Imaging Repressed Memories in Motor Conversion Disorder

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Objectives: Conversion disorders comprise neurologically unexplained symptoms with a presumed psychiatric cause, though a neuroscientific basis for this is lacking. The standard psychiatric model of conversion holds that events and processes that might explain the symptoms are, by hypothesis, either repressed or subconscious. This makes assessments based on subjective reports unreliable. We circumvent this by using a standardized method to quantify stressful life events and by assessing objectively the neural correlates of emotion processing. **Methods:** Single case study of a 37-year-old woman with clinically repressed recall and unexplained right-sided paralysis. We describe the application of the Life Events and Difficulties Schedule (LEDS) to her history, and a novel functional magnetic resonance imaging (fMRI) procedure exploring emotion processing of traumatic and control memories. **Results:** While in the scanner, cued recall of the clinically repressed event was associated with regional brain activations characteristic of emotional arousal, including the amygdala and right inferior frontal lobe, when compared with an equally severe event from the patient's past, as rated by the LEDS. Such recall was also associated with decreased motor activity in the area corresponding to the subjectively paralyzed limb. **Conclusion:** This case study provides neuroimaging evidence for a connection between traumatic events and ongoing neurological symptoms. **Key words:** conversion, dissociative, repressed, fMRI, paralysis, memory.

CT = computerized tomography; **MRI** = magnetic resonance imaging; **EEG** = Electroencephalogram; **ICD** = International Classification of Diseases; **fMRI** = functional magnetic resonance imaging; **LEDS** = Life Events & Difficulties Schedule; **mg** = milligrams; **BA** = Brodmann area; **T** = Tesla; **BOLD** = Blood Oxygen Level Dependent; **TR** = Time to repeat.

INTRODUCTION

Conversion disorder (hysteria) is a psychiatric condition in which patients present with medically unexplained neurological symptoms. It may be present in up to a third of neurology referrals, affecting predominantly women in adulthood (1). The symptoms typically occur in response to psychological stress, and are usually understood to be a way of managing the conflict or its painful affect (2), however a neuroscientific basis for the connection between stress and symptoms remains elusive (3). Recent functional magnetic resonance imaging (fMRI) studies have shed some light on the neuroanatomical basis for motor symptom maintenance (4,5), and have examined the suppression of new memories in healthy controls (6), but there have been no studies looking at the processing of the key stressful experiences in hysterical conversion. Some of the difficulty in examining the connection between events and symptoms retrospectively lies in the fact that conversion is thought to reduce the emotional salience of the stressor, through a process of repression or dissociation, such that subjective report of both the events and their relevance may be unreliable.

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We describe a patient with prototypical emotional repression and motor conversion. In addition to the usual investigations and multi-disciplinary management, we describe the use of the Life Events & Difficulties Schedule (LEDS) (7) to provide more objective ratings of her life events, and a novel use of fMRI to elucidate the processing of the emotional events relevant to her neurological symptoms.

METHODS

Case Report

A 37-year-old woman was admitted to our unit in 2005. Her index problems began in May 2004, when, a month after a suicide attempt by her daughter, her partner of 12 years informed her that he was leaving her. During the conversation she 'felt something pop in [her] head,' became 'blank,' spoke 'gibberish,' and collapsed. Her partner reported that she was stiff and unresponsive for 3 minutes, except that she blinked her eyes at his request. On recovery, she described persisting right-sided weakness and numbness. A 4-week neurology admission (and subsequent outpatient investigations) revealed normal electroencephalogram (EEG), brain computerized tomography (CT) & MRI, and routine blood tests. On examination, she had flaccid paralysis of both upper and lower limbs on the right, with loss of all sensation bisecting the trunk, but with normal reflexes. The findings on her neurological examination were otherwise normal.

In her history, she reported that she had been born 6-weeks prematurely, but had developed normally thereafter. She had a disrupted family life, being raised in various care homes from the age of 4, and suffered sexual abuse. Her father was imprisoned when she was a child and subsequent disturbance in her behavior led to her seeing a child psychologist for one month. As an adolescent, she had some contact with the justice system. She also had a history of deliberate self harm. At 27, in the context of relationship difficulties, she took an overdose of paracetamol but received no psychiatric follow-up. She held a number of unskilled jobs, including most recently as a delivery driver. Her family history is notable for epilepsy in a sibling.

She was referred to our neuropsychiatry service, where a diagnosis of mixed dissociative (conversion) disorder was made, according to ICD-10 criteria (8) and admission recommended. In the intervening period, she spontaneously recovered motor power in her leg, though her arm remained weak, with 0/5 power. She had several recurrences of her seizures, in which witnesses reported she lost consciousness, shook on the left side of her body,

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and was unable to speak for several days afterward. She commenced antidepressant treatment (amitriptyline 50 mg nocte) to help her sleep.

