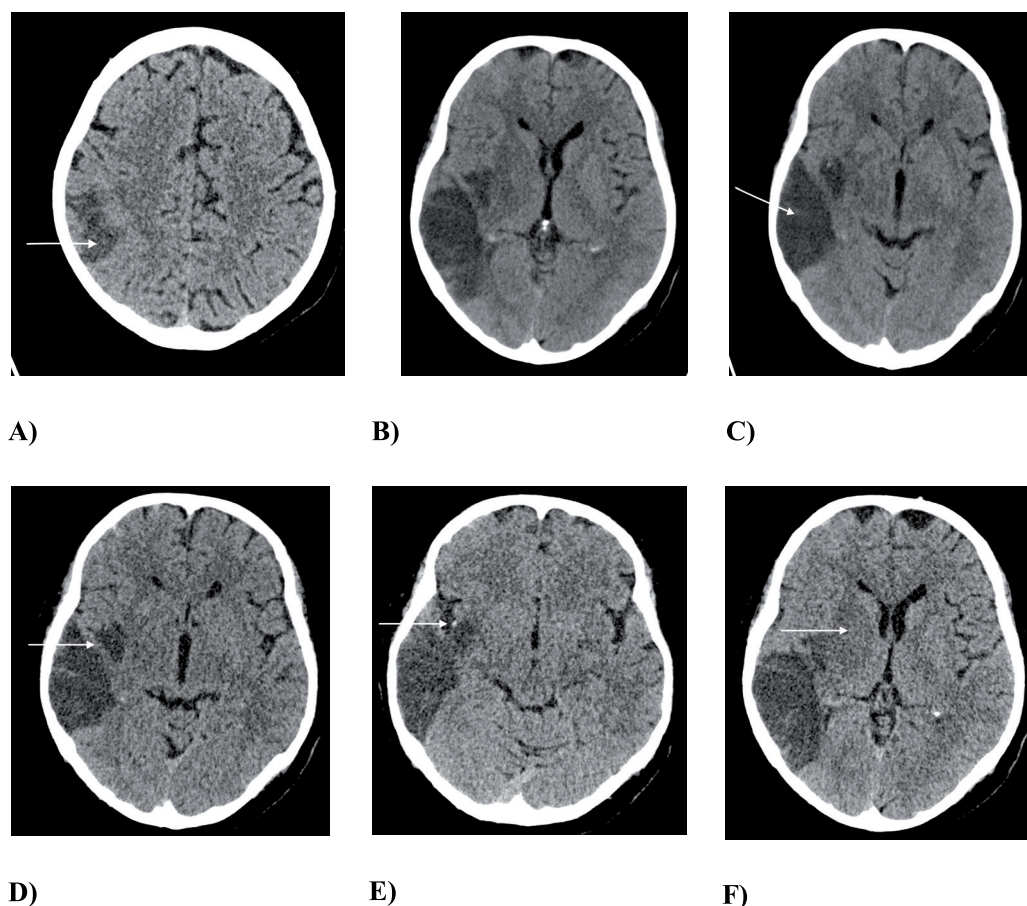


Fig. 1. Brain CT scan of the patient: A) angular sulcus BA 39; B) temporal operculum BA6; C) posterior superior temporal gyrus BA 22; D) spared superior temporal gyrus BA 41-42; E) hiperdense of the right middle carotid artery; F) focal hiperdense of the right middle cerebral artery
Note: The lesions exists in the right hemisphere

Neuropsychological assessment

The aphasia scores were assessed by a Polish version of the Western Aphasia Battery (PL-WAB) (Pąchalska 2010).

