SUMMARY

The goal of the study was twofold: 1) to evaluate the QEEG/ERPs indexes of functional brain impairment in a TBI patient diagnosed with chronic lost cognitive control and lost self caused by post traumatic, and here increasing over time, delusional misidentification syndrome concluded with Cotard syndrome in the blooming stage, with nihilistic delusions concerning the body and existence, and the delusion of being dead, and 2) to explore the mind of a patient whose identity has been disengaged, and who experiences the loss of his self and relations with his immediate surroundings with all the tragic consequences that entails.

I herein present a 52-year-old patient, who – after a serious head injury due to a car accident 20 years ago, which resulted in focal injuries in the frontal and temporal areas of the right hemisphere – developed Cotard syndrome. After arousal from a 63-day coma and 98 days of post-traumatic amnesia, he manifested: (1) the loss of autobiographical memory, (2) a lost self, (3) forgotten family ties (including his lover).

The study revealed that the patient’s cognitive control system is completely destroyed: no cognitive components have been found. Recall from memory has been completely disturbed (a low amplitude of N170). The two hemispheres work incoherently with the right hemisphere revealing a serious delay in memory recall.

Key words: memory, working autobiographic memory, executive dysfunction, delusional misidentification syndromes, Capgras syndrome; Fregoli syndrome, Cotard syndrome, P300; N170
INTRODUCTION

Cotard’s delusion, one of the forms of delusional misidentification syndromes (DMS)\(^1\), is a rare disease, especially after TBI. In this syndrome the affected person holds the delusional belief that they are already dead, do not exist, are putrefying or have lost their blood and internal organs (Berrios & Luque 1995a, b; Berrios & Luque 1999; Mendez & Ramírez-Bermúdez 2011).

New neurotechnologies indicate that DMS, and therefore Cotard’s syndrome, is associated with cognitive deficits, especially disturbances in working memory (WM), this being observed in a significant reduction in the P300 amplitude of event-related potentials (ERPs) in the right frontal region (Pąchalska, Kaczmarek, Kropotov 2014; Papageorgiou, Ventouras, Lykoura et al. 2003) of 9 patients examined with DMS and 11 healthy individuals matched in terms of sex, age and education. The subjects performed tasks from the computer version of the Wechsler Memory Test Battery digits. Auditory event-related potentials measured before testing showed a significant reduction in the P300 amplitude in the right frontal region in patients with DMS when compared to healthy subjects. The delay of P300 amplitude in the medial region of the brain was significantly prolonged in 90% of respondents from the group of patients with DMS. In addition, the processing of data in WM by patients with DMS was significantly lower than in the control group. These results support the hypothesis that in patients with DMS, the right frontal region, which mediates automated processes with the distribution of attention resources, including inter-hemispheric junction circuits, is not functioning properly due to damage to the gray matter of the brain cortex itself.

Statistical analysis of a hundred-patient cohort indicates that the denial of self-existence is a symptom present in 69% of the cases of Cotard’s syndrome; yet, paradoxically, 55% of the patients present delusions of immortality (Debruyne et al. 2011; Yarnada, Katsuragi & Fujii 2007).

A patient afflicted with this syndrome usually denies their existence, the existence of a certain body part, or the existence of a portion of the body (Pąchalska, 2019). Cotard’s syndrome usually develops into three stages:

- **germination stage** – the symptoms of psychotic depression and of hypochondria;
- **blooming stage** – the full development of the syndrome and the delusions of negation;
- **chronic stage** – continued, severe delusions along with chronic psychiatric depression.

There are no diagnostic criteria for Cotard’s syndrome within the Diagnostic and Statistical Manual of Mental Disorders, Fifth Edition (DSM-5), Cotard’s delusion might be placed under the category of somatic delusions, those that involve bodily functions or sensations. Identification of the syndrome relies heavily on clinical interpretation. However, in my opinion, Cotard’s delusion should not be con-

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\(^1\) DMS, an umbrella term for syndromes of intermetamorphosis, Fregoli, Capgras and Cotard syndromes
fused with Delusional Disorders as defined by the DMS-5, which involve a different spectrum of symptoms, ones that are less severe and have a less detrimental effect on functioning itself.

