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# The neuropsychological and neurophysiological profile of women with pseudoseizure

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**Abstract**

**Objective:** Our aim in this study was to compare the assessments of neuropsychological tests and the p50 neurophysiological test of patients with seizure diagnosed as conversion disorder and healthy control subjects, and to investigate the neurological status in conversion disorder with pseudoseizure.

**Methods:** A total of 22 female conversion disorder patients with convulsions diagnosed according to SCID-I/CV and 22 healthy women were included in the assessment. The participants were administered WMS-R, the cancellation test, and the Stroop test as neuropsychological tests and p50 was assessed as a neurophysiological test.

**Results:** The patient's results for the neuropsychological tests were found to be significantly low compared to the control group. The p50 sensory gating ratios of the patient group were statistically significantly lower than the controls. There was no significant correlation between the neuropsychological test scores and gating ratios of the patient and control groups.

**Conclusions:** This study is the first to check sensory gating in conversion disorder patients with pseudoseizure and its most important result is finding reduced p50 sensory gating in patients. Our results suggest that these patients have a neurological tendency to this disease due to functional neurophysiological features.



## 1. Introduction

Pseudoseizures are a subtype of conversion disorder (CD) that closely resembles epileptic seizures but is not accompanied by tongue biting, urinary incontinence, severe injury or unconsciousness [1]. Many psychodynamic views, neurobiological and genetic factors and sociocultural approaches have been used to explain the etiology of CD but it is generally thought to be a multi-factorial disorder [2]. Breuer and Freud defined pseudoneurological signs as the bodily representations of unconscious mental conflicts [3].

Although there are only a few studies on the role of biological factors in CD, some recent publications report a possible pathology in the cerebral functions of these patients [4–7]. These studies have mostly been carried out in the subtype of CD with motor signs and are associated with hemispheric dominance, event-related evoked potentials, structural and functional brain imaging and neuropsychological tests (NPT). A study by Yazıcı and Kostakoğlu [8] has reported decreased cerebral blood flow in the left parietal region in 1 patient and left temporal region in 4 patients with difficulty walking while Spence et al. [9] have reported decreased left dorsolateral frontal cortex efficiency in CD patients with signs of left hemiparesis. Another study in CD patients with sensory and motor loss has reported decreased regional cerebral blood flow in the thalamus, putamen and caudate nucleus contralateral to the involved part, and improved cerebral blood flow together with an improvement in the signs [10]. Labate et al. have postulated that the

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cerebellum, in addition to motor and premotor areas of the right hemisphere, also plays an important role in psychogenic seizures [11].

The CD model suggested by Ludwig indicates that the symptoms are related to an attention disorder due to increased corticofugal inhibition of afferent stimuli [12]. Study results till now have shown results parallel to this view. Neurophysiological studies have also emphasized the importance of the higher level cortical mechanisms in CD and revealed that frontal cortical and limbic activation related to emotional stress has an inhibitory effect on the basal ganglia–thalamocortical pathways and thus prevents the conscious sensory and motor process [4].

Studies using NPT that provide data on the location and type of disturbances reflected in the behavior in the cerebral field undertaken by scanning the cerebral fields that could possibly have structural damage [13] have supported the view that the main problem in CD is in the fields of memory, scanning stimuli and information processing and that these originate from corticofugal inhibition of afferent stimuli [4].

Some assumptions regarding information processing, which can be summarized as the perception and evaluation of the stimulus and giving the appropriate response, contribute to our view that CD is a problem with information processing. Sensory stimuli are processed in the brain via two-way processes from below to above vice versa. The lower level systems classify and define the stimuli received and transfer them to the upper level processing centers (bottom-up processing), while the prefrontal mechanisms try to control the incoming stimulus with activities such as getting ready for the received stimulus, prediction, attention and planning (top-down processing) [14,15].

Sensory gating plays quite an important role in this two-way process. Sensory gating is the first step in the pre-attention stage of information processing and enables the passage of only parts of the stimuli in the brain while filtering the unrelated stimuli [16]. P50 is thought to reflect the sensory gating mechanism and prevent excessive information loading. The deficit in p50 suppression reflects an impairment of the central inhibitory circuits that modulate cortical responses to sensory inputs [17]. Some studies show an important role of the prefrontal cortex on P50 suppression [18].

