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

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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

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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.