The underlying neurophysiology and psychopathology of Cotard’s syndrome might be related to problems of delusional misidentification. Neurologically, post-traumatic Cotard’s delusion (with nihilistic delusions concerned the body and existence, and the delusion of being dead), with chronic loss of the self, and later the negation of the self, is thought to be related to Capgras delusion (people replaced by impostors); each type of delusion is thought to result from a neural misfiring in the fusiform face of the brain (which is connected with recognizing faces) and in the amygdala (which provides associations of emotions to a recognized face) (Pąchalska, MacQueen, Brown 2012; Kropotov 2009; 2016).

It should be noted that in the world subject literature, there are only two articles presenting a TBI patient with Cotard’s syndrome at the blooming stage, in which the full development of the syndrome and the delusions of negation occur (Young, Robertson, Hellawell 1992; Pąchalska 2019).

The goal of the study was twofold: 1) to evaluate the QEEG/ERPs indexes of functional brain impairment in a TBI patient diagnosed with chronic loss of the self and post traumatic, and here increasing over time, delusional misidentification syndrome concluded with Cotard’s syndrome in the blooming stage, with nihilistic delusions concerning the body and existence, and the delusion of being dead, and 2) to explore the mind of a patient whose identity has been disengaged, and who has experienced the loss of his self and relations with his immediate surroundings with all the tragic consequences that entails.

**CASE STUDY**

I herein present a 52-year-old patient, who – after a serious head injury due to a car accident 20 years ago which resulted in focal injuries in the frontal and temporal areas of the right hemisphere – developed Cotard’s syndrome with nihilistic delusions concerning the body and existence, and the delusion of being dead. The trauma has caused serious and overwhelming changes in his entire life. Prior to the accident, he was a gynaecologist with a second-degree specialization, the head of the OB-GYN department at a provincial hospital in southern Poland. He was married at the time of injury. The marriage continued and they currently have 3 children aged between 14 and 29 years old. The oldest daughter graduated from the Academy of Fine Arts and is pursuing a career as a painter. At the time of his injury, he had a lover who was believed to be the love of his life both by her and by his family.

20 years ago, while driving his car he drove into a tree to avoid a head-on collision. The accident resulted in multi-organ injuries and a very severe TBI. After the accident, he was conscious for a short period of time, however his memory concerning the event was not satisfactory. An intracranial hematoma developed immediately after hospital admission, and the patient lost consciousness. His Glasgow Coma Scale score was the lowest possible (3 points). After arousal
from a 63-day coma and 98 days of post-traumatic amnesia, he manifested: (1) the loss of autobiographical memory, (2) a lost self identity, (3) forgotten family ties and those towards his lover.

A neuroimaging examination four months after the injury revealed malacia in the right lower frontal gyrus and in the right temporal gyrus (long arrow); a hypointensive gliotic lesion, right lower frontal gyrus (short arrow); oval porencephaly and atrophy of the right gyrus rectus (short arrow) and hyperintensive small gliotic area in the right temporal gyrus (long arrow) (see: Fig. 1).

In neuropsychological rehabilitation he manifested left side hemiparesis, anosognosia (unawareness of his impairments), mild post injury aphasia, sleep problems including narcolepsy, and nightmares.

**Ophtalmological testing**

The visual field was normal for both eyes – carried out using a 2010 Carl Zeiss Meditec Humphrey Visual Field Analyzer. Optical coherence tomography (Optovue RTVue OCT) did not show any significant changes in the morphology of the macula and nerve fibre layer (Fig. 2, 3 and 4).

**Neuropsychological testing: 3 months after injury**

In neuropsychological testing we observed mild naming problems, executive dysfunctions, inability to perform everyday activities [ADL], cognitive problems [memory, selective attention, visuospatial disorientation] (see Table 1).

He also experienced the loss of autobiographical memory (confused close relatives), progressive deterioration in personal identity (did not recognize himself, did not remember his name, did not recognize his family).
Fig. 2. Maps of nerve fibres and GCC complexes
Source: clinical material of M. Pąchalska

Fig. 3. Maps of the macula morphology in the right eye
Source: clinical material of M. Pąchalska
The accident brought dramatic changes in his life style. He no longer smokes and denies that he ever did smoke or drink. He refuted that the undrunk bottle of brandy had ever been in his possession. Prior to his accident he was a vegetarian, a smoker, a moderate drinker, and a known wit, who enjoyed social intercourse and parties. Following the injury, he had no recall of this lifestyle and personal history. Following his injury, he initially misidentified objects. Later, however, he was able to name objects relevant to his past, e.g. cigarettes, a bottle of cognac (even the brand) but he did not recognize these as being from his own experiences or events connected with him. Afterwards he did not recognize his own image in a mirror as exemplified in the following conversation with a therapist:

T: Who is that? [a physiotherapist named Jacek, holding him up in front of a full-length mirror]

Who do you see there?