We tried to understand how the information processing worked in patients with pseudoseizures using sensory gating and NPT. We used the neuropsychological test Wechsler Memory Scale-Revised (WMS-R) to evaluate attention and memory and the Stroop Test (ST) and Cancellation Test (CT) to evaluate the attention. We wanted to test our assumption that the controlling and suppressive mechanism from above to below would be more effective in attention and sensory processes than mechanisms from below to above. We therefore planned to compare the NPT and p50 results of pseudoseizure patients with a healthy control group. Our aim was to contribute to the elucidation of CD neurobiology, obtain data that might hold clues for the cerebral region responsible for the development of the disorder, and measure how the above tests could be used for the diagnosis.

## 2. Methods

### 2.1. Subjects

A total of 24 patients who had presented at the Psychiatry Outpatients Department, had been diagnosed with CD with pseudoseizures according to the Structured Clinical Interview for DSM-IV-Clinical Version (SCID-I/CV) [19] and provided informed consent to participate in the study were included in our study. This study was approved by the local ethic committee. The patient group consisted of 22 females and 2 males but we did not include the data of the 2 male patients to prevent any complicating factors. The control group consisted of 22 healthy female volunteers who were matched with the patient group for age, gender and education. The inclusion criteria for the study for both the patient and the control groups were using the right hand, presenting for treatment for the first time, not having taken a psychotropic drug in the last two weeks, not having a history of any neurological or neurosurgical disorder, no childhood history of a sequel-causing disease and/or head trauma, no mental retardation or additional medical problem, no alcohol-substance abuse or usage disorder in the last year and no other psychiatric and/or personality disorder diagnosis according to the DSM-IV-TR [1].

### 2.2. Procedure

After administration of SCID-I/CV by an experienced investigator to the patients and healthy controls, the socio-demographic data form, Hamilton Depression Rating Scale (HDRS) [20], Hamilton Anxiety Rating Scale (HARS) [21] and the neuropsychological tests of WMS-R, CT, and ST were administered. Following the consecutive administration of all neuropsychological tests, the p50 test was administered at the neurology laboratory on the same day.

### 2.3. Neuropsychological tests

#### 2.3.1. Wechsler Memory Scale-Revised (WMS-R)

WMS-R, developed by Wechsler [22], is a psychometrically advanced measurement tool that evaluates memory in a comprehensive manner. The study form consists of personal and actual information, orientation, mental control, logical memory, forward number range, reverse number range, and visual recall subtests. The Turkish standardization studies of the WMS-R have been conducted [23].

#### 2.3.2. Stroop Test (ST)

The ST was originally developed by Stroop as an experimental task in 1935 [24]. The ‘Stroop effect’ is based on reading the names of colors printed differently from the color that they denote. There are various individually administered forms of ST. Turkish standardization studies of the Stroop Test have been conducted [23].

#### 2.3.3. Cancellation Test (CT)

This test was originally developed by Weintraub and Mesulam [25] to measure a sensory component of the parietal lobes related to perceptual errors, a motor component related to visual search and scanning, and a motivational component related to expectation and affect. Turkish standardization studies of the Cancellation Test have been conducted [23].

### 2.4. P50 Sensory Gating

The P50 component is the first positive wave appearing 45–75 ms after an auditory stimulus. The second P50 amplitude is smaller than the first with consecutive stimuli at 500 ms intervals in healthy individuals. The decreased P50 amplitude in normal individuals is believed to be through gating of information flow and the filtering of information thought to be unnecessary or irrelevant [26].

The P50 procedure was administered by a neurophysiology technician at the neurology lab in our study. The subjects were in the supine position during the P50 test administration. The auditory evoked potentials were recorded by using the auditory "oddball two-tone discrimination task". Silver disk scalp electrodes were placed according to the international 10–20 Electroencephalography Electrode Connection System after the scalp was cleaned. The active electrodes were placed on the Fz (frontal), Cz (vertex) and Pz (parietal) areas, the ground electrode on the Fpz (prefrontal) region and the reference electrodes to the two mastoid protuberances, and then connected with each other.

The P50 wave amplitude was measured with responses to two identical auditory clicks that were repetitive, of high intensity and of short duration. The P50 component latency and amplitude were measured separately for both clicks. The first component of the P50 wave (S1) was accepted as the positive wave creating a peak 40–80 ms after the first click. The second component of the P50 wave (S2) was considered as a positive wave 500±10 ms afterwards. The main wave was divided into four waves after recording and the S1 and S2 waves were extended. The S1 and S2 waves were then overlapped on the computer screen so that their configuration, latency and amplitude could be determined in a clearer way. The latency and amplitude of the positive peak (p) and following negative peak (n) were measured separately for the S1 and S2 waves. The P50 gating ratio (sensory gating ratio) was calculated with the $[1 - (S2 / S1)] \times 100$ formula.

