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Emotion regulation and functional neurological symptoms: Does emotion processing convert into sensorimotor activity?

Johanna Fiess [a,*], Brigitte Rockstroh [a], Roger Schmidt [b], Astrid Steffen [a]

[a] Department of Psychology, University of Konstanz, Germany
[b] Neurological Rehabilitation Center Kliniken Schmieder, Konstanz, Germany

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ABSTRACT

Objective: Functional neurological symptoms (FNS) are hypothetically explained as a shift of emotion processing to sensorimotor deficits, but psychophysiological evidence supporting this hypothesis is scarce. The present study measured neuromagnetic and somatic sensation during emotion regulation to examine frontocortical and sensorimotor activity as signals of altered emotion processing.

Methods: Magnetoencephalographic (MEG) activity was mapped during an emotion regulation task in 20 patients with FNS and 20 healthy comparison participants (HC). Participants were instructed to (A) passively watch unpleasant or neutral pictures or (B) down-regulate their emotional response to unpleasant pictures utilizing cognitive reappraisal strategies. Group- and task-specific cortical activity was evaluated via 8–12 Hz (alpha) power modulation, while modulation of somatic sensation was measured via perception and discomfort thresholds of transcutaneous electrical nerve stimulation.

Results: Implementing emotion regulation strategies induced frontocortical alpha power modulation in HC but not in patients, who showed prominent activity modulation in sensorimotor regions. Compared to HC, discomfort threshold for transcutaneous stimulation decreased after the task in patients, who also expressed increased symptom intensity.

Conclusions: Reduced frontocortical, but enhanced sensorimotor involvement in emotion regulation efforts offers a trace to modeling a conversion of (aversive) feelings into (aversive) somatic sensations in FNS.

Introduction

Paralysis, paresis, or numbness of body parts without neurological/medical explanation are assigned to Functional Neurological Symptoms (FNS; [1,2]). FNS are seen in individuals receiving diagnoses of conversion or dissociative disorders (e.g. ICD, DSM). Various labels [1] and various hypotheses on the origin of these *medically unexplained* symptoms may reflect the still poorly understood nature of a complex psychophysiological disorder. Links between FNS and intense negative emotions have been assumed since Hippocrates and Plato (cf. [3]). They coined FNS as 'hysteria' to delineate a pathological relation between emotion and any bodily responses. Charcot attributed FNS to 'functional brain lesions' associated with 'psychic trauma'. Freud (cf. [3,4]) specified 'functional lesions' as disturbed cerebral dynamics: This, together with unconscious repression of negative experiences was described as fostering a conversion of intra-psychic conflicts into physical symptoms. Freud explained this conversion as a 'defense mechanism'. Different from conversion, Janet proposed dissociation of psychoform (i.e. cognitive) and/or somatoform (i.e. physical; cf. [3,5–7]) processes consequent upon an interplay of adverse experience(s) and individual predisposition. Redirection of emotion expression in bodily symptoms upon (emotional) stress has been emphasized again in recent models and definitions of FNS [8,9]. Current models vary in their emphasis on the impact of intense negative emotions (eventually upon trauma) in the genesis of FNS [3,5,7].

Prominent alexithymia in FNS, i.e. the inability to identify or describe one's own feelings [10,11] has been suggested as indexing altered emotion processing in patients suffering from FNS. Yet, this delineation does not inform how intense emotion 'converts' into bodily symptoms. Compromised neuronal emotion processing in patients with FNS is suggested by augmented (fronto) cortical activity [8,12] and less habituation of amygdala activity [12,13] in response to emotionally salient stimuli. In addition, impaired emotion recognition has been reported [12,14,15]. Vuilleumier [7] discussed deficient motor execution as a possible consequence of altered connectivity between (hyperactive) ventromedial prefrontal, precuneus and limbic structures, and (hypoactive) sensorimotor structures. Altered affective and sensory representations are accompanied by efficient awareness of emotional states [7,13,16–18]. Each change in emotion perception

* Corresponding author at: Department of Psychology, University of Konstanz, P.O. Box 905, D-78457 Konstanz, Germany.
E-mail addresses: johanna.fiess@uni-konstanz.de (J. Fiess), brigitte.rockstroh@uni-konstanz.de (B. Rockstroh), r.schmidt@kliniken-schmieder.de (R. Schmidt), astrid.steffen@uni-konstanz.de (A. Steffen).