P: I don’t know. Oh my God! That monster is staring at me [shouting].

T: And who else do you see in the mirror?

P: I don’t know, but perhaps Jacek, I think you said so, isn’t that right?

Soon he developed bursts of aggressiveness and started to display Capgras syndrome. An extract from an interview with the patient (in the presence of his parents, his wife and his oldest daughter, who was 19 at the time).
Tab. 1. Symptoms noted in the patient during longitudinal observation (for 20 years since the brain injury.

<table>
<thead>
<tr>
<th>Time after the stroke</th>
<th>Symptom</th>
<th>Utterance/behavior</th>
<th>Interpretation</th>
<th>References</th>
</tr>
</thead>
<tbody>
<tr>
<td>&gt; 6 months</td>
<td>Mistaken identification of objects</td>
<td>T: What objects are here on the table? P: “Malboro” cigarettes, stethoscope, doctor’s diploma. I: Are these your belongings? P: [shouts] Help! Oh my God! I have never smoked and I do not smoke. T: And doctor’s diploma? P: [He looks at the picture and cries] Help! An old chap in the photo. I am 8 years old.</td>
<td>Belief that his own objects do not belong to him.</td>
<td>(see also Pąchalska, MacQueen, Kaczmarek et al., 2011)</td>
</tr>
<tr>
<td>&gt; 12 months</td>
<td>Mistaken identification of himself (mirror symptom)</td>
<td>T: Who is it? Who do you see in the mirror? P: [cries] Help! Oh God! This monster is looking at me [spits at the person in the mirror]. T: And who else do you see in the mirror? P: I don’t know, may be Jacek, you have told so, haven’t you?</td>
<td>Belief that the image in the mirror is another person.</td>
<td>(see also Coltheart 2011)</td>
</tr>
<tr>
<td>&gt; 15 months</td>
<td>Confabulations about himself (disintegration of vocational identity)</td>
<td>T: How old are you? P: I am 19 years old. T: Your identity card shows you are 45. P: [shouting] No! The government has not only changed the money and I cannot recognize them but they also changed the calendar to avoid paying a pension...they added 30 years to the calendar and now I am 45 years old while in fact I am 19. And that move of the government has disrupted my life. T: Are you a doctor? P: [shouts] Help! Oh God! I am a car mechanic. There is my repair shop [pointing to the administration building of the Rehabilitation Center].</td>
<td>Belief that his age is different from the real one and that his profession is also different.</td>
<td>(see Pąchalska, MacQueen, Kaczmarek et al., 2011)</td>
</tr>
<tr>
<td>&gt; 24 months</td>
<td>Capgras syndrome</td>
<td>T: You are happy that your family has visited you, aren’t you? P: [shouts] Help! Me? Not at all! I have no family. I do not know those people. My family died in the car accident. T: And... P: I do not know those people. These are doubles... doubles of all my family. T: Is this women you wife? [in the presence of the wife] P: [shouts] This is not my wife... I have no wife. I am afraid of this Lady. Wife: What are you saying? Don’t you remember me? We have three children. Our family love you! P: [shouts] Help! I do not have any family – they all died the car accident. I have no children. I am completely alone! T: But she has also your beloved dog with her. P: [shouts] Help! That dustman in a fur? A stray? I haven’t got any dog [kicks the dog which fawns all over him].</td>
<td>Delusional belief that (usually) near relative or spouse has been replaced by an identical double.</td>
<td>(see also Ellis, Luauté and Retterstøl, 1994; Bilikiewicz and Strzyżewski, 1992)</td>
</tr>
</tbody>
</table>
Tab. 1. Symptoms noted in the patient during longitudinal observation (for 20 years since the brain injury.