### 2.5. Statistical analysis

The data obtained from our study were analysed with the "SPSS (Statistical Package for the Social Sciences) v. 16.0 for Windows" package software. The means of the two groups were compared with the t-test. Fisher's exact chi-square test was used to compare nonparametric data. The Mann–Whitney U test was used for data without normal distribution. Data regarding patient age, starting age of the disorder, and disease duration were presented as mean X± Standard Deviation (SD). Numbers and percentages were used to define categorical data. A p value <0.05 was considered statistically significant.

## 3. Results

### 3.1. Sociodemographic features

A total of 44 subjects including 22 female patients and 22 healthy subjects were included in the study. The sociodemographic features revealed no statistically significant difference between the patient and control groups for age, gender, or education ($p > 0.05$), while there was a difference regarding occupation ($p = 0.002$) (Table 1). The presence of a psychiatric disorder history in the family was statistically significantly higher in the patient group than the control group ($p = 0.004$) (Table 1).

The mean disease duration was 25.81±31.88 months in the patient group. The mean age the disorder had started was 22.13±6.59 years. The frequency of losing consciousness was less than once a month in 54.6% (n=12) and once a month or more in 45.4% (n=10) of the patients. The patient history revealed depression, panic disorder and anxiety disorder in one patient each. A stressor was defined before losing consciousness in 20 patients (90.9%) and was not present in two patients (9.1%).

### 3.2. Neuropsychological test results

The WMS-R test results revealed no statistically significant difference between the personal and actual information and orientation scores between the patient and control groups. The patient group results for mental control, instant and logical memory long term, visual recall long term score were statistically significantly lower than the control group ($p = 0.422$, $p = 0.023$, $p = 0.001$, $p = 0.0001$, $p = 0.025$ respectively). The reverse number range score was statistically significantly lower in the patient group than the control group ($p = 0.036$) (Table 2).

The patient group had statistically significantly longer durations for Stroop black/white reading, telling the rectangle color, reading color verbs and telling the colors of colored words ($p = 0.001$, $p = 0.001$, $p = 0.001$, $p = 0.018$ respectively) (Table 3).

When we looked at the sign test results of the patient group, the scanning durations for organized letters, organized figures, and random figures were statistically significantly longer in the patient group than the control group ($p = 0.006$, $p = 0.035$, $p = 0.003$ respectively) (Table 4).

### 3.3. P50 Gating results

Comparison of the P50 gating ratio between the patient and control groups revealed that P50 gating ratios were statistically significantly lower in the patient group than the control group ($p = 0.006$) (Table 5). We have presented the p50 traces of a patient with decreased sensory gating and a control group subject with normal sensory gating in Figs. 1 and 2.

Table 1
Comparison of the patient group and control group regarding sociodemographic features.