She was admitted to the Lishman Unit at the Maudsley (psychiatric) Hospital in August 2005. She received multi-disciplinary treatment from physiotherapy, cognitive-behavioral therapy, and psychiatry. Her amitriptyline was increased to 100 mg, for low mood, though this did not meet the criteria for a depressive episode. She also underwent the further investigation of her blood tests (including a normal post-seizure prolactin), a normal sleep EEG, and normal nerve-conduction studies (including transcranial magnetic stimulation, which evoked muscular twitches in her affected limbs by cortical stimulation). She underwent neuropsychological assessment, which found her to be functioning below her predicted premorbid level, in the average/low-average range, with a particular difficulty with abstract reasoning. She also underwent the fMRI investigation of her relevant traumatic experiences, as described below. After 6 weeks she was discharged with significant improvement in all her symptoms, though with some residual weakness in her right hand.

Life Events Assessment and fMRI Scanning

She was interviewed at length, focusing on the 3 months preceding the onset of her symptoms, to identify potentially stressful life events. These events were then rated using the LEDS (7). This is a highly reliable, validated measure that significantly overcomes the problems of recall and interviewer bias that operate when probing past events. The LEDS employs a recorded, extensive semi-structured interview to detect events, and the creation of a narrative of each life event or difficulty. These narratives are given to a panel, blind to diagnosis, which provides an external, consensus rating of severity that reflects how the 'average' person might regard the event taking account of the wider contextual circumstances. Two adverse life events were identified—her daughter's attempted suicide, and her partner's announcement that he was leaving her—both of which scored the highest rating of severity on the LEDS. Clinically, the latter event was readily identifiable as crucial to the genesis of her symptoms, both by its immediate temporal antecedence, and by the potential secondary gain that it accrued (preventing, or at least delaying, her partner's leaving). Subjectively, her report of these events was equally characteristic: she described her daughter's suicide attempt as a harrowing experience, but, in marked contrast to the severity assigned by the LEDS, claimed that her partner's announced intention was not at all distressing. Consequently, though receptive to psychological explanations, she did not feel that her partner's announcement was important—consistent with a model of emotional repression.

Based on this interview, adapting the technique of Maguire & Mummery (9), we created a set of auditory probes for use in the fMRI scanner. Twenty-four length-matched statements were recorded for each of the two adverse events, and for a nonstressful (lowest rating on the LEDS) control event from the same epoch (a weekend visit to her sister). One quarter of these were changed to make them false. For example, she recalled having to break into her daughter's room during the overdose: her statement "It was easy to kick the door down" was changed to "It was hard to kick the door down". Maguire & Mummery found that in correctly identifying the truth or falsity of the statements' subjects had to vividly recall the events.

The probes were presented auditorily to her while in a neuro-optimized 1.5T GE MRI scanner. Statements were presented in blocks of eight from each event, using a counterbalanced design to facilitate recall and minimize the overlap of affective response between events. Before each block of eight, a word identifying the next block ("weekend", "overdose" or "break-up") was presented visually for eight seconds, via a reflector system. The block of statements was then played, taking up to four seconds per statement, and she was given seven seconds after each to respond true or false with a button press of her left hand, while T_2* images were acquired (TR 3.1s; 38 × 3 mm slices, 0.3 mm gap). The total scanning time was 14 minutes 20 seconds. Images were processed using XBAM_v3.4, a fMRI analysis software package written at the Institute of Psychiatry in London. Data were first processed to minimize motion related artifacts, and then smoothed with a 7.2 mm Gaussian filter. The experimental model was fitted to the time series and a goodness-of-fit statistic computed at each voxel. The permutation of this statistic generates a null distribution under the assumption of no experimentally-determined response. The observed and permuted test statistic maps were then transformed into standard space using a two-stage warping procedure. Analyses of variance (ANOVAs) were carried out on the test-statistic maps by computing the between-condition differences at each voxel in standard space. The probability of this difference under the null hypothesis was inferred by reference to the permuted null distribution, tested at the cluster level. See http://brainmap.co.uk for full details.

RESULTS

The patient answered 83.3% of the questions correctly and reported that she had to "really put [herself] back" in the events to do so. There were no significant differences in the proportion of correct responses ($\chi^2 = 2.4$, $df = 2$, $p = .3$) or reaction times (Kruskal-Wallis, $p = .8$) between the three events.