| > 36 months | Fregoli syndrome | T: Godd morning. P: [shouts] Help! You are my girl-friend. You have dressed up in a white apron! You follow me! T: I am working here in the Rehabilitation Center. P: [shouts] Help! Let me out of the lift. You dress up and you want to caught me away to your house. People! Help! | Delusional belief that people he meets are in fact the same person dressed up to disguise him. | Pąchalska, 2007a |
| > 48 months | Intermetamor fosis | | |
| > 10 years | Cotard syndrome (a stage of full bloom of symptoms) | T: Are you happy that your wife has visited you? P: [shouts] What rubbish! I am probably dead. Walking corps. I have lost all my blood in the car crash. I am rottling from inside. T: But I hear you voice. P: [shouts] Its rubbish! | Delusional belief that he is dead (metaphoricall y and really) he has lost his blood and his body is rottling. | (por. tež Pużyński 1993) |
| > 18 years | Cotard syndrome (chronic stage) | T: Hello. P: I am not here any longer. I do not live any longer. My family is also dead. T: But I am talking with you. P: [quietly] You are wrong. I am dead. Don’t you see that it is a gravelyard? | Delusional belief that he is not alive and that his family is also dead. | (see also Berrios and Luque, 1995a and b; 1999). |
| > 20 years | Complete disintegration of Self-system | The patient does not start any action including talking. He does not meet his need by himself, and does not perform any everyday actions by himself. Yet he fulfills most commands especially with the help of a caregiver. Only involuntary functions are preserved (scratching, stretching, etc.) | Consciousnes s disorders. Breaking the connectivity of the brain with the body. He does not function as an organism. | see Goldstein, 1998; Pąchalska, 2007a) |

Source: Clinical material of M. Pąchalska

T: You are pleased that you’ve been visited by your family, aren’t you?
P: Me? Of course not! I don’t have a family. I don’t know these people. My family was all killed in an accident.
T: And…
P: I don’t know these people. They are doubles… doubles of my entire family or I don’t know [shouting]
T: Is this woman your wife Teresa? [in the presence of his wife, who is visiting]
P.A. [shouting] That is not my wife… That is not my wife. I don’t have a wife. That woman’s a doctor and I’m afraid of her.
Wife: What are you saying? Don’t you remember me? We’ve had three children together. Our family loves you!
P.A. ‘.… I don’t have a family – they were all killed in an accident. I don’t have any children. I’ve got no one and I love no one! I’m completely alone!’
When the patient’s lover was visiting him he also did not recognize her, and shouted at her not to kiss him, as it is not permissible for a doctor to establish such a close bond with a patient. This suggested that he recognized her as a former patient, but not as his lover. Two hours after her visit a therapist asked:

So, you were visited by your girlfriend?
P: That hag is supposed to be my lover?
T: She is a beautiful woman, isn’t she?
P: Perhaps I could consider the woman beautiful, yes…I would regard her as beautiful, she is about 40, isn’t she? My girlfriend cannot be so old!!! Besides, I have never had a lover.

It is nearly impossible to establish, in his present state, to what extent this was a repression, to what extent – amnesia, to what merely confusion. Yet, about half an hour after his lover had left the room, the patient asked the therapist:

Aren’t you…hm…my fiancée or something…she must be somewhere, but I (?) do not know where and nobody can find her.

The treatment was based on a comprehensive model of rehabilitation, including neuropsychological rehabilitation (Pąchalska 2007).

**Neuropsychological examination: 12 months follow up**

Neuropsychological testing revealed the following symptoms: minor naming difficulties, minor problems with the production of syntactically correct sentences, working memory problems and severe learning problems (PA did not remember anything for longer than a few minutes at most), especially in delayed memory tasks, severe problems of autobiographical memory, persistent disturbances of executive functions, e.g. the patient used to soap himself and to enter the room naked in the presence of children, features of frontal lobe syndrome, persistent identity impairment (he did not recognize himself though he knew his name). It was found using the Test of Word List Memory (Wechsler Memory Scale-III, WMS-III) in the 1st examination (3 months after the accident) and in the 2nd examination (12 months follow up) that the patient dynamics of memory had deteriorated, especially after distraction, in trial V, in which he remembered one word only, and after 30 minutes, in trial VI, he could not remember even a single word (Fig. 5).