| | | Patient group (n=22) X±SD | Control group (n=22) X±SD | p |
|---|---|---|---|---|
| Age | | 24.27±7.86 | 24.59±7.98 | 0.895 |
| Years of Education | | 10.45±3.63 | 10.36±3.55 | 0.934 |
| | | n (%) | n (%) | p |
| Childhood Residence | Village | 3 (13.6%) | 1 (4.5%) | 0.494 |
| | Town | 7 (31.8%) | 6 (27.3%) | |
| | City | 12 (54.5%) | 15 (68.2%) | |
| Current Residence | Village | 1 (4.5%) | 0 (0%) | 0.534 |
| | Town | 4 (18.2%) | 3 (13.6%) | |
| | City | 17 (77.3%) | 19 (86.4%) | |
| Occupation | Housewife | 8 (36.4%) | 1 (4.5%) | **0.002** |
| | Civil Servant | 0 (0%) | 1 (4.5%) | |
| | Laborer | 0 (0%) | 8 (36.4%) | |
| | Student | 14 (63.6%) | 12 (54.5%) | |
| Marital Status | Married | 7 (31.8%) | 6 (27.3%) | 0.946 |
| | Single | 14 (63.6%) | 15 (68.2%) | |
| | Divorced | 1 (4.5%) | 1 (4.5%) | |
| Family Type | Nuclear | 18 (81.8%) | 19 (86.4%) | 0.429 |
| | Extended | 4 (18.2%) | 2 (9.1%) | |
| | Separated | 0 (0%) | 1 (4.5%) | |
| Raised by | Mother | 20 (90.9%) | 21 (95.5%) | 0.599 |
| | Grandparent | 1 (4.5%) | 1 (4.5%) | |
| | Other | 1 (4.5%) | 0 (0%) | |
| Type of attention | Excessive Attention | 2 (9.1%) | 3 (13.6%) | 0.097 |
| | Adequate Attention | 12 (54.5%) | 17 (77.3%) | |
| | No attention | 8 (36.4%) | 2 (9.1%) | |
| Type of Discipline | Excessive Control | 6 (27.35%) | 5 (22.75%) | 0.084 |
| | Adequate Control | 12 (54.55%) | 17 (77.3%) | |
| | Little Control | 4 (18.2%) | 0 (0%) | |
| Family Status as a Child | Integrated | 20 (90.9%) | 21 (95.5%) | 0.550 |
| | Separated | 2 (9.1%) | 1 (4.5%) | |
| Mother's employment status as a child | Mother working | 2 (9.1%) | 2 (9.1%) | 1.000 |
| | Mother not working | 20 (90.9%) | 20 (90.9%) | |
| Was separated from the mother | Yes | 1 (4.5%) | 1 (4.5%) | 1.000 |
| | No | 21 (95.5%) | 21 (95.5%) | |
| Sexual Trauma in Childhood | Yes | 2 (9.1%) | 1 (4.5%) | 0.550 |
| | No | 20 (90.9%) | 21 (95.5%) | |
| Physical Trauma in Childhood | Yes | 1 (4.5%) | 1 (4.5%) | 1.000 |
| | No | 21 (95.5%) | 21 (95.5%) | |
| Psychiatric Family History | Yes | 7 (31.8%) | 0 (0%) | **0.004*** |
| | No | 15 (68.2%) | 22 (100%) | |
| Psychiatric Family History (n=7) | Conversion Disorder | 4 (57.1%) | 0 (0%) | **0.004*** |
| | Anxiety Disorder | 1 (14.3%) | 0 (0%) | |
| | Panic Disorder | 1 (14.3%) | 0 (0%) | |
| | Bipolar affective disorder | 1 (14.3%) | 0 (0%) | |
| Chronic Disease in the family | Yes | 3 (13.6%) | 1 (4.5%) | 0.294 |
| | No | 19 (86.4%) | 21 (95.5%) | |

X±SD: Mean±Standard Deviation.
* Statistically significant.

There was no statistically significant relationship between the patients' age, disease duration, disease starting age, fainting frequency and gating ratio ($p=0.518$ and $r=.146$, $p=0.554$ and $r=.141$, $p=0.707$ and $r=.090$, $p=0.476$ and $r=-0.169$ respectively).

Evaluation of the patients' gating ratios together with the NPT results revealed no statistically significant relationship between the WMS-R, Stroop, check test and gating ratio scores. There was again no statistically significant relationship between WMS-R, Stroop, check test scores and gating ratios in the healthy group ($p>0.05$).

## 4. Discussion

Three groups of etiological factors listed as predisposing, onsetting and perpetuating are thought to be present in CD [4]. Our study aimed to study the predisposing factors and

Table 2
Comparison of patient and control group regarding WMS-R scores.

| | Patient (n=22) mean±SD | Control (n=22) mean±SD | p |
|---|---|---|---|
| Personal and actual findings | 4.77±0.97 | 5.22±0.75 | 0.090[a] |
| Orientation | 4.90±0.52 | 5.00±0.00 | 0.422[a] |
| Mental control | 6.86±1.16 | 7.77±1.10 | **0.023**[b]* |
| Logical memory instant | 7.47±2.54 | 9.95±1.81 | **0.001**[b]* |
| Logical memory long-term | 6.25±2.32 | 13.40±21.65 | **0.0001**[b]* |
| Forward number range | 4.40±1.68 | 4.86±1.72 | 0.381[a] |
| Reverse number range | 2.68±1.55 | 3.72±1.63 | **0.036**[a]* |
| Number range total | 7.09±2.82 | 8.59±2.97 | 0.094[a] |
| Visual recall instant | 11.13±2.51 | 12.13±1.12 | 0.096[a] |
| Visual recall long-term | 10.22±2.77 | 11.77±1.41 | **0.025**[a]* |

X±SD: Mean±Standard Deviation.
* Statistically significant.
[a] t test.
[b] Mann–Whitney U test.

our most important result was the significantly lower sensory gating and disturbed attention processes in the pseudoseizure conversion patients compared to the control group. Our results are consistent with a possible structural neurological predisposition in hysteria as stated by Charcot. The decreased p50 and disturbed NPT that measure memory and attention in pseudoseizure patients may be a sign of a kind of neurological predisposition. The decreased p50 gating in these patients independent of disease duration and fainting frequency indicates that the p50 sensory gating is disturbed not during the disease process but before it, causing a predisposition to pseudoseizures.