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might also interfere with processes that individuals use to control "which emotions they have, when they have them, and how they experience and express these emotions", i.e., with emotion regulation ([19, p. 275, 20]; and see below).

Different theories of emotion propose a close relationship between the perception of bodily changes and emotion processing [21,22]. Perez and colleagues [23] emphasized "alterations in neurocircuits mediating emotional processing, regulation and awareness, […] and perceptual awareness" (p. 9). Regarding body sensation, patients with FNS displayed diminished accuracy of heartbeat discrimination (i.e. visceral sensitivity [24]). However, perception of such visceral signals does not capture somatic sensation such as touch or pain. More proximal to somatic sensation, transcutaneous electrical nerve stimulation (TENS) measures the sensory perception threshold and the individual discomfort threshold [25]. Nevertheless, perception of physical symptoms may be biased by body-focused attention and particular attention on illness-related information [26–28], thus individual discomfort threshold should be taken into account, when evaluating somatic sensation in patients with FNS.

The present study addressed the potential links between emotion processing, somatic sensations, and FNS by mapping electromagnetic activity during a standard emotion regulation task in participants with FNS and healthy comparison subjects (HC). Instruction-induced emotion regulation usually prompts a decrease of the late positive event-related potential (LPP) amplitude relative to the automatic response to emotionally salient stimuli [29–33]. Using high spatio-temporal resolution magnetoencephalography (MEG) Popov et al. [34] showed that a task-induced decrease in frontocortical 8–12 Hz (alpha) power, which is associated with readiness for information processing ([34], cf. [35,36]), varied systematically with both the processing of emotional salience and the down-regulation of emotional responses to aversive pictures by cognitive reappraisal. If FNS are related to altered emotion regulation, patients with FNS should show more alexithymia and less task-induced modulation of frontocortical alpha power than participants without FNS (HC). If altered emotion processing was related to an emphasis on bodily, sensorimotor processing, neuronal networks associated with sensorimotor functions should be activated in patients with FNS more than in HC. If sensorimotor activity was involved in (abnormal) emotion regulation, perception/discomfort thresholds of somatic sensation should be altered in patients with FNS compared to HC. In parallel, symptom intensity would be expected to increase temporarily in patients.

Materials and methods

Participants

The study included twenty inpatients of the local rehabilitation center (Kliniken Schmieder) with an ICD diagnosis of dissociative disorder (ICD-codes F44.4, F44.6, F44.7) and 20 healthy comparison subjects (HC). Diagnoses were given by at least two experienced psychiatrists and neurologists following standard ICD-10 guidelines. Patients were assigned to the study when they displayed at least one prominent (negative) functional neurological symptom (FNS; i.e., negative somatoform dissociative symptom, such as motor disorders or hypesthesia). Seventeen patients suffered from motor weakness on the left and 12 on the right side of body. Left-sided sensory disturbance was reported by 15 and right-sided by 11 patients with FNS. There was no difference in laterality of symptoms, i.e. between left- and right-sided motor weakness and left- and right-sided sensory disturbances, respectively. HC were recruited from the local community using flyers and oral advertising, and screened with the Mini International Neuropsychiatric Interview [37] to exclude any psychiatric disorder. For all participants, exclusion criteria were any history of a central nervous lesion or disorders (e.g. epilepsy or degenerative disorders). Groups did not differ with respect to mean age, gender distribution or years of school education (see Table 1). All participants had normal or corrected-to-normal vision. According to a standard handedness inventory, one patient with FNS and one HC were left-handed, 17 patients with FNS and 19 HC were right-handed [38].

Prior to the experimental session, participants were informed about the study design and procedures and signed written informed consent. Then, sociodemographic data were assessed together with information about the clinical status: type and severity of symptoms, alexithymia as feature of emotion processing, general psychological strain and comorbid psychopathology. Symptom severity was verified with the Somatoform Dissociation Questionnaire (SDQ-20; [39]; German Version by [40]; scores range from 20 to 100). As expected, patients with FNS experienced somatoform dissociation during the preceding twelve months more frequently than HC. In addition, patients were characterized by their general psychological strain and comorbid psychopathology using the Symptom Check List Revised (SCL-90-R; [41–43]; see Supplemental Table 1 and Supplemental Fig. 1). Comorbid psychopathology was considered relevant for data analyses if the respective symptom score exceeded 2 SD of the mean of a normative healthy group (normative data for the German version of the SCL-90-R, $N = 2141$; [44]). Characteristic emotion processing was delineated by alexithymia and assessed with the Toronto Alexithymia Scale (TAS-26; [45–47]). Patients with FNS showed a higher alexithymia score than HC, although only three patients reached the cutoff of an increased alexithymia score (≥54; [48,49]).