The results of the test made three weeks after the stroke (Examination 1), after 12 months, before neurotherapy (Examination 2) and 18 months, after neurotherapy (Examination 3) are shown in Table 1. In the first examination, three weeks after the stroke, she demonstrated crossed transcortical sensory aphasia, e.g., fluency 8.3, auditory comprehension 2.1 repetition 9.6 and object naming 2.4. The speech was fluent but empty, often unintelligible because of semantic paraphasias (e.g. wheelbase instead of glass). She used words in a senseless and incorrect combination. The profile was similar to transcortical sensory aphasia (see: Kertesz 1979). We found a strong ability to repeat words and phrases: she repeated, for

Table 1. The results of the Polish version of the Western Aphasia Battery (PI-WAB) at three weeks (Examination 1), 12 months, before the administration of the experimental neurotherapy, (Examination 2) and 18 months (Examination 3) after the administration of the experimental neurotherapy

Category PI-WAB	After onset		
	Examination 1 (3 weeks/months)	Examination 2 (12 months)	Examination 3 (18 months)
Spontaneous speech (20)	15.5	15.8	19.3
Information content (10) Empty speech	1.5	2.0	9.7
Fluency (10)	8.3	8.5	10.0
Comprehension (10)	2.1	2.7	9.0
Yes/no questions (3)	1.0	1.3	3.0
Auditory word recognition (3)	0.3	0.7	3.0
Sequential commands (4)	2	2.1	4.0
Repetition (10)	9.6	9.7	10.0
Naming (10)	2.4	2.6	9.7

example, a lot of questions given during the test rather than answer them („echo-lalia”). There was also a left side neglect in drawings only. In addition to preserved repetition, both linguistic prosody and affective prosody were well preserved, however an acceleration in the speed of speech occurred.

She received 36 sessions of aphasia therapy with the use of a comprehensive model of rehabilitation (the procedure is described in more detail in Pachalska 2008). However, this procedure was ineffective, and after 12 months, before the administration of the experimental neurotherapy, her aphasia profile did not change much, and a moderate degree of crossed transcortical sensory aphasia (CTSA) consequently still existed (see Table 1).

One year after the stroke she was admitted to the out-patient clinic of the “Promyk Słońca” Foundation in Wrocław. We wrote for her a special neurotherapy program protocol. We re-tested her with neuropsychological testing as well as ERPs before the entire experiment, as well as after the completion of the program (See: Table 1).

The experimental procedure described in the present paper was approved by the respective medical ethics committees. The patient gave written, informed consent for the anonymous publication of her case history.

EEG recording and artifact correction

EEG was recorded with a 19-channel electroencephalographic PC-controlled system, the “Mitsar-201” (CE 0537) manufactured by Mitsar Co., Ltd. Electrodes were applied using caps manufactured by Electro-Cap International, Inc. The tin recessed electrodes contacted the scalp using ECI ELECTRO-GEL. Quantitative data were obtained by means of WinEEG software (Kropotov 2009, 2016; Kropotov & Ponomarev 2011; Kropotov et al 2011). The EEG was initially recorded referentially to the linked ears. The EEG was computationally re-referenced to the common average montage.

Eye blink 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. The method is described in Jung et al. (). In addition, epochs with an 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 follow: (1) 100 μ V for non-filtered EEG; (2) 50 μ V for slow waves in 0-1 Hz band; and (3) 35 μ V for fast waves filtered in the band 20-35 Hz.

ERPs assessment

For assessment indexes of brain functioning (neuromarkers) a variant of the cued GO/NOGO task was used (Kropotov & Ponomarev, 2009; Kropotov et al., 2011). In this task, images of animal (*a*) and plant (*p*) categories served as relevant stimuli. The trials consisted of the presentations of paired stimuli *s1-s2* with inter-stimulus intervals of 1000 ms and inter-trial intervals of 3000 ms. Four categories of trials were used: *a-a*, *a-p*, *p-p* and *p-h+novel sound*, where *h* is an image of a human. The duration of stimuli was 100 ms. The subject's task was to respond by pressing a button with the right hand to *a-a* trials (GO trials) and to withhold from responding in *a-p* trials (NOGO trials). The trials were grouped into four blocks with one hundred trials each. In each block a unique set of five *a*, five *p*, and five *h* stimuli were selected. Each block consisted of a pseudo-random presentation (requiring an equal number of trials in four categories) of 400 trials with 100 trials within each trial category. The patient practiced the task before the recording started. She sat upright in a comfortable chair looking at a computer screen. Stimuli were presented on 17-inch CRT computer screens which were positioned 1.5 meters in front of the subjects and occupied 3.8° of the visual field. The patient rested for a few minutes after each 200 trials. The QEEG/ERP was assessed before and after neurotherapy and compared (the second with the first recording).