He possessed murky recollections of childhood up to the age of eight – during this period he dreamed of becoming a car mechanic. He vaguely remembered his first year at school, but from the age of eight, as he claimed, everything was foggy and empty…everything had to be searched for. Asked to draw a picture he represented himself as a ladybird (see: Fig. 6)

Beside consciousness impairment, a high level of anxiety, behavioural disturbances resulting in an inability to perform daily duties, paradoxical behaviours, with deficits in conforming to social norms were noted. He used to laugh aloud and yell in inappropriate moments, e.g. during Midnight Mass. In addition, delusions appeared as in the following complaints: the government has not only changed the money and I cannot recognize it, but also the calendar to avoid paying the life annuity…they added 30 years to the established calendar and as
a result I am 48 years old, but truly I am 18 years old. They want to get rid of me. I’m scared.

During this period, in group therapy, he met another patient who had been pregnant and had lost the child. He advised her professionally and told her what drugs she could take for the depression that had developed following the loss of the child. The advice given was highly professional in nature. Asked to draw a picture depicting the most important memories from his childhood, he drew four pregnant women and entitled the drawing: “Maternity” (Fig. 7). He also stated: “...they could have been my patients. But I can’t really recall whether I had such patients. When I think about it I see a black hole, ...such a void.”

Fig. 5. Test of Word List Memory (Wechsler Memory Scale-III, WMS-III).
Source: own research

Fig. 6. The patient’s drawing representing himself as a ladybird, which is always searching for something
Source: own research
Lost self

The patient lost his self and borrowed the identity of others. Below we give the characteristic symptoms of misidentification and their progress. The initial symptoms of misidentification appeared soon after the accident. He identified the clinic as a garage and he believed to be a mechanic. He sometimes accepted his former identity as suggested by the therapist but in general he would protest, shout and present himself as a car mechanic. With time he tended to adopt the identity of any person he happened to meet as in the following few examples:

- A patient Jurek, aged 19, moving in a wheelchair after a knee operation. After seeing Jurek, he shouted that he couldn’t walk because he had had a knee operation and would have to use a wheelchair.
- An art therapist Zbyszek, aged 29. He took brushes from the art therapist and did not want to give them back. He insisted that he had to paint a picture in the open because he had to earn a living.
- A department chaplain, aged 27. When the priest was celebrating Communion on the ward, PA took his Bible, which had been left for a moment on the table, and did not want to give back. He shouted that they wanted to steal the Bible from him and that he would have nothing to pray from.

Of importance is the fact that after a few minutes (sometimes an hour) PA forgot about his adopted identity and again did not really know who he was.

The remaining cognitive functions as well as the stocks of acquired knowledge (including medical) that do not concern PA personally remained largely intact. He was able to correctly describe in detail gynecological operations but he was extremely surprised that he could do so, saying: “I had no idea I could do this”.

Fig. 7. The patient’s drawing entitled: “Maternity”.
Source: own research
Results from the Test of Remembering of Autobiographic Events in the 1st examination (3 months after the accident) and in the 2nd examination (12 months follow up) showed a deterioration in the percentage of recalled events. It was also noted that after the lapse of a certain period of time the intensity of the affect connected with PA’s recollection of a given event diminishes in comparison to the affect that accompanied the event itself. The magnitude of the fall of negative affect exceeds the dimensions of the positive affect. Considering the automatic scaling on the graph, the differences at the time of storage of 45 seconds appear to be slight, though these differences – like the others – are still statistically significant.

After one minute the patient forgets equally a negative or a positive autobiographic event (e.g. a match burn on a finger; beard being shaved by a nurse or even drinking tea).

As the years went by, the disorders grew and 10 years after the accident it was noted that the patient did not care about personal hygiene at all, he did not wash himself, and he dealt with his physiological needs by sitting on the bed of his young daughter. He experienced a generalized sense of unreality in the world and claimed that he was dead because he had lost all his blood and was empty inside. He shouted that he was afraid because the doubles of his relatives were walking around. The neuropsychiatrist diagnosed him with the co-occurrence of Cotard’s syndrome in the blooming stage, with nihilistic delusions concerned the body and existence, and the delusion of being dead.

At present, 20 years after the accident, Cotard’s syndrome has intensified. The patient not only claims that he is dead, but that his whole family are also dead because he died in an accident. The patient became completely dependent on others. He stopped feeling anger, joy, excitement as well as hunger, thirst, cold and warmth. He must be fed, though he can still do it himself. These bizarre
behaviours and delusions, the total disintegration of the self system (loss of self) and the associated loss of individual and social identity meant that the family no longer wanted to deal with it. The wife, who stated that the patient had a complete disappearance of both psychological and bodily sensations and that it was unbearable for her and the children, filed for divorce while the patient was in a social welfare home.