Procedure and materials

The study design was approved by the ethics committee of the University of Konstanz. Fig. 1 provides a schematic overview of the 3-hour experimental session, comprising the following steps:

(1) & (7) *Individual FNS intensity* was assessed using an eleven-point Likert-scale ranging from (0) 'no symptoms' to (10) 'maximum intensity'. Assumptions of normal distribution were not fulfilled, and Wilcoxon signed-rank tests were therefore used to analyze the *FNS intensity change* score (before and after the emotion regulation task).

Table 1
Socio-demographic and clinical characteristics of the study sample.

	Patients with FNS	HC	Patients with FNS vs. HC
N	20	20	
Gender (f/m)	13/7	10/10	*n. s.*
Age ($M \pm$ SD)	42.2 ± 13.6	48.9 ± 12.4	*n. s.*
Years schooling ($M \pm$ SD)	10.6 ± 3.3	11.1 ± 1.5	*n. s.*
SDQ-20 (median (*IQR*))	33.5 (*28.25–41*)	21 (*20–22.75*)	$U = 9.5, z = -5.19^{***}, r = -0.82$
TAS-26 (median (*IQR*))	48.5 (*41.5–52*)	36 (*30.25–41.5*)	$U = 65.5, z = -3.64^{***}, r = -0.58$

Note. FNS = functional neurological symptoms; HC = healthy control subjects; SDQ-20 = FNS severity verified using the Somatoform Dissociation Questionnaire; TAS-26 = Toronto Alexithymia Scale.
***: $p < .001$; IQR = interquartile range.

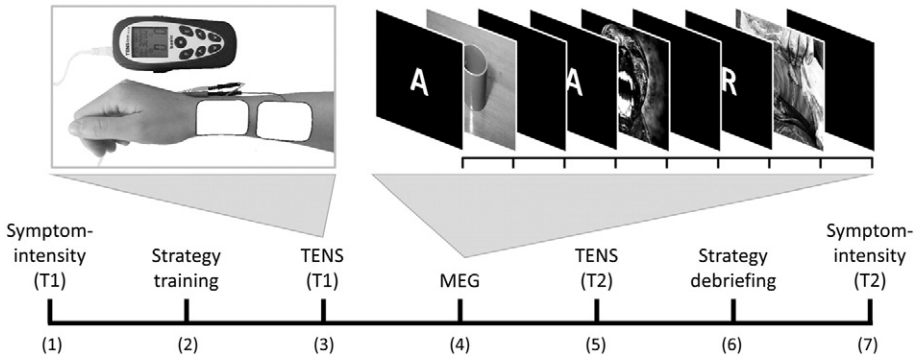

Fig. 1. Session overview: (1) Pre-task assessment of individual symptom intensity on an 11-point Likert scale, (2) instruction and training of cognitive reappraisal strategy for the later emotion regulation task, (3) pre-task assessment of somatic sensation (TENS: transcutaneous electrical nerve stimulation), (4) emotion regulation task with MEG-monitoring, (5) post-task somatic sensation assessment (TENS), (6) debriefing on emotion regulation strategies used during (4), (7) post-task symptom intensity rating.

(2) *Instruction and training*: Participants were informed that pictures with neutral of aversive content would be presented in the experimental task ((4) in Fig. 1). They were instructed that upon A (*anschauen*, *watch*) they should passively pay attention to the picture that would appear after the letter disappeared, while upon R (*regulieren*, *regulate*), they should implement their individual, trained and practiced strategies. Examples of potential strategies were given (like "This is not a real scene, this is a movie," or "The injured person is about to be rescued."), and the individually preferred strategy was inquired. Several practice runs were implemented, after which participants were asked to verbalize, which strategies they used and found efficient.