Event Related Potentials (ERPs)

Event related potentials (ERPs) were used 1) to assess functional neuromarkers of brain damage for constructing protocols of TMS, 2) to monitor the effects of the TMS sessions on brain functioning. The cued GO/NOGO task was used to index the stages of visual/auditory information flow and the operations of cognitive control. The results of the analysis of the sensory-related component in response to the visual stimulus are presented in Fig. 2.

- a. Event-related potential at T6 before (Pre, red line) and after (Post, green line) sessions of TMS therapy in contrast to the grand average ERP of the healthy control group (HC, N=35, grey line) of the same age. Y-axis – amplitude in micro-volts, X-axis – post-stimulus time in ms. Vertical gray line – onset of ignored visual stimulus of 100 ms duration.
- b. maps of ERPs at maximums of positive late waves (indicated by arrow) for post and pre recordings in comparison to the map for the healthy control group.
- c. sLORETA images of the Post-Pre difference wave.

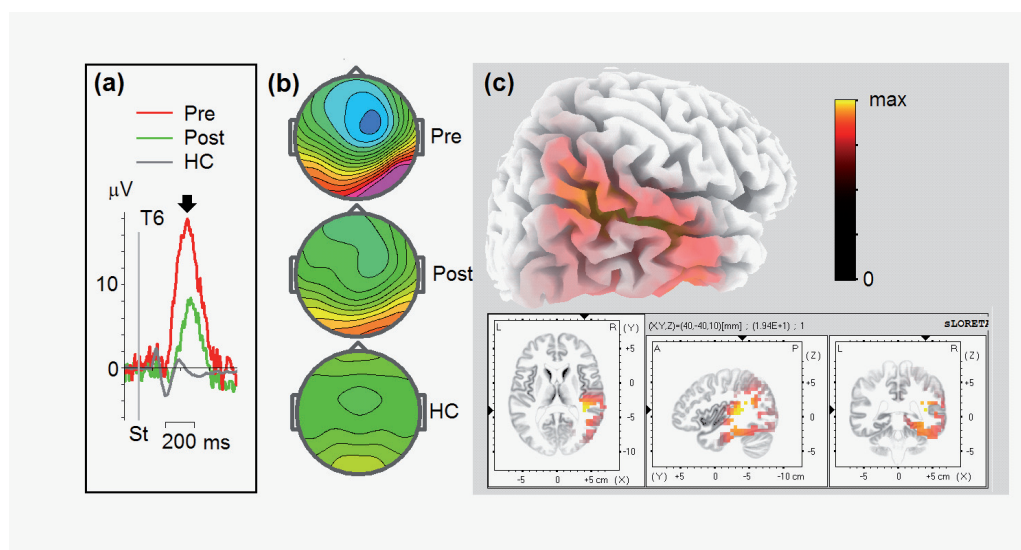


Fig. 2. Brain responses to ignored visual stimuli in the cued GO/NOGO task before TMS therapy and after TMS sessions in comparison to the responses of the healthy control (HC) group. (a) Event-related potential at T6 before (Pre, red line) and after (Post, green line) sessions of TMS therapy in contrast to the grand average ERP of the healthy control group (HC, N=35, grey line) of the same age. Y-axis – amplitude in microvolts, X-axis – post-stimulus time in ms. Vertical gray line – onset of ignored visual stimulus of 100 ms duration. (b) maps of ERPs at maximums of positive late waves (indicated by arrow) for post and pre recordings in comparison to the map for the healthy control group. (c) sLORETA images of the Post-Pre difference wave.