Sensory gating plays a role as a fundamental physiological mechanism in protecting the brain from the complexity of excessive stimuli [26]. Decreased sensory gating in pseudoseizure patients may therefore indicate excessive perception of stressful stimuli in a way that overwhelms the capacity to cope. Weak gating of sensory stimuli and the attempt to process them in higher centers will affect the attention and memory processes and therefore the working memory (WM).

The prefrontal cortex plays a central role in anticipatory attention by exerting top-down control over the selection and integration of perceptuomotor processing [27] and change of internal alertness to become prepared for the upcoming stimulus [28]. Excessive stimulation can also prevent the proper functioning of WM. This WM disorder will lead to inadequate suppression of unnecessary stimuli and dissociation will be used to try to solve the problem [29].

Table 3
Comparison of the patient group and the control group regarding the Stroop test.

| | Patient (n=22) X±SD | Control (n=22) X±SD | p |
|---|---|---|---|
| Stroop1 | 34.54±8.11 | 27.18±4.98 | **0.001*** |
| Stroop 2 | 45.72±13.72 | 34.50±5.50 | **0.001*** |
| Stroop 3 | 35.04±8.72 | 27.40±4.13 | **0.001*** |
| Stroop 4 | 84.40±25.56 | 69.31±13.41 | **0.018*** |
| Duration Difference | 49.36±19.70 | 41.90±10.87 | 0.128 |
| Number of Errors | 1.27±1.45 | 0.50±0.59 | **0.026*** |
| Spontaneous Correction | 3.63±1.67 | 2.13±1.64 | **0.005*** |

X±SD: Mean±Standard Deviation.
* Statistically significant.

This explanation is also supported by the studies of Bakvis et al. [30,31]. They state that the brain constricts the stimulus areas to be able to control excessive emotional stimulation and puts an emotional distance between the stimulus and itself by creating a dissociated state in pseudoseizure development. The authors explain the impossibility of defining a specific trigger for pseudoseizure with the change of emotionally loaded stimuli in preconscious information processes and the interpretation of nonthreatening social stimuli as dangerous as well. They have documented a baseline level of autonomic hypervigilance, and a positive attentional bias when processing social threat stimuli at a preconscious level has been documented in pseudoseizure patients [30]. Studies that show increased cognitive threat vigilance [30] and increased biological stress system activity [32] in pseudoseizure patients indicate that the stress sensitivity increases.

Although previous neurophysiological studies in patients with a diagnosis of CD have studied other auditory evoked potentials such as p300 and brain stem evoked auditory potentials, we have not come across any study on p50 gating in this group [33,34]. Köse et al. [33] compared pseudoseizure patients and other CD patients with neurological signs with a healthy control group by studying auditory evoked potentials (p300) related to information processed as a whole, attention, differentiating stimuli and comparing the content with the memory. Event-related auditory evoked potentials were obtained by using a randomized stimulus sequence and the pseudoseizure group was found to have longer P1, N1, P2, N2, and P3 latencies compared to the control group. Their study found prolonged p300, which is related to the late phase of information processing. Our study showed that there was also a problem with filtering the stimuli related to the early phase of information processing.

Cüreoğlu et al. [34] evaluated the brain stem evoked auditory potentials in their study on 37 patients with a diagnosis of CD and a control group consisting of 37 healthy patients. The study aimed to determine suboptimal brain stem lesions and the investigators found that the lower brain stem was affected in patients with a CD diagnosis. The fact that the p50 ratio was decreased in our patients indicates that the brain stem is affected as it plays a role in gating. The brain stem and temporal cortex seem to be important areas for p50 sensory gating besides the hippocampus [35], indicating that regions other than the brain stem may also be affected in CD.