(3) & (5) *Somatic sensation* was measured by transcutaneous electrical nerve stimulation (TENS) with a frequency set to 100 Hz and pulse duration fixed at 150 μs. The 5 × 5-cm self-adhesive electrodes were placed on left forearm over the distal radial bone near the wrist joint. Stimulus intensity was increased in steps of 1 mA until the participant perceived any (tingling) sensation (*perception threshold*). Next, the stimulus intensity was increased until the participant experienced the sensation as uncomfortable (*discomfort threshold*), upon which stimulation was stopped (see [25]). For hypothesis testing, difference scores before and after the emotion regulation task were calculated for the two levels of intensity (i.e., (a) perception and (b) discomfort threshold), and then compared between groups. Assumptions of normal distribution were not fulfilled; group differences were thus analyzed by Mann–Whitney U-tests and the difference between the measurements was analyzed by Wilcoxon signed-rank tests.

(4) The *emotion regulation task* involved a stimulus set of 70 unpleasant, highly arousing and 70 neutral, less-arousing colored images from the International Affective Picture System (IAPS; [50]). All stimuli were presented via a projection system on a screen about 70 cm from the subject's eye. Across 210 trials stimulus presentation varied with three conditions, passive attending to neutral (neutral watch, NW) or unpleasant pictures (unpleasant watch, UW) and regulating emotion to unpleasant stimuli (unpleasant regulate, UR). The respective condition was indicated by an instruction cue, either the capital letter A (representing the German word for *to watch*: *anschauen*) or the capital letter R (representing the German word for *to regulate*: *regulieren*, here in the sense of *reappraise*). Conditions (NW, UW, UR) varied in pseudorandom order across trials. Each trial started with a 2-s cue, followed by 2-s picture presentation. The next trial started after an inter-trial interval (black screen) randomly varying between 2 and 2.5 s in length.

(6) At the end of the MEG-measurement, the participant was asked to report the strategies used in the emotion regulation task. Subjects most often reported to have used self-instructions/verbalization like "This is not a real scene, it is from a movie," or "The injured person will be helped." Thus, the manipulation check verified that all participants had used the cognitive reappraisal strategies trained before the experiment.

MEG data acquisition and analyses

The MEG (magnetoencephalography) was recorded using a 148-channel whole-cortex magnetometer (MAGNES 2500 WH, 4D Neuroimaging, San Diego, USA). Data were sampled at a rate of 678.17 Hz and filtered with a 0.1–200 Hz bandpass filter. The head position was monitored using five coils (nasion, inion, Cz, left and right ear canal) and by head shape using a Polhemus 3Space Fasttrack. Signals recorded by eleven MEG reference sensors were used to remove external, non-biological noise. Further data analysis was conducted using the FieldTrip open source MATLAB toolbox [51]. The continuous MEG signal was epoched in trials of 9-s length (5 s pre-stimulus) to avoid boundary effects in power spectra calculations. Five excessively noisy sensors were discarded from all participants' analyses. After rejecting trials containing movement artifacts and superconducting quantum interference device (SQUID) jumps (based on visual inspection), the strongest components corresponding to cardiac and eye movement artifacts were projected out of MEG signal using independent component analysis (ICA). Neither groups nor conditions differed in the number of trials retained ($M \pm$ SD per condition: FNS: NW: 65.25 ± 3.84, UW: 64.2 ± 3.82, UR: 64.25 ± 4.68; HC: NW: 64.05 ± 4.49, UW: 64.85 ± 3.75, UR: 64.5 ± 3.74).

Frequency analysis

An estimate of the planar gradient was calculated for each MEG sensor using a nearest-neighbor method [52]. Time-frequency representations (2–50 Hz) were obtained on Hanning-tapered time windows with a fixed window length (0.5 s), resulting in a 2 Hz frequency spectral resolution. Stimulus-evoked activity was estimated as a change of power relative to a pre-trial baseline −3 s to −2 s, expressed as percent. Changes were averaged per condition (NW, UW, UR) and per group (patients with FNS, HC).