One can see that before the TMS treatment the temporal area (T6) generates a strong P2 wave in response to visual stimulus indicating a hyper-sensitivity of the neurons located at temporal areas of the right hemisphere. The TMS sessions reduced this hyper-sensitivity substantially.

Behavioral parameters

The TMS therapy not only reduced hyper-sensitivity of the right temporal area but also changed the behavioral parameters of the cognitive control as presented in Table 2. One can see that the behavioral parameters of the patient in the task were quite abnormal resulting in an excessive number of omission errors for GO trials, commission errors for NOGO trials and reaction time (RT) variability. These behavioral parameters were normalized after the TMS treatment.

Experimental neurotherapy program

The protocol of the neurotherapy program for this patient was constructed on the basis of QEEG/ERP analysis. She took part in a experimental neurotherapy program which contained TMS sessions combined with aphasia therapy. This includes:

Table 2. Behavioral parameters in the cued GO/NOGO task before (Pre) and after (Post) of TMS sessions in comparison to the averages computed for the healthy control group (HC).

	Omission errors (%)	Commission errors (%)	Reaction time in ms (RT)	Error of RT variance in ms
Pre	27	36	471	34.7
p-value of comparison to HC	<0.01%	<0.01%	0.50	<0.01
Post	12	0	438	12.6
p-value of comparison to HC	0.11	0.78	0.48	0.11
HC	4.7	0.08	396	7.6

- 20 sessions of TMS intervention. Each session consisted of 25 repetitions of 1) low frequency TMS (1 Hz) intended to inhibit the left hemisphere, and high frequency (5 Hz) to stimulate the right hemisphere.
- 20 sessions of aphasia therapy with the use of a comprehensive model of rehabilitation (the procedure is described in more detail in Pachalska 2008).

The experimental neurotherapy program was administered by the same therapist team. The experiment was reviewed and approved by the respective medical ethics committees, and the patient gave written informed consent for the anonymous publication of her case history.

After the completion of the experimental neurotherapy program, 18 months after its onset, in the third examination (see: Table I) , she improved her language skills, and her profile was similar to the norm. She only presented word finding problem, but only for very rare objects. She returned to social life.

DISCUSSION

This is one of the first studies to demonstrate the application of ERP methodology for constructing protocols of TMS combined with aphasia therapy and for monitoring the results of the treatment. The methodology has been described in detail in (Pachalska et al., 2014; Kropotov, 2016). According to this methodology the local stroke results in the hyper-sensitivity of neurons located near the area of damage due to the disruption of local inhibition and to an increase in excitatory inputs because of sprouting. This is a compensatory mechanism that the brain develops for curing damage. Consequently, any therapy must be directed to helping the brain to restore the lost function. We suggested the application of a high frequency (activating) TMS to the area of hyper-sensitivity – around T6 in the 10/20 international system of electrodes' placement.

The experimental neurotherapy program (TMS combined with comprehensive aphasia therapy) gave very impressive results:

- the success of treatment is objectively manifested in a significant decrease in neuronal excitability over T6 electrodes (see Fig. 1);
- the improvement in cognitive control functions (see Table 2);
- the improvement in language functions (language profile was similar to the norm, see Table 1);
- the functional improvement (the patient returned to social life).

How can we explain the symptoms presented by our patient?

First of all, we should consider the diagnosis: was it crossed Wernicke's aphasia or crossed transcortical sensory aphasia? It has to be emphasized that Wernicke's aphasia patients can have problems not only at the level of the language sounds (acoustic-agnosic aphasia) or the memory of words (acoustic amnesic aphasia), but also at the level of the associations between words with specific meanings (Robson et al. 2012) (so-called amnesic or nominal or extrasylvian sensory aphasia, associated with damage in the left BA37 and left BA39).