**EEG recording and artefact 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 2009; Kropotov, Ponomarev, Hollup 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 artefacts 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 (Kropotov 2016).

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 µV for non-filtered EEG; (2). 50 µV for slow waves in 0-1 Hz band; and (3). 35 µV for fast waves filtered in the band 20-35 Hz.

**ERPs assessment**

For the assessment indexes of brain functioning (neuromarkers) a variant of the cued GO/NOGO task was used (Kropotov & Ponomarev, 2009; Kropotov, Ponomarev, Hollup et al. 2011). In this task, images of animal (a), plant (p), and human (h) categories served as relevant stimuli. The trials consisted of presenting 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 novelty sound. 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. He 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.
RESULTS

Parameters of performance in the cued GO/NOGO task in comparison to the average parameters of a group of 100 healthy subjects from the Human Brain Index (HBI) database are presented in Table 2. It should be stressed here that to keep the performance at an optimal level the patient was periodically reminded of the goal of the task – pressing a button to a pair of two animals. This observation shows that the patient has a very short WM – a neuromarker of the frontal lobe damage.

Figure 9 demonstrates comparison of the patient’s neuromarker of cognitive control with the corresponding neuromarker of healthy subjects (norm). The P300 NOGO wave serves as the neuromarker in this case. It was found that the patient’s brain does not generate a prominent P300 NOGO despite the fact that the latency and variation of response in this patient does not differ from the reference.

Table 2. Behavioural parameters in the cued GO/NOGO task: comparison to a group of healthy subjects of the corresponding age from the HBI database (N=100)

<table>
<thead>
<tr>
<th>Subject</th>
<th>Omission errors</th>
<th>Commission errors</th>
<th>Reaction time (RT)</th>
<th>RT variance</th>
</tr>
</thead>
<tbody>
<tr>
<td>Patient</td>
<td>7%</td>
<td>5%</td>
<td>291ms</td>
<td>8.2 ms</td>
</tr>
<tr>
<td>Norm</td>
<td>3.6%</td>
<td>1.2%</td>
<td>398ms</td>
<td>7.4 ms</td>
</tr>
<tr>
<td>P value</td>
<td>0.74</td>
<td>0.07</td>
<td>0.28</td>
<td>0.80</td>
</tr>
</tbody>
</table>

Fig. 9. Reduction of P300 NOGO of the patient’s ERPs in the cued GO/NOGO task
(a) NOGO ERPs in response to the second stimulus in the trials for the patient (green line), for a group averaged (red line), and the difference wave (patient – healthy controls, blue line). Below the blue line the blue box depicts the time window in which the patient’s ERP differs from the grand average at p<0.05).
(b) Map at the difference wave at time indicated by arrow at (a).
Source: own research
Figure 10 illustrates the impairment of executive components in the patient. Besides the inability to recall from the episodic memory the subject suffers from executive dysfunction. All executive components are impaired.

Processing in the right inferior temporal cortex was significantly delayed. Processing in the parietal cortex was significantly faster than in the norms. This dysbalance in the timing of processing in the dorsal and right ventral visual streams created an inability to correctly extract information from the long term episodic memory.

**Event Related Potentials (ERPs)**

ERPs results registered in response to visual stimuli in the GO / NOGO task are illustrated in Fig. 11. In 100 healthy persons, the N170 component of ERPs taken from Human Brain Index, HBI (Kropotov 2009; 2016) appears symmetrically in the left and right parts of the brain (Fig. 11, on the left), however the patient’s ERP results show that there is a very low amplitude of the N170 component of ERPs and that this appears non-symmetrically in the left and right parts of the brain (Fig. 11, on the right).