Task-induced modulation of oscillatory activity in the time window from 0.3 s to 2 s after picture onset in the frequency band of 8–12 Hz were evaluated by cluster-based, dependent-sample *t*-test with Monte Carlo randomization ($N = 1000$) analysis [53]. This procedure controls for the type I error rate in the context of multiple comparisons. Sensor clusters were identified as differentially active when group differences (patients with FNS vs. HC) between the planned comparisons of

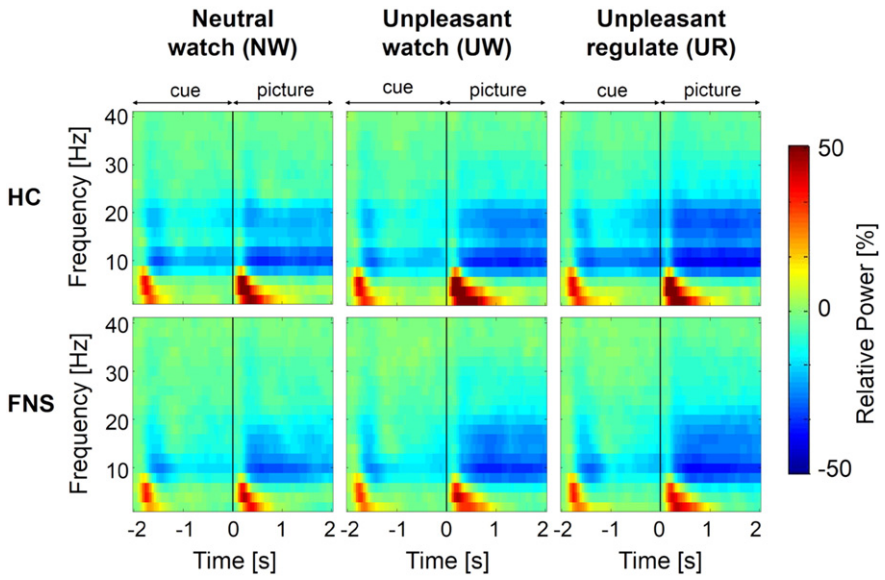

Fig. 2. Time–frequency representation (TFR) of power in the 0–40 Hz frequency range during cue (−2 s to 0 s) and picture presentation (0 s to 2 s). TFRs of power are averaged across all MEG sensors in percent change from pre-stimulus baseline (−3 s to −2 s); warm colors indicate an increase and cold colors a decrease in power relative to baseline in the respective frequency range. TFRs are presented separately for each group (HC: healthy control participants; FNS: patients with functional neurological symptoms) for the three conditions: passive watching of neutral pictures (NW), passive watching of unpleasant pictures (UW) and reappraising the emotional response while watching unpleasant pictures (UR).

conditions exceeded a 5% significance threshold. Planned comparisons targeted the emotion effect, comparing NW and UW, passive watching of unpleasant versus neutral stimuli, and the regulation effect, comparing UW and UR, passive watching versus down-regulation of unpleasant stimuli [33]. Group differences between these effects were evaluated using independent-sample *t*-test statistics. The same statistical analyses were conducted in the time window from −1 s to 0 s before stimulus onset. Here, the comparison of the response to the cues R (*regulate*) and A (*passive watch*) was defined as the preparation effect.

The *source reconstruction* of potential generators of the sensor level effects was realized using a dynamic imaging of coherent sources beamformer (DICS; [54]) after defining a time-frequency window based on sensor space data (Fig. 2). Cross-spectral density matrices of all three conditions per group were calculated using a multitaper method with a center frequency of 10 ± 2 Hz for a time period of 0.3 s to 2 s after stimulus onset. For each subject, a realistically shaped, single-shell head model [55] was computed, either based on an individual structural MRI scan (available for 6 patients with FNS and 8 HC) or based on an affine transformation of an MNI (Montreal Neurological Institute) template brain (http://www.bic.mni.mcgill.ca/brainweb). Dependent-sample *t*-test statistics were used for testing differences in source power varying with the emotion effect and the regulation effect 0.3 s to 2 s after picture onset. In addition, independent-sample *t*-tests compared the effects between groups (patients with FNS vs. HC) within the same time window. Analyses used two-sided testing with an α-significance level set to .05.