It is important to note that so called extrasylvian (or transcortical) sensory aphasia can be considered as another subtype of Wernicke's aphasia; indeed, many authors interpret extrasylvian (or transcortical) sensory aphasia in this way (e.g., Brown 1981; Lecours et al., 1981). The lesion is, however, in the left hemisphere, and in our patient the lesion is in the right hemisphere, and it was associated with crossed extrasylvian (or transcortical) sensory aphasia which was associated with damage in the right BA39 only. It should be pointed out that this is a new finding (right BA39). Our patient did not meet fully the above mentioned criteria, therefore we consider a possible TSA diagnosis (see also Lecours et al 1981; Castro-Caldas & Confraria 1984).

According to Damasio (1991) TSA is associated with lesions involving the temporal-occipital area (BA37), the angular gyrus (BA39) or the white matter underlying these regions, but sparing the primary auditory cortex (BA41 and 42), and BA22 (see also Berthier, 1999). Lesions in BA37 result in amnesic (or anomic or nominal) aphasia (or the first subtype of transcortical sensory aphasia) whereas lesions in BA39 result in semantic aphasia or semantic anomia (or a second subtype of transcortical sensory aphasia).

Our patient, as was suggested by Damasio (1991), had an extensive posterior lesion involving the temporal-occipital area (BA37), the angular gyrus (BA39), and additionally in the area BA6 and the anterior part of the fusiform gyrus. The lesion involved the area BA 22, however the area BA 41/42 and the lower temporal gyrus were not damaged.

Luria (1976) suggested that when the lesions are restricted to BA 37 or BA 39, specific and well described language impairments are observed manifested as TSA (see also Ardilla 2014). With more extended lesions, additional clinical manifestations, such as jargon, can be found. Our patient, despite more extended lesions, did not present jargon, but this clinical manifestations, according to Kertesz (1979) this might only be observed in the acute stage of the brain pathology, and progressively disappears (see also Ishizaki et al 2012).

It is possible, that in the first period after the stroke she presented mixed crossed transcortical sensory aphasia, and during the process of rehabilitation the profile of aphasia might undergo changes, as it was suggested by Kertesz (1988) or even restored to mild anomia, as it is suggested by Dronkers and Larsen (2001:29). Therefore we might observe different symptoms which do not belong to the type of aphasia seen before, or during the rehabilitation. There is

only one objection: our patient was treated according to a standard aphasia therapy protocol (see: Basso et al. 1985; Mateer 1999; Pachalska 2011) during the course of a single year, and we did not observe any improvement in her language skills, while aphasia was in a chronic state. Therefore, we established the following diagnosis: chronic transcortical sensory aphasia.

What are the explanations of the therapeutic success?

A recent study that combined this type of intervention and pre-post fMRI provided the first evidence of the beneficial effects of TMS that underlie the restoration of the left hemisphere activity in some patients (Pascual-Leone et al 2000; Martin et al. 2009a,b; Meinzer et al. 2011). The results of our study showed that this could be working in another direction: the beneficial effects of TMS may underlie the restoration of right hemisphere activity. In the case of our patient TMS provided the possibility to create new connections in the brain, which resulted in better attention and memory, consequently in better naming (Naeser et al 2005 a, b) and understanding (Pachalska 2011). The speed of speech returned to the norm and consequently a restoration in normal speech occurred. This process could be explained by the microgenetic theory of symptom formation based on the principles of learning and neuroplasticity (Brown and Pachalska 2003; Kropotov 2016).

CONCLUSIONS

Before the TMS treatment combined with aphasia therapy the temporal area (T6) generates a strong P2 wave in response to visual stimulus indicating a hyper-sensitivity of the neurons located at the temporal areas of the right hemisphere. The TMS sessions reduced this hyper-sensitivity substantially. Neurotherapy might be helpful in the reduction of language disturbances after the stroke. ERPs in a GO/NOGO task can be used in the assessment of functional brain changes and during treatment in those patients with crossed aphasia after the stroke.

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