**Title:**

**A novel treatment protocol (Nocebo Hypothesis Cognitive Behavioural Therapy; NH-CBT) for Functional Neurological Symptom Disorder / Conversion Disorder: A retrospective consecutive case series**

**Abstract**

**Background:** Theories concerning the aetiology of Functional Neurological Symptom Disorder (FNSD; aka Conversion Disorder) have historically inferred that psychological factors or dissociative states underlie patients’ symptoms. Current psychological models of functional neurological symptoms suggest that some type of “top-down” representations / beliefs are activated automatically (without conscious awareness), leading to symptoms. It is assumed that these representation or beliefs are similar to the idea “I am neurologically damaged”, as in our clinical experience, almost all patients have some reason to doubt the integrity of their neurological system.

**Aims:** It was hypothesized that FNSD arises from a belief of being neurologically damaged (via a mechanism akin to a nocebo response), and an interdisciplinary treatment protocol was developed consistent with this hypothesis, transparently sharing this theory with participants.

**Methods**: A retrospective consecutive case series design was utilised, measuring functional independence and symptom remission.

**Results:** Of the 13 episodes of care, 12 resulted in complete or almost complete symptom remission. Length of stay in rehabilitation was also reduced compared to previous treatment attempts.

**Conclusions:** It appears as if the treatment protocol may be very effective, and

further controlled study appears warranted.

**Introduction:**

Functional Neurological Symptom Disorder (FNSD; aka Conversion Disorder) is characterised by symptoms normally associated with neurological disorders, but with no identified structural cause. Historically, this condition has been thought to be related to repressed psychological conflicts which were ‘converted’ into physical symptoms, and/or dissociative processes.

Functional magnetic resonance imaging (fMRI) studies have suggested that there are differences between the neurological functioning of people with Conversion Disorder and ‘normal’ controls (e.g. no activation of the sensorimotor cortex during stimulation of the affected limb; Ghaffar et al, 2006), and there are also observed differences between people with Conversion Disorder and healthy ‘simulators’ (Stone *et al*, 2007). In a recent review of neuroimaging and neurophysiological studies of patients with various functional neurological symptoms, Voon et al (2016) suggested that neural networks and neurophysiologic mechanisms may mediate these symptoms.

However, no cause or mechanism has ever been firmly established, and there is no strong evidence of effective psychological treatment for Conversion Disorder (Ruddy and House, 2005; Kroenke, 2007). There have been some treatment successes described, generally resulting in modest positive benefits at best (see Carson et al, 2012, for an overview). Examples include cognitive behavioural therapy (LaFrance and Friedman, 2009), hypnotherapy (Moene et al, 2003) and in-patient physical rehabilitation, incorporating some simple behavioural modification principles (Jordbru et al, 2014).

Psychological models of functional neurological symptoms broadly speaking focus on the idea of the activation of some type of “top-down” processing being important, such as the Integrative Cognitive Model posited by Richard Brown and colleagues (Brown, 2004; Brown and Reuber, 2016a), which suggests that some type of “rogue representation” / “seizure scaffold” is automatically activated, in the context of a “high level inhibitory processing dysfunction”.

Similar to this is the account of functional symptoms described by Edwards et al (2012), which suggests that these symptoms autonomously arise from inferences based on prior beliefs about and experience of physical illness and injury. They suggested that these symptoms “can be seen as an extreme instance of a common phenomenon in predictive coding networks—the overweighting of prior beliefs over sensory data—which underlies normal experiences from optical illusions to placebo effects”.

Other notable research findings include the creation of “conversion-like” symptoms (with comparable fMRI findings) using hypnotic suggestion (Deeley *et al*, 2013), and improvement on some measures after treatment of Conversion Disorder with hypnotherapy (Moene *et al*, 2003).

Several years of clinical experience working with people with functional neurological symptoms has led to some notable observations. Firstly, there are a significant proportion of patients who did not have significant psychological trauma histories or emotional dysregulation, and this has been shown by various studies (see Brown and Reuber, 2016b). Secondly, almost all of our patients had some reason to doubt the integrity of their neurological system, either having a confirmed neurological condition (with objective evidence suggesting that this was not the cause of their functional neurological symptoms), or having perceived that they had been diagnosed with a neurological condition by an experienced medical professional, or being related to one or more people with a neurological condition (e.g. the only person in their family not diagnosed with epilepsy).

These type of observations support the idea that FNSD arises as the result of a specific suggestion or belief, analagous to the “top-down” representations outlined by Richard Brown and colleagues, and by Edwards et al (2012). One could assume that these representations / beliefs are similar to the idea ‘I am neurologically damaged’. Considering the fMRI evidence, once the representation / belief is present, it is plausible that neurological functioning changes in response to some (sometimes unidentified) initial trigger, creating functional neurological symptoms (e.g. weakness or tremor). This would be consistent with the idea of a nocebo response, defined by Colloca and Miller (2011) as “*the expectancy-induced changes in the patient’s brain-body unit”*. If present, such symptoms are almost certain to strengthen the belief in one’s neurological impairment, which would then create a maintenance (i.e. ‘vicious) cycle.