Results

Alpha power modulation during the cue interval

Time–frequency spectrograms (Fig. 2) and the time course of power (Fig. 3A) indicate a decrease in power in the 8–12 Hz (alpha) range from pre-trial baseline (−3 s to −2 s) during the 2-s cue (A or R, watch or regulate) that announced the respective task (watch or regulate). In HC, this decrease was larger upon the cue that signaled subsequent implementation of emotion regulation strategies compared to the cue that signaled passive watching (*preparation effect*; nonparametric statistical contrasts, $p < .01$). Patients with FNS did not show this preparation effect. On group level, a cluster-based nonparametric permutation test verified the larger differential cue-induced alpha-power modulation in HC compared with patients with FNS ($p < .05$, time-window −1 to 0 s before picture onset).

Alpha power modulation during the picture interval

Time–frequency spectrograms (Fig. 2) and time course of power (Fig. 3A) illustrate further alpha power decrease (8–12 Hz) 0.3 s to 2 s after picture onset relative to pre-trial baseline (−3 s to −2 s). In HC, a marginally significant emotion effect ($p = .06$) indicated a tendency of greater alpha power decrease upon unpleasant relative to neutral stimuli, while a significant regulation effect ($p < .01$) confirmed greater alpha power decrease when HC implemented emotion regulation compared to passive watching of unpleasant pictures. In contrast, a pronounced *emotion effect* of greater alpha power decrease when watching unpleasant relative to neutral pictures ($p < .01$), but no *regulation effect* was verified in patients with FNS. A cluster-based nonparametric permutation test showed group-specific differential alpha-power modulation 0.3 s to 2 s after picture onset in a central posterior sensor cluster ($p < .05$).

Source analyses verified a group-specific topography of task-induced alpha power modulation ($p < .05$; Fig. 3B): Bilateral frontocortical alpha power decrease upon down-regulation was greater in HC than in patients with FNS, whereas patients with FNS exhibited a strong regulation effect in the left central region corresponding to sensorimotor cortex. This activity pattern did not differ between patients with ($n = 6$) and without ($n = 14$) additional significant comorbid depression and/or anxiety (assessed by SCL-90-R; see Supplemental Fig. 1). The (smaller) frontocortical alpha power decrease[1] varied with (larger) FNS severity ($r = .32$, $p = .04$), and the (larger) sensorimotor alpha power decrease[2] varied with (larger) FNS severity as a trend ($r = -.29$, $p = .07$). Source activity related to the emotion effect did not differ between groups.

[1] MNI coordinates for the maximum voxel within the frontal cluster: x = −6, y = 38, z = 62.

[2] MNI coordinates for the maximum voxel within the sensorimotor cluster: x = −55, y = −10, z = 50.