The two hemispheres work incoherently, the right hemisphere has serious delay in recalling from memory. No cognitive components are found. It means that his memory recall has been completely disturbed (low amplitude of N170) and the cognitive control system is completely destroyed.
DISCUSSION

It is known from the subject literature that the right and left hemispheres process data in a different way. The left hemisphere is associated with the isolation of objects (e.g. faces), while the right adds the emotional meaning of the face (Pąchalska, Kaczmarek & Bednarek 2020). In the examined patient, compared to the results from the HBI normative database obtained in 100 people, an asymmetrical N170 component appeared in the right hemisphere, while in the left hemisphere this component was close to normal. This means that the patient has asynchronous right and left hemisphere workings. If we consider that the N170 component is an indicator of data recovery from memory, it can be assumed that in this patient the emotional significance is extracted from memory longer than extracting the face itself. Therefore, the face of a loved one (e.g. his wife, daughters, son), and even his own face in the mirror, seems strange to the patient, because he is completely devoid of emotional context. This fact explains why the patient does not recognize himself (a mirror symptom) and why he considers his loved ones to be doubles nor does he feel any emotions. Therefore, he developed Cotard’s syndrome in the blooming stage, in which the full development of the syndrome and the delusions of negation occur.

How to explain forgotten family ties?

Disturbances of working memory cannot totally explain the loss of the self and forgotten family ties. Prior to the accident, PA’s family had relatively normal family ties. The bond between the patient and his wife was strained by the fact that he had a lover. After the accident, the family gradually underwent collapse, so that at the present moment all family bonds and ties are seriously shaken. The patient does not remember that he loves anyone, and his family now no longer loves him. As the cortical-subcortical atrophy of the anterior hemisphere
of the right brain and the associated destabilization of neural connections with
the cingulated cortex has deteriorated, the patient loses emotional, cognitive and
motor control, as a result of which his functioning significantly deteriorates (see
also Damasio 1999).

**The transition from misidentification through Capgras to auto-Fregoli
and Cotard’s delusion**

Current research has shown that lesions in the right temporal lobe and the
fusiform gyrus may contribute to DMSs. The MRIs of patients exemplifying DMS
symptoms have shown parahippocampal and hippocampal damage in the ante-
rior fusiform gyrus, as well as the middle and inferior of the right temporal gyri.
The inferior and medial of the right temporal gyri are the storage locations for
long-term memory in retrieving information on visual recognition, specifically of
faces; thus, damage to these intricate connections could be one of the leading
factors in face misidentification disorders (Hudson and Grace 2000; Lykouras,
Typaldou, Gournellis et al. 2002; Stewart 2008).

Coexistence in DMSs the syndrome of subjective doubles, the syndrome of in-
termetamorphosis, Fregoli delusion, Capgras syndrome and Cotard’s syndrome
are enhanced when coupled with other mental disorders such as schizophrenia,
bipolar disorder and other mood disorders. Depersonalization and dereali-
zation symptoms are usually manifested in patients exhibiting two misidentifica-
tion delusions. However, such symptoms have been observed to cease once the co-
existing DMSs are fully developed (Lykouras, Typaldou, Gournellis et al. 2002).

A brain-damaged patient may initially be unable to identify familiar persons, and
may also not recognize themselves in a mirror. Over time, Capgras syndrome may
appear, and the patients believe that highly familiar persons have been replaced
by doubles that only resemble the real relatives. He also presented a kind of self-
oriented Capgras, in which the patient says or implies that he himself is a double.
Capgras syndrome may evolve into Fregoli syndrome, in which the patient is
convinced that a complete stranger is someone he knows very well. At the next
stage Fregoli may give way to “auto-Fregoli” symptoms, and the patient appro-
priates the identity of a complete stranger (see also: Pąchalska, MacQueen &
Brown 2012). Pąchalska, MacQueen & Brown (2012) points out that Capgras
and Fregoli syndromes lie on a single continuum of deterioration, which begins
with Capgras, evolves into Fregoli, and may end as auto-Fregoli. In the pre-
presented patient the “auto-Fregoli” gave way to Cotard’s delusion, and in effect the
patient began to maintain that he was dead. Patients with Capgras syndrome
project changes in themselves into others, while in Cotard’s delusion, the patient
attributes the change to themselves exaggerating its extent (Fig. 12).

The final stage in the progression described here, then, is a complete loss of
identity, as is most commonly seen in the advanced stages of neurodegenerative
diseases, e.g. Alzheimer’s disease or Semantic Dementia (Mendez & Ramírez-
Bermúdez 2011).
A similar mechanism worked for our patient. Generalized brain atrophy and the associated destabilization of many neuronal connections led to the loss of meaning of words and the breakdown of the minimal (working) and longitudinal (autobiographical) self, which caused the patient to drift in the unreal world and think that he was dead and experienced various illusions from bodies that occupied his thoughts.