For the above hypothesis to be plausible, one has to assume that negative expectation / belief / conditioning (a nocebo response) can indeed induce changes in neurological functioning, and consequently produce functional neurological symptoms. There are numerous sources of evidence that negative (and positive) expectation produces changes in different neuroanatomical pathways, implicating different neurotransmitters (see Eknoyan *et al*, 2013, for a summary), although where negative expectations are concerned, this is more difficult to study systematically due to ethical concerns, and most of the evidence involves an increase in perceived pain (with observable neurological correlates), e.g. in response to non-harmful stimuli.

Numerous studies have demonstrated an increase in symptomatology after negative suggestion, mostly as a result of informing research participants of potential side-effects in trials of medication, where the same information is given alongside administration of a placebo. High rates of consequent adverse events have been found in the placebo arm of trials, including dry mouth, visual problems, constipation, fatigue, reported memory difficulties, and sexual dysfunction (see Colloca and Miller; 2011, for a summary).

However, clear demonstrations of the role of expectation or belief in functional neurological symptoms are sparse. As well as the aforementioned studies related to hypnosis, an interesting study by Benedetti et al (2003) showed a significant reduction in hand movement velocity in participants with Parkinson’s Disease following a ‘sham’ turning off of their deep brain stimulator (this had followed several instances of actually turning off their stimulator during the same task in preceding weeks). There are also numerous reports in the literature of rapid and/or dramatic placebo response (i.e. symptom reduction) in individuals with functional neurological symptoms, further suggesting an important role of expectations / beliefs in this population.

It is also pertinent to note that commonly, the symptoms of people with FNSD are worse when they are consciously attending to them (e.g. trying to move their affected limb, or attending to their tremor). This has parallels with the basis for Hoover’s tests (see Ziv et al, 1998), in that conscious, non-automatic movement is relatively absent in comparison to reflexive, automatic movement in people with functional neurological symptoms. This presents a challenge, but also an opportunity, for rehabilitation.

The aim of this study is to test the following hypothesis: if FNSD is the result of something akin to a nocebo effect / response, a treatment protocol consistent with this theory should be effective.

**Method:**

This study has a retrospective consecutive case series design, with no control group. It is essentially a summary of the results of a consistently applied treatment protocol in a neuro-rehabilitation setting, supported by the rehabilitation unit’s routinely used outcome measures. Whilst not specifically measured, it is estimated that the in-patient participants were treated with the customary intensity of intensity (approximately 2-4 hours of therapy a day, for 5 days a week, with nursing support available 24 hours a day).

For 17 months, every patient admitted with functional neurological symptoms (i.e. after a full neurological assessment found no structural cause for the symptoms) was included in the case series. There were no exclusions. Twelve participants (six male, six female; age range 19-63; mean = 41.2) met this criterion, with one participant presenting twice. Eleven participants were admitted as in-patients, one was seen as an out-patient (three hours total input). In terms of clinical presentation, the sample had a variety of functional symptoms. To briefly describe the sample’s predominant (i.e. most disabling) symptom, seven presented with weakness / reduced mobility, three with tremor, and two with non-epileptic seizures. Two of the sample had mixed symptomatology (e.g. tremor and weakness).

The following list outlines some of the participants’ circumstances that could be categorised as “reasons to doubt the integrity of their neurological system”:

* A confirmed neurological diagnosis (that did not explain the symptoms), including epilepsy, neurofibromatosis, an excised brain tumour as an infant, and multiple mild traumatic brain injuries.
* Significant spinal surgery in the past.
* Initial diagnosis of multiple sclerosis, with a second neurological opinion (diagnosis of functional symptoms) many months later.
* Taken to hospital in dramatic circumstances (e.g. in a helicopter), with paramedical and medical staff initially suspecting serious structural neurological impairment.
* Intellectual disability (one participant) or a diagnostic label of “borderline intellectual disability” (two participants).

The participants were treated using the following protocol.