The analysis of the fMRI scans showed a consistent pattern between conditions (at voxel and cluster thresholds of $p < .05$). The 'break-up' condition showed greater activation than both the 'neutral' and the 'overdose' conditions, in the right medial temporal lobe (Talairach coordinates 22,–15,–20, $p = .015$) extending to the amygdala (see Figure 1), the right inferior frontal lobe (Brodmann area (BA) 46), the right parietal lobe, and the cingulate gyrus/ premotor area (BA 32/6). It showed a large relative de-activation in the left primary motor cortex (BA 4), in the area corresponding to the affected right upper limb, when compared with the 'overdose' condition (Talairach coordinates –25,–4,56, $p = .003$; see Figure 2).

DISCUSSION

Analysis of the fMRI scan of a single patient with a complex paradigm must be interpreted with caution, as the risk of both type 1 and type 2 error is considerable. Nevertheless, this is the first neurophysiological examination of the recall of what was, clinically, an emotionally repressed event, compared with the recall of an equally severe event (as rated by LEDS), and a neutral event from the same epoch. It suggests that, by contrast with the subjective report of the low emotional arousal experienced, the recall of the event was highly emotionally valent (amygdala and inferior frontal activation), was associated with inhibitory/premotor activity (cingulum/premotor area), and with motor deactivation in the affected limb (primary motor cortex). Though this appears to show a link between the emotional event and the motor symptoms, the interpretation of the emotion activation is complex.

The recall of emotional events activates a network centered on the amygdalae and the right inferior frontal cortex (10). Intact amygdalae are necessary for vivid emotional recall (11), and amygdala activation, both at encoding (12) and at retrieval (13), correlates with successful recall of emotional events. The patient had no recall deficit with regards to the key event—in fact she made (nonsignificantly) fewer recall errors than with the subjectively more emotional event—but she did deny that she found the key event emotionally salient, in apparent conflict with her functional activation. One interpretation of this activation is suggested by the role of the amygdala in emotional recall, namely that the 'break-up' event was *more* emotionally salient than the 'overdose' (or the neutral event),

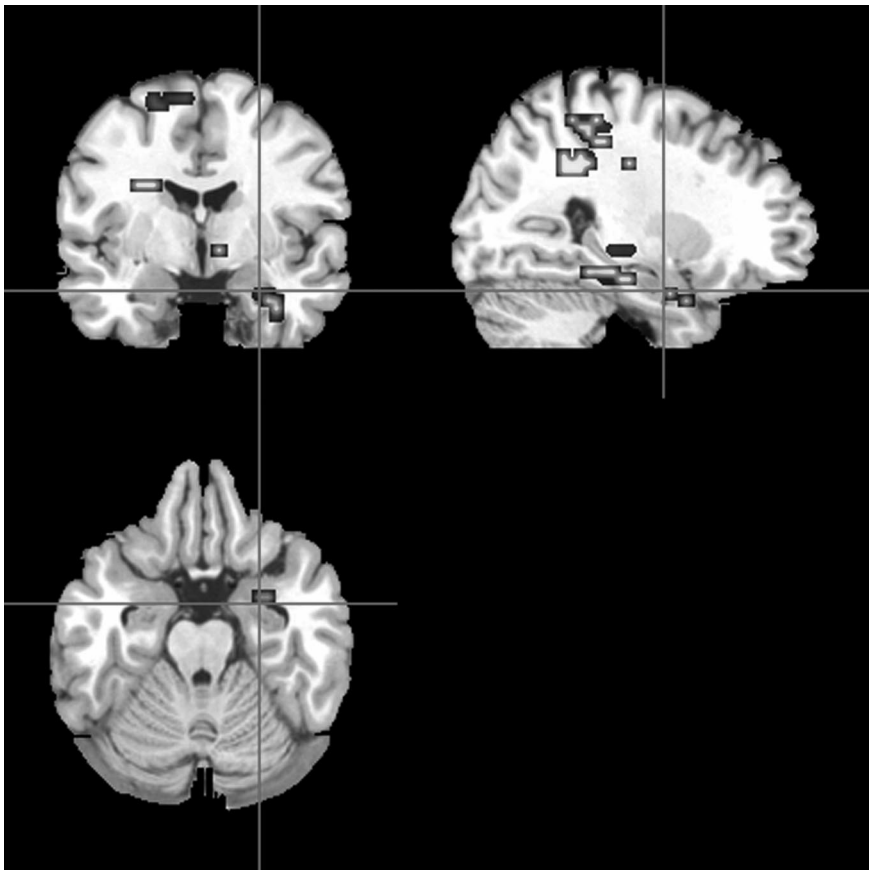

Figure 1. The Right Amygdala (crosshairs) showing greater activation during recall of the repressed event compared with recall of an equally severe event.