It is worth recalling that the Integrated self system included the individual (objective and subjective) and social (collective and cultural) self (Pachalska 2019). This concept, however, should include the minimal (working) and longitudinal (autobiographical) self, which is the basis for the formation of the self system. Therefore, I have developed a modified model of the self system, which requires the nesting of the minimal (working) and longitudinal (autobiographical) self and a change in the understanding of the concepts of individual and social self in terms of the thought process (cf. Fig. 13). Therefore:

1. The individual self includes:

A) The objective self, understood as the organism, i.e., in Goldstein's (1995) approach, the body together with its states and processes occurring in it. The subject self has consciousness, but it lacks self-awareness and meta-consciousness (awareness of mental operations on its own subject). The subject does not express their own thoughts but acts according to ready-made schemes: they are not the author of themselves. As soon as you realize the existence of the outside world, your subject self also becomes

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Fig. 12. The transition from misidentification through Capgras to auto-Fregoli and Cotard's delusion (psychological death).
Source: Pąchalska, MacQueen, Brown (2012), modified.
the object of perception. This process enables the subjective self to be formed;

B) The subjective (cognitive) self, having consciousness, self-awareness and meta-consciousness, enabling one to know oneself and act in accordance with one’s own needs and values as well as the requirements of the environment. He/she has a sense of separateness, autonomy, insight (introspection), the possibility of self-assessment and self-control and creativity (see Pachalska 2007). The subjective self conditions the appearance of individual identity.

2. The social self, includes:
   A) the relational self, understood as an image and description of the You - You (interactions), from an individual and social perspective taking into account relationships with other important people and social groups around which social identity develops.
B) the cultural self, understood as an image and description of the We – We from an individual and social perspective including nesting in the culture or subculture of a given social group around which cultural identity develops.

The microgenetic approach to the self-system takes into account the concept of the nesting of the minimal (working) and longitudinal (autobiographical) self in the individual and social self in the processual approach, and creates the basis for the development of the self system. It also allows for a better explanation of the disruption or disintegration of this system in persons with various kinds of brain damage (see also Prigatano 2009).

The consequence of memory loss, especially autobiographic memory, as happened in our patient, is not merely the loss of a certain kind or amount of information, but the disintegration of the self. The important point is the sequence of qualitatively different stages by which one syndrome becomes another. The symptoms may differ from the previous one but they reflect the same process and lead in the same direction. This direction is related to the development of the self in ontogeny. The disruption or decay of outer layers uncover the nature of the underlying layers. This is in accordance with the observations of Jackson and confirms the microgenetic theory of the unfolding of the self (Pačhalska, Kaczmarek & Bednarek 2020).

As was observed in our case, Cotard’s syndrome withdraws the afflicted person from other people due to the neglect of their personal hygiene and physical health. Loss of consciousness, self-awareness, self-reflection skills and actual interiorization of recognized values such as not embedding them in broadly understood culture or subculture is associated with the loss of meta-consciousness. Under these conditions, the Self system breaks down. The inability to cognitively control these disorders leads to the occurrence of illusions about one’s own existence and distorting the image of the world. This patient lives as if in a dream and therefore loses total control over their own life. That is why they are placed in a social care home by their family.

To sum up, the case described above exemplifies the gradual loss of the self related to the disturbances of memory, especially autobiographic memory. At the first stage of illness, he lost his social self, which resulted in the belief that his relatives are strangers. The next stage was the loss of the minimal self and as its consequence the patient “borrowed” the identities of those he had happened to meet. At the final stage, Cotard’s syndrome, he has lost all of his selves. Hence, since none of his selves, both minimal and longitudinal, exists any longer, he is living in a completely unreal, strange, and hence terrifying world. Every day he wakes up not knowing who he is and who the people that surround him are.

CONCLUSIONS

The study revealed that the patient’s cognitive control system is completely destroyed: no cognitive components are found in the QEEG/ERP results. Memory recall has been completely disturbed (low amplitude of N170). Moreover, the
work of the two hemispheres work incoherently, with the right hemisphere revealing serious delay in recalling from memory.

**REFERENCES**


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