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Functional networks of motor inhibition in conversion disorder patients and feigning subjects

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ABSTRACT

The neural correlates of motor inhibition leading to paresis in conversion disorder are not well known. The key question is whether they are different of those of normal subjects feigning the symptoms. Thirteen conversion disorder patients with hemiparesis and twelve healthy controls were investigated using functional magnetic resonance tomography under conditions of passive motor stimulation of the paretic/feigned paretic and the non-paretic hand. Healthy controls were also investigated in a non-feigning condition. During passive movement of the affected right hand conversion disorder patients exhibited activations in the bilateral triangular part of the inferior frontal gyri (IFG), with a left side dominance compared to controls in non-feigning condition. Feigning controls revealed for the same condition a weak unilateral activation in the right triangular part of IFG and an activity decrease in frontal midline areas, which couldn't be observed in patients. The results suggest that motor inhibition in conversion disorder patients is mediated by the IFG that was also involved in inhibition processes in normal subjects. The activity pattern in feigning controls resembled that of conversion disorder patients but with a clear difference in the medial prefrontal cortex. Healthy controls showed decreased activity in this region during feigning compared to non-feigning conditions suggesting a reduced sense of self-agency during feigning. Remarkably, no activity differences could be observed in medial prefrontal cortex for patients vs healthy controls in feigning or non-feigning conditions suggesting self-agency related activity in patients to be in between those of non-feigning and feigning healthy subjects.

1. Introduction

The phenomenon of conversion disorder (CD) is considered to be historically the first described psychic disease (Catonne, 1992). It can mimic every possible neurological symptom such as hyp- and dysaesthesia, visual and auditory defects, motor symptoms as flaccid or spastic-like paresis, coordination or gait disorders, tremor, loss of speech as well as amnesia, pain, fatigue or pseudo-seizures (Stone et al., 2005). In general, the patients present with neurological disease but whose signs show inconsistency and are incongruent with the normal rules of pathology. They are common in clinical practice; their deficits are disabling and can be diagnosed accurately. The mechanisms underpinning such disorders are not well understood as examination demonstrates an intact voluntary motor system which paradoxically cannot be utilized on demand and in which the symptoms are perceived as involuntary.

Even for well-defined symptoms like motor CD the neural correlates are far from being understood. Brain areas in the lateral and medial frontal cortex as well as the supplementary motor area and basal ganglia have been suggested to be involved in this condition (for review see (Bell et al., 2011)). The diversity of the employed study paradigms like motor execution (Spence et al., 2000; Stone et al., 2007; van Beilen et al., 2011), Go/Nogo (Cojan et al., 2009), implicit (de Lange et al., 2007, 2008) and explicit motor imagery (Burgmer et al., 2013; van Beilen et al., 2011) or vibratory stimulation (Burke et al., 2014; Vuilleumier et al., 2001) have provided a wide range of brain areas that could be involved in the clinical condition but did not isolate a core component. In addition the different and rather small sample sizes, the lack of control in motor imagery and motor execution

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paradigms in paretic patients as well as the heterogeneity of the included patients probably contributed to the divergent results.

The key question in this regard is whether the neural correlates of inhibition in patients with motor CD are different from those of normal subjects feigning the same symptoms. One study compared motor CD patients and feigning controls and found higher activity for intended execution in the supplementary motor area and a down regulation of the primary motor cortex contralateral in feigning controls (Stone et al., 2007). It remained however unclear whether this finding was due to motor inhibition itself or to the insufficient intention to move. Another study in two CD patients and two feigners found a down regulation of the right prefrontal cortex in feigners (Spence et al., 2000). Motor inhibition in a Go/Nogo-paradigm was also compared in feigners, controls and in one CD patient revealing higher activity in the right IFG in feigners (Cojan et al., 2009). Only one study directly compared motor CD patients, feigners and controls in a bigger sample size (van Beilen et al., 2011). This study employed motor execution und imagery and found during movement of the affected hand a complex pattern of activations including contralateral premotor cortex, anterior cingulate gyrus, superior temporal gyrus, the frontal operculum, dorsolateral frontal cortex, supramarginal gyrus and caudatus in feigners versus controls.

Importantly, no study so far trained the feigners in order to ensure a high quality of the feigning. This is an important aspect, given that the neural correlates of the feigning are to be compared to those of CD patients with a paresis. Therefore in the current approach the subjects took part to a structured training prior to the study. Before scanning two independent observers rated the quality of the feigned paresis without specifically bringing this into the attention of the subjects.