P50 has mostly been studied in schizophrenia and many schizophrenic patients have been shown to have decreased p50 sensory gating. P50 studies on patient groups with schizophrenia, bipolar affective disorder, panic disorder, obsessive compulsive disorder and depression have revealed

Table 4
Comparison of the patient and control groups regarding the check test.

| | | Patient (n=22) mean±SD | Control (n=22) mean±SD | p |
|---|---|---|---|---|
| CT/Organized letters | Number of targets checked | 59.27±1.03 | 59.36±0.95 | 0.763 |
| CT/Organized letters | Number of targets skipped | 0.72±1.03 | 0.63±0.95 | 0.763 |
| CT/Organized letters | Number of targets wrongly checked | 0.00±0.00 | 0.00±0.00 | 1.000 |
| CT/Organized letters | Total number of errors | 0.72±1.03 | 0.63±0.95 | 0.763 |
| CT/Organized letters | Scanning duration | 124.09±38.44 | 98.18±17.79 | **0.006*** |
| CT/Organized figures | Number of targets checked | 59.22±1.30 | 58.90±2.28 | 0.574 |
| CT/Organized figures | Number of targets skipped | 0.77±1.30 | 1.09±2.28 | 0.574 |
| CT/Organized figures | Number of incorrectly checked targets | 0.09±0.29 | 0.13±0.46 | 0.702 |
| CT/Organized figures | Total number of errors | 0.86±1.39 | 1.13±2.37 | 0.645 |
| CT/Organized figures | Scanning Duration | 118.18±28.37 | 102.13±19.83 | **0.035*** |
| CT/Random letters | Number of targets checked | 59.54±0.85 | 59.40±0.85 | 0.600 |
| CT/Random letters | Number of targets skipped | 0.45±0.85 | 0.59±0.85 | 0.600 |
| CT/Random letters | Number of incorrectly checked targets | 0.00±0.00 | 0.00±0.00 | 1.000 |
| CT/Random letters | Total number of errors | 0.45±0.85 | 0.59±0.85 | 0.600 |
| CT/Random letters | Scanning Duration | 131.31±38.45 | 111.27±33.75 | 0.073 |
| CT/Random figures | Number of targets checked | 59.50±0.85 | 59.63±0.78 | 0.586 |
| CT/Random figures | Number of targets skipped | 0.50±0.85 | 0.36±0.78 | 0.586 |
| CT/Random figures | Number of incorrectly checked targets | 0.00±0.00 | 0.00±0.00 | 1,000 |
| CT/Random figures | Total number of errors | 0.50±0.85 | 0.36±0.78 | 0.586 |
| CT/Random figures | Scanning Duration | 118.45±31.69 | 91.13±25.86 | **0.003*** |
| CT Total | Number of targets checked | 237.54±3.40 | 237.31±3.66 | 0.832 |
| CT Total | Scanning Duration | 492.04±120.66 | 402.72±81.50 | **0.006*** |

SD: Standard Deviation, CT: Cancellation Test.
* Statistically significant.

worse gating than the control groups [16,35–41]. Gating was also subnormal in our pseudoseizure patient group as in other psychiatric disorders. Wang et al. [41] have demonstrated in their study on depressive patients that the gating decreases as the Hamilton depression score increases. The fact that we had excluded axis I diagnoses other than depression–anxiety disorders in our patients with a diagnosis of CD increases the reliability of our studies as it prevents the complicating influence of depression and anxiety on the pathology in conversion.

We did not find a statistically significant relationship between sensory gating and the disorder duration or fainting frequency in our study ($p=0.554$, $p=0.066$ respectively) indicating that the low degree of gating instead of decreased gating during the disease process may be causing a predisposition to pseudoseizures.

Another important result of our study was the low NPT performances of the pseudoseizure group when compared with the healthy control group. Other studies have compared the NPT profiles of pseudoseizure patients and epileptic patients. Our study is the first to compare the NPT profiles of pseudoseizure patients and healthy controls. Strutt et al. [42] compared the WMS NPT profile of 33 female pseudoseizure patients and 25 female left temporal lobe epilepsy patients and found that pseudoseizure patients had worse attention than the epileptic group. We also found a statistically significantly lower attention-measuring WMS-R subtest, ST and CT score in the pseudoseizure group compared to the control group. This reveals that attention is worse in pseudoseizure patients than in healthy controls. It is necessary to repeat the study in a population where these two patient groups are included together to test the possibility of using attention-measuring NPT and p50 sensory gating to discriminate between pseudoseizures and epileptic seizures.