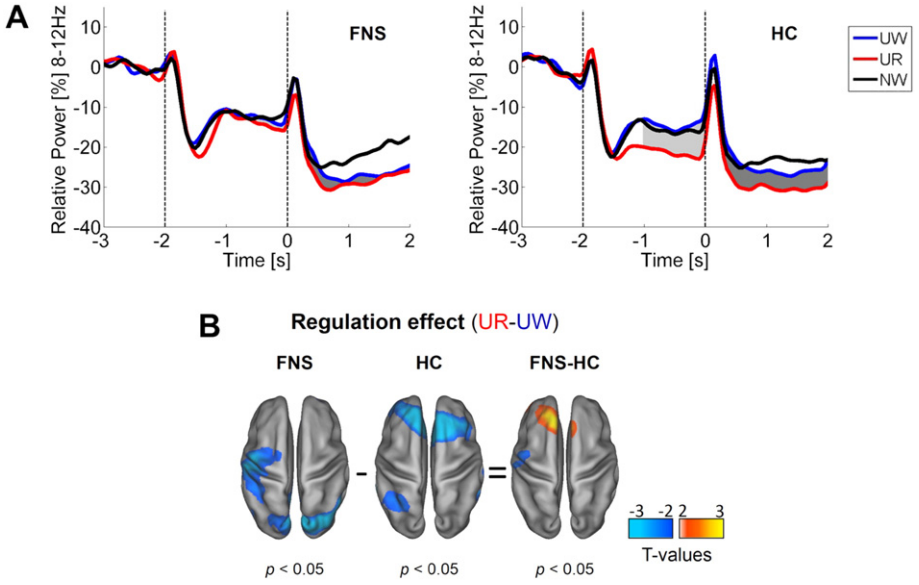

Fig. 3. A: Time course of power in the 8–12 Hz band expressed as change (in percent) from pre-stimulus baseline (−3 s to −2 s). Time courses of power changes during cue (−2 s to 0 s), and picture interval (0 s to 2 s) are averaged per group (HC: healthy control subjects; FNS: patients with functional neurological symptoms) and separately for the three conditions: passively attending to neutral (neutral watch, NW), or unpleasant pictures (unpleasant watch, UW), and regulating emotion to unpleasant stimuli (unpleasant regulate, UR). Gray-shaded areas mark the interval of power modulation by condition, i.e., the preparation effect (−1 s to 0 s; light gray) and the regulation effect (0.3 s to 2 s; dark gray). B: Source reconstruction of power for regions of statistical differences between the conditions and groups, respectively, projected onto a schematic cortical sheet (top view). Only voxels with differences at $p < .05$ significance level are presented. Left and middle: regulation effect per group (patients with FNS and HC); negative *t*-values represent larger decreases in alpha power (0.3 s to 2 s) from pre-cue baseline (−3.7 s to −2 s). Right: group differences (FNS–HC) in regulation effect; negative *t*-values represent larger differences in alpha-power decrease in regulation effect in patients with FNS than in HC, positive values represent greater differences in alpha-power decrease in regulation effect in HC than in patients with FNS.

Somatic sensation

Before the emotion regulation task, neither the transcutaneous stimulation intensity level perceived first (perception threshold) nor the rating of stimulation intensity as uncomfortable (discomfort threshold) differed between groups (perception threshold: patients with FNS: *Mdn* = 6.5, *IQR* 5–7.75; HC: *Mdn* = 5, *IQR* 5–7; discomfort threshold: patients with FNS: *Mdn* = 12, *IQR* 9–15; HC: *Mdn* = 10.5, *IQR* 9–13). After the emotion regulation, discomfort threshold in patients with FNS was lowered compared to HC ($U = 107$, $z = -2.57$, $p = .01$, $r = -0.41$).

FNS intensity change

After the emotion regulation task patients with FNS reported more intense symptoms than before (Wilcoxon signed-rank test, $Z = -3.44$, $p = .001$, $r = -0.54$).

Discussion

Can emotion processing convert into sensorimotor activity? Early models (e.g. Charcot, Janet and Freud) explained this shift of emotional to somatic sensations in FNS as conversion of intra-psychic conflicts into physical symptoms, and thus as a 'defense mechanism' [5,6,56,57]. At that time, the mechanism of this conversion was only vaguely described as "cerebral imbalance".

It is conceivable that the transfer from cognitive/emotional to somatosensory systems is manifest in an altered involvement of sensorimotor and frontocortical regions during emotion processing. The present study addressed this potential involvement by comparing oscillatory activity in a standard emotion regulation task, together with effects on peripheral somatic sensation in participants with (patients) and without (healthy comparison) FNS.

Imaging studies employing similar tasks verified that emotion regulation is associated with prefrontal and subcortical activity [34,58]. In the present study, patients with FNS and HC alike practiced cognitive reappraisal as an emotion regulation strategy, but only HC showed the frontocortical activity that usually accompanies cognitive reappraisal. Although patients with FNS exhibited an intact 'emotion effect', that is, unaffected processing of emotionally arousing stimuli, they did not show a frontocortical 'regulation effect'. This lack of frontal alpha power modulation during emotion regulation may indicate reduced cognitive control in patients with FNS compared to HC. In the present study, patients with FNS already showed less alpha power modulation than HC during the preparation interval. This suggests that patients with FNS were less efficient in preparing down-regulation of emotions, when confronted with unpleasant stimuli. Whether this indicates a cognitive deficit (getting ready to apply cognitive reappraisal strategies upon the respective cue) or the dominance of arousal by unpleasant stimuli cannot be concluded from the present data. For movement preparation, Vuilleumier ([7], referring to [17,18,59]) assumed that an altered interplay between limbic and prefrontal structures due to altered "self-relevant affective representations and memories" (p. 333) might mediate altered responses on motor tasks in patients with predominant motor FNS.