Neural activity was elicited by passive motor stimulation of the "paretic" extremity contrasted versus a rest condition. Passive movement presents a strong proprioceptive-kinaesthetic stimulus that is mostly independent of the concurrent paresis. It typically elicits activity in the sensorimotor network that is also active when the movement is voluntarily executed (Weiller et al., 1996). In this way we were able to use the same well-controlled stimulation setup that elicits robust activity in the network responsible for the control and execution of movements (Hassa et al., 2011). The hemodynamic activity elicited by passive movement of feigning subjects was compared to that of themselves in non-feigning condition and to that of motor CD patients.

2. Methods

2.1. Subjects

2.1.1. Healthy controls

The first measurement (controls in non-feigning condition, CN) of the study in 12 healthy controls was performed 3 years before the second. At that time the mean age of the controls was 39.0 ± 10.7 years with a range between 22 and 56 years. This time the mean age of the subjects was 42.5 ± 10.9 years. During this second measurement (controls in feigning condition, CF) the controls simulated a motor paresis of the right arm. None of the healthy controls had a history of neurological or psychiatric disease or neurological deficits. The controls were recruited across the staff of the rehabilitation hospital.

2.1.2. Patients

Thirteen patients (ten women, three men, with a mean age of 38.6 ± 11.0 years ranging from 21 to 51 years) with the symptom of a flaccid hemiparesis or a hemiparesis with an increase in muscle tone were included into the study. All patients were diagnosed with a conversion disorder according to DSM-IV criteria (Diagnostic and Statistical Manual of Mental Disorders, Version IV, 1994). In four patients the paresis was on the left side, in nine patients on the right side. The mean duration of symptoms was 83 weeks, with a range of 12–177 weeks (see Table 1). All patients underwent extensive neurological diagnostic procedures including MRI of brain and spinal cord, somatosensory evoked potentials, motor evoked potentials, peripheral nerve conduction examinations and EMG recordings. All diagnostic procedures did not reveal any pathological result. Patients with severe neurologic or psychiatric disorders including seizures, post-traumatic stress or panic disorder, major depression or other major affective or psychotic disorders were excluded from the study. However, patients with light forms of depression, stress and panic disorders were not excluded since they might be part of the conversion disorder. All patients were recruited in a rehabilitation hospital where they underwent rehabilitation therapy in a special psychosomatic medicine department.

A board certified neurologist inspected the structural MRI of all subjects ensuring that no subject had any structural brain damage. The Ethical Committee of the University of Constance, approved the study and all participants gave written informed consent.

2.2. Feigning training

Healthy controls trained at least trice per day in a structured video and mental imagery training to feign a right arm paresis and documented the frequency and duration of the training sessions. They were informed about the goals of the study to ensure a convincing simulation. The 6-day training was performed 6–8 days before the fMRI scan.

2.3. Evaluation of the quality of simulation

The subjects maintained the feigned right arm paresis throughout the experiment (from entering the room until the end of the fMRI data acquisition) and were observed during pre-established situations before and in preparation for the MRI. In one situation the testing was explicit (positioning of the simulated paretic arm on a ball in lying position), while in seven other situations it was implicit: (e.g. lying down on the back, grasping the questionnaire). The subjects knew about the rating of the simulation but did not know when this would happen. The rating was performed by two trained investigators and documented on an analogue scale from 1 to 5 points for each of the eight situations. After the fMRI the participants completed two questionnaires. In the first questionnaire they evaluated the training, the second focussed on their estimation of the quality of feigning and the effort to maintain it.

2.4. fMRI design

The paradigm consisted of passive movements of both wrists. Subjects/patients were placed supine on the table of the MRI scanner with their head fixed in the head-holder of the MRI headcoil and their forearms were placed on cushions in comfortable position. Participants were instructed to relax and not to interfere voluntarily with the passive movements. This was trained outside the scanner before the experiment. An investigator performed passive flexion-extension movements of the wrist of 70–90° at a fixed rate of 1 Hz for 16 s paced by a visual signal (invisible to the subjects). The rest condition (rest) was interposed between the blocks and served as baseline for analysis. The 2 conditions (right hand; left hand) were intermixed in a pseudorandomized order and interspersed with the rest condition lasting alternately 8 and 16 s. Six blocks of each condition (24 blocks) were performed in two runs with total duration of 23.5 min. To maintain alertness the subjects were asked to count the number of small red square dots that were superimposed to a face per block and report them after the run. The planed fMRI comparisons were: passive movement of the right hand vs rest and passive movement of the left hand vs rest.

2.5. MRI data acquisition

Images were acquired on a 1.5 Tesla Philips Gyroscan (Philips Medical, Hamburg). Functional T2*-weighted echo echoplanar imaging (EPI) was performed (32 axial slices of 3.1 mm thickness with 1 mm gap, FOV