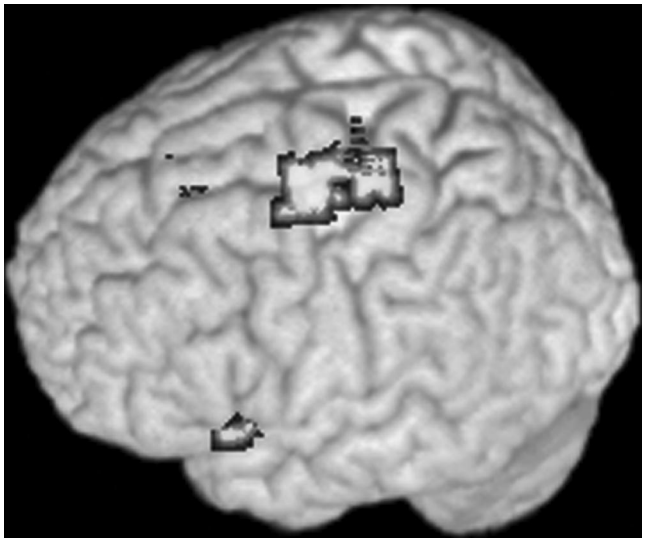

Figure 2. The Left Motor Cortex showing relative deactivation during the recall of the repressed event compared with recall of an equally severe event.

and this would concord with the dramatic symptoms the 'break-up' event appeared to produce. This would be consistent with dissociation, in the sense that the emotional experience may have been processed at a physiological level but apparently cut-off from phenomenal awareness. However, functional neuroimaging studies of dissociation, and of memory suppression, suggest these processes require prefrontal activation, presumed to reflect inhibition of memories and emotional salience as reflected in hippocampal (6) and amygdala (14) deactivation, respectively. The results can also be understood in terms of the separation of semantic from episodic memory proposed by Kihlstrom (15), and by theories of emotion which separate the labeling, based on social and cognitive cues, from the felt experience of emotion (16).

REFERENCES

1. Carson AJ, Ringbauer B, Stone J, McKenzie L, Warlow C, Sharpe M. Do medically unexplained symptoms matter? A prospective cohort study of 300 new referrals to neurology outpatient clinics. J Neurol Neurosurg Psychiatry 2000;68:207–10.
2. World Health Organization. The ICD-10 Classification of Mental and Behavioural Disorders: Clinical Descriptions and Diagnostic Guidelines. Geneva, Switzerland: WHO; 1992.
3. Broome MR. A neuroscience of hysteria? Current Opinion in Psychiatry 2004;17:465–469.
4. Spence SA, Crimlisk HL, Cope H, Ron MA, Grasby PM. Discrete neurophysiological correlates in prefrontal cortex during hysterical and feigned disorder of movement. Lancet 2000;355:1243–4.
5. Marshall JC, Halligan PW, Fink GR, Wade DT, Frackowiak RS. The functional anatomy of a hysterical paralysis. Cognition 1997;64:B1–8.
6. Anderson MC, Ochsner KN, Kuhl B, Cooper J, Robertson E, Gabrieli SW, Glover GH, Gabrieli JD. Neural systems underlying the suppression of unwanted memories. Science 2004;303:232–5.
7. Brown GW, Harris TO. The Social Origins of Depression: A Study of Psychiatric Disorder in Women. London: Tavistock; 1978.
8. WHO. The ICD-10 Classification of Mental and Behavioural Disorders. Geneva: World Health Organization; 1992.
9. Maguire EA, Mummery CJ. Differential modulation of a common

memory retrieval network revealed by positron emission tomography. Hippocampus 1999;9:54–61.
10. Dolan RJ, Lane R, Chua P, Fletcher P. Dissociable temporal lobe activations during emotional episodic memory retrieval. Neuroimage 2000; 11:203–9.
11. Cahill L, McGaugh JL. A novel demonstration of enhanced memory associated with emotional arousal. Conscious Cogn 1995;4:410–21.
12. Dolcos F, LaBar KS, Cabeza R. Interaction between the amygdala and the medial temporal lobe memory system predicts better memory for emotional events. Neuron 2004;42:855–63.
13. Dolcos F, LaBar KS, Cabeza R. Remembering one year later: role of the amygdala and the medial temporal lobe memory system in retrieving emotional memories. Proc Natl Acad Sci USA 2005;102:2626–31.
14. Lanius RA, Williamson PC, Boksman K, Densmore M, Gupta M, Neufeld RW, Gati JS, Menon RS. Brain activation during script-driven imagery induced dissociative responses in PTSD: a functional magnetic resonance imaging investigation. Biol Psychiatry 2002;52:305–11.
15. Kihlstrom JF. The cognitive unconscious. Science 1987;237:1445–52.
16. Schachter S, Singer JE. Cognitive, social, and physiological determinants of emotional state. Psychol Rev 1962;69:379–99.