Disorders in attention and near memory have previously been reported with CD [4]. Similar to this report, we found that the scores for WMS-R attention-measuring, reverse number range subtest and verbal instant short memory verbal and visual long term (delayed) memory scores were statistically significantly lower in the pseudoseizure patients than the healthy control group. When the pseudoseizure patients were compared with the healthy control group regarding the WMS-R scores in our study, we saw that there was no statistically significant difference in the personal and actual findings and orientation subtests in pseudoseizure patients but the instant logical memory, delayed logical memory and delayed visual recall subtest scores were low, indicating disturbed memory in CD subjects independent of the intellectual capacity. Our results indicate a decrease in the delayed visual, instant and

Table 5
Comparison of patient and control groups regarding p50 results.

| | Patient (n:22) | Control (n:22) | p |
|---|---|---|---|
| S1 OP amplitude | 1.40±1.09 | 2.11±1.90 | 0.133 |
| S2 OP amplitude | 1.25±1.19 | 0.69±0.72 | 0.068 |
| S1 O latency | 44.45±14.12 | 38.13±13.17 | 0.132 |
| S2 O latency | 47.95±13.75 | 40.86±13.24 | 0.089 |
| Gating OP | 36.21±32.09 | 63.66±29.22 | **0.006*** |

Gating OP: Sensory Gating Ratio.
* Statistically significant.

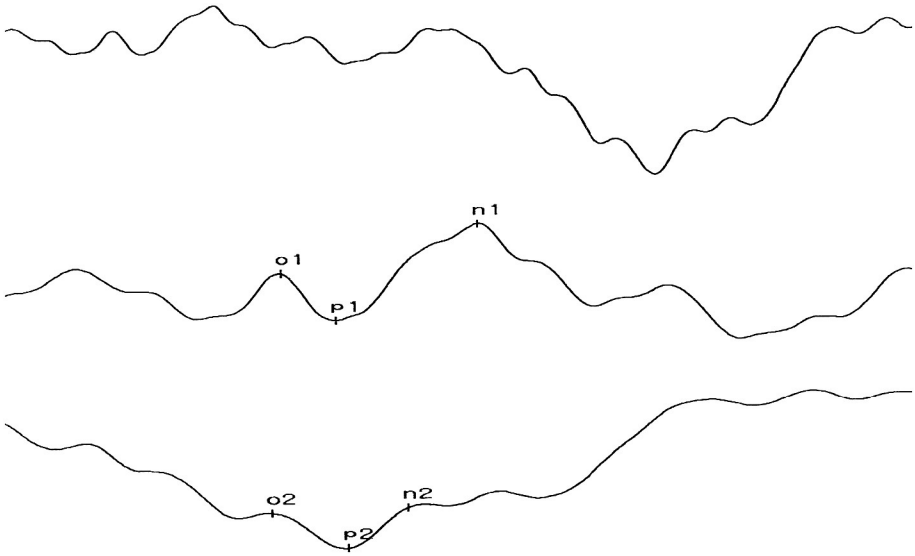


Fig. 1. The p50 trace of a patient with decreased sensory gating.

delayed verbal memories of pseudoseizure patients. There was no significant difference with the control group in instant visual recall but the difference between the pseudoseizure and control groups in the delayed visual recall indicates a problem especially in delayed memory. The statistically significantly lower mean scores for the mental control subtests that measure concentration in the pseudoseizure group compared to the control group indicate that the memory problem in these patients may be associated with attention. The WMS-R is accepted to measure the functionality of the temporal lobe and hippocampus cerebral regions [13]. The disturbed WMS-R subtests in our study indicate that the temporal lobe and hippocampus are functionally affected in pseudoseizure patients.

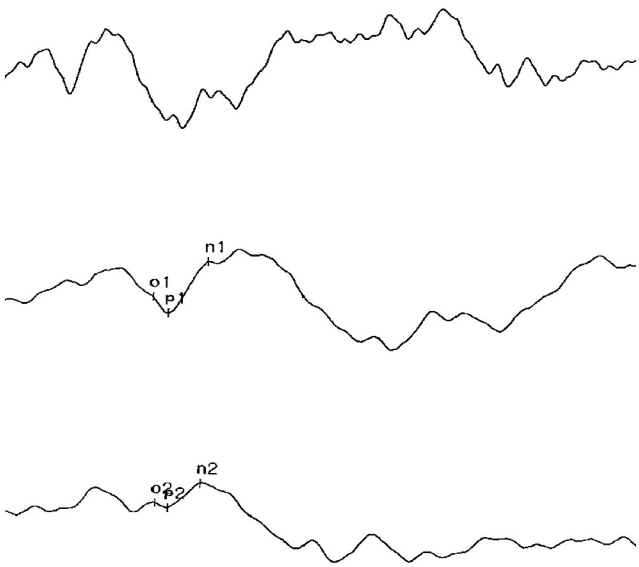


Fig. 2. The p50 trace of a normal healthy control group subject with normal sensory gating.