Despite the lack of frontocortical activity patients with FNS showed sensorimotor activity modulation during the present emotion regulation task — and thus no lack of emotion regulation capacity, but rather a different pattern of cortical correlates. Efforts to control upcoming feelings by cognitive reappraisal prompted the involvement of sensorimotor areas in patients with FNS in contrast to frontocortical areas in HC. This involvement of sensorimotor networks was specifically related to the down-regulation of upcoming negative feelings and not to the processing of emotionally arousing stimuli per se. This argues against a general inability of emotion regulation, and nonspecific somatic arousal or muscle tension induced by an emotional task or the experimental setting. It rather suggests an imbalance of frontocortical-sensorimotor involvement in the effort to regulate negative emotions. The sensorimotor activity during emotion regulation in patients with FNS was prominent in the left hemisphere. Since no motor response was required, this lateralized

activity cannot be attributed to motor preparation. Moreover, a hyperactivity of left-hemispheric sensorimotor regions could not be attributed to predominantly right-sided motor symptoms, as motor and sensory symptoms were equally distributed among patients and between body sides. Motor preparation (unlike motor response execution) has been reported to be intact in patients with FNS [7,16]. It cannot be concluded whether a left-hemispheric dominance of alpha power modulation can be related to the rehearsal of cognitive reappraisal strategies in patients with FNS (cf. [60]), since cognitive strategy implementation was not quantified for correlation analyses.

In the present study, self-rated alexithymia was used as additional measure of altered emotion processing in patients with FNS: Would altered emotion regulation as evident in oscillatory activity patterns in an experimental emotion regulation design be evident in a particular perception and expression of one's own emotions? Whereas alexithymia has been reported in individuals expressing FNS, somatization and/or dissociative disorders [10,11,61,62], only three patients of the present sample displayed above-threshold [48,49] alexithymia scores. Moreover, no relationships between alexithymia (TAS-26) scores, somatic discomfort threshold or oscillatory activity patterns were found in the present study. This might result from the sample selection (unusually low on alexithymia), and that the low alexithymia scores compromised the statistical verification of a relationship. Alternatively, it might indicate that self-rated alexithymia and experimentally controlled emotion regulation tackle different aspects of emotion processing: The emotion regulation task can be supposed to stimulate more cognitive effort to control emotional arousal, whereas the TAS-26 as a self-report instrument screens the ability to identify or describe feelings and the amount of externally oriented thinking.

After the emotion regulation task, the somatic discomfort threshold was lower than before task onset in patients with FNS, and patients reported increased symptom intensity. It is tempting to relate these changes in somatic sensations to the task-induced sensorimotor activity as both represent somatic activity, in an effort to down-regulate negative emotions. This activity pattern may reflect the altered emotion processing characteristic for FNS. The concerted sensorimotor and somatic activity during emotion regulation in patients with FNS strengthens the hypothesis of a link between emotion and somatic processing in medically unexplained neurological symptoms, and offers a psychophysiological delineation of a conversion of (aversive) feelings into (aversive) somatic sensations in FNS. If confirmed in further, larger samples, this would identify the psychophysiological model of conversion assumed (but not verified) in earlier models [5,6].

Alternative explanations have to be considered: Task-induced sensorimotor activity and altered somatic sensations may be considered a consequence of arousal and tension prompted by the experimental setting or comorbid psychopathology in patients. Patients with FNS have been reported to exhibit comorbid depression and anxiety (e.g., [63]). In the present study, patients expressed greater general distress than HC (Supplemental Table 1). However, the symptom severity of comorbid depression and/or anxiety did not affect the task-induced alpha power modulation. It is possible that the greater effort that patients invested in the emotion regulation task augmented individual symptom intensity, somatic sensitivity (to TENS) and sensorimotor activity.

Conclusions from the present results are limited by the small sample size and the cross-sectional design, which does not justify conclusions on the origin of altered emotion processing in patients with FNS or on the origin of the hypothesized conversion. However, the demonstrated imbalance of frontocortical and sensorimotor activity specifically related to emotion regulation in patients with FNS provides a basis for psychophysiological modeling of a conversion of (aversive) feelings into (aversive) somatic sensations in FNS, thereby substantiating a hypothesis more than a century old. Advancing our understanding of how emotion processing is linked to body sensations and symptoms should influence the adjustment of diagnostics and treatment strategies.

Supplementary data to this article can be found online at http://dx.doi.org/10.1016/j.jpsychores.2015.10.009.

Conflicts of Interest

The authors have no conflict of interest.

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