The Stroop mean reading duration, mean number of errors and spontaneous corrections were higher in the pseudoseizure group than the healthy group. The ST measures the ability to change the response under a disturbing influence, the information processing speed and the attention, and provides information on the individual's cognitive rigidity–flexibility degree, motor movement arrangement and difficulties in control [43,44]. We could therefore say that the pseudozeizure group had difficulties with cognitive flexibility, set changing and solving problems. Disturbances in these functions and behavior programming in general are especially a direct sign of a frontal lobe disorder [25,43]. The increased Stroop durations, number of errors and spontaneous corrections in the pseudoseizure patients compared to the control group therefore indicate that the frontal lobes are affected in these patients. We found an increased duration for Stroop 4 that measures focused attention in the patient group compared to the control group and this was consistent with information in the literature stating disturbed attention in CD.

Most of the studies on the ST have revealed that the test performance is related to the left prefrontal lobe [45]. All the patients in our study used their right hand and Stroop performance was decreased indicating that the left prefrontal lobe could have been affected.

CT is the continued attention test and measures visual scanning, response rate, visual motor speed and adaptation. It is generally a visual–spatial function and also measures sensory and motor elements and continuous attention. It is closely related to the right hemisphere and specifically the right parietal lobe [25]. The prolonged CT total scanning duration in our pseudoseizure group compared to the control group indicates a problem with continued attention in the pseudoseizure patients. Devinsky et al. [46] have reported an increased rate of right hemispheric pathology in pseudoseizure patients compared to those with epileptic seizures. The CT results, related to the right hemisphere, were also disturbed in

our pseudoseizure group compared to the control group indicating that the right hemisphere has been affected. Studies that investigate the difference between the hemispheres in this patient group are therefore needed. The longer Stroop 4 duration in the pseudoseizure patients compared to the control group indicates decreased focused attention while the longer CT scanning duration indicates decreased continued attention.

The cerebral areas responsible for the pseudoseizure development process could be the prefrontal cortex as the WMS-R and ST results were disturbed, the parietal cortex as the CT was disturbed, and the thalamus, brain stem and the prefrontal cortex as the p50 sensory gating was disturbed in our study. However, our results also support a problem in the thalamocorticofugal pathways instead of individual disturbances of the cerebral regions [12].

Another result of our study was that we did not find a relationship between NPT and p50 gating in the pseudoseizure group. Some studies in the literature report an association between the amplitude and latencies of evoked potentials components and NPT performance [40,47]. Hashimato et al. [40] found no statistically significant relationship between the p50 gating ratio and NPT results in their study on obsessive compulsive disorder patients whereas a correlation was reported for schizophrenia. We also found no relationship between p50 and NPT results in pseudoseizure patients. It can therefore be said that the gating deficit in pseudoseizure patients is due to a mechanism similar to that found in anxiety disorders. Although NPT and the p50 measuring the pre-attention period were disturbed in our patient group, the lack of correlation between these two factors indicates that these processes are disturbed independently in this patient group.

One of the limitations of our study was the fact that we only included patients with pseudoseizure CD as it is commonly encountered and is a point of interest. Similar future studies that include a larger patient group by detailing the subtypes with motor signs and sensory signs for patients with CD and that use comapping are therefore required. Another limitation of the study was that we only included females in the statistical evaluation as male cases are very rare. Although the patient and control groups were gender-matched in this study, it would be beneficial for future studies to try to include cases from both genders to be able to compare female and male patients within themselves. Another limitation of our study was that it had a regional design. The fact that gating and NPT results are similar to the anxiety spectrum of disorders, together with the presence of anxiety spectrum disorders in the family, indicates that similar mechanisms are effective in these disorders. We therefore feel it would be beneficial to repeat the study in patients with additional diagnoses of anxiety and depression.

In conclusion, this is the first study where patients with the pseudoseizure subtype of CD and control subjects were compared for p50 and NPT performances. Our results indicate that pseudoseizure patient receive pre-attention stimuli in excess followed by the WM becoming disturbed by the decreased focused and continued attention, leaving it unable to compensate and leading to a marked decrease in problem solving abilities and finally solving the problem by dissociation. These results indicate that interventions to increase gating and attention may be effective in the treatment.

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