* Medical staff explained key evidence regarding the improbability or impossibility of structural changes to the neurological system.
* A clinical psychologist gathered information about their medical history, the onset and course of symptoms, the participant’s understanding of the medical evidence, their personal belief about the causes of their symptoms, and their understanding of the terms ‘subconscious’ and ‘placebo effect’. This was useful in helping judge how much, and what degree of psychoeducation would be necessary in order to proceed. There was little or no conversation about emotional factors.
* A formulation was then transparently and collaboratively developed with the participant that incorporated relevant history within the ‘nocebo’ model (see Figure 1). In essence, this served to explain the discrepancy between the medical findings and the participant’s subjective experience. It was hoped that this would engender in the participant an alternative belief about their symptoms, to replace the one currently held (often something approximate to “the doctors have missed something”, or “this is being caused by stress”). Care was taken to minimise feelings of shame.
* The participant was consequently encouraged to examine a different perspective on their symptoms. This might commonly involve creating a new personal narrative about how events transpired around the onset of symptoms, but this time assuming that the new “nocebo model” formulation was more accurate. Whilst every individual was different, the general aim was to challenge participants’ beliefs that they were damaged, or in danger, or having a poor long-term prognosis. Instead, a narrative / metaphor such as e.g. ‘some simple misfiring of their intact neurological system’, ‘no more dangerous than a stutter’ was provided as psychoeducation, and as an alternative possibility. A ‘false alarm’ metaphor was often used, as was a hardware / software metaphor.
* The participant was then given opportunities to experience themselves as functioning better. This invariably involved physiotherapy sessions, e.g. focusing on getting their affected limbs moving again. This was often done by varying what the participant attended to (e.g. trying to get them to walk without attending to their body movement, using distraction such as music, rhythm or attentionally absorbing video games), and using video recording / feedback (with the participant’s own smartphone where possible) to show them any improvement in limb movement. Video feedback appeared essential, especially given the observation that improved mobility appeared to accompany a lack of conscious attention paid to one’s movements – the participant cannot notice their improvement in real time, as when attention was directed towards particular parts of the body, symptoms usually worsened. A treadmill was often used with those with weakness / movement difficulties, due to the frequent observation that this led to a more reliable production of improved limb movement.
* Improved functioning was framed as further evidence that the participant’s symptoms were caused by a nocebo response (e.g. ‘you have improved, yet we only changed your beliefs about the symptoms / what you attended to – we didn’t touch your legs’).
* This treatment cycle (varying attention, creating improved, more ‘automatic’ movement, giving feedback of some sort, such as video, and then reflecting on what that means about the cause of symptoms) was repeated, with more and more complex or effortful tasks. Any use of walking aids was reduced or eliminated at the earliest possible opportunity, with the attending physiotherapist often ensuring safety precautions were taken.
* For the majority of participants, an Occupational Therapist was also involved, utilising any increase in physical functioning in order to support them to return to any activity that they had not been able to perform whilst symptomatic.
* Any variability in functioning was immediately highlighted by the rehabilitation team member in attendance. For example, if the participant’s leg moved as they reached sideways for a drink bottle whilst seated (i.e. whilst not consciously focusing on moving their leg), this was framed as evidence that the leg ‘worked fine’ in certain circumstances.
* In the case of those participants who felt that their symptoms were triggered by aspects of their environment (typically those with non-epileptic seizures or tremor), graded exposure principles / treatments were incorporated. Participants would be gradually exposed to situations that they initially believed would trigger symptoms, encouraging them to look for evidence that they could tolerate the triggers or control their bodies. They were reminded of the possibility that the belief itself could be triggering the symptoms, not the situational triggers themselves.
* Once symptom elimination was achieved, participants were encouraged to push themselves to their physical limits to further prove to themselves that they were not neurologically damaged. This idea originated from the first participant in the case series who, of their own volition, decided to run up some stairs once they felt able to walk up them.

*Insert Figure 1 here*

Only one of the participants was seen as an outpatient – at the point of treatment, she experienced weakness in her legs, but only in the context of walking uphill. Treatment took the form of one 2½ hour session plus two weekly follow-up phone calls, and did not include any formal physiotherapy input – she simply walked up a hill with the psychologist at the end of the session (with significantly less subjectively experienced weakness). This participant reported symptom elimination within two weeks, maintained on follow-up.

The Functional Independence Measure (FIM) is completed on admission and discharge as part of normal practice at the rehabilitation centre. This is essentially a validated measure of disability / burden of care in rehabilitation patients (Mackintosh, 2009), comprising of 18 items (13 motor, 5 cognitive), each scoring between 1 (total assistance) and 7 (complete independence), with any item score of 5 or less indicating some level of dependence on others. The total FIM score therefore ranges from 18 to 126. In this retrospective study, it was completed for 11 of the 13 episodes of care, i.e. all except the out-patient, plus one other.

To assess relapse rate, participants were followed up between 12 and 26 months post-discharge, either by telephone, or (in the case of one participant who was not contactable) by looking at hospital records to see if they had re-presented to health services. During the telephone interviews, participants were asked if they had experienced any functional symptoms, and if so, follow-up questions regarding frequency, duration and severity were asked.

**Results:**

Of the 13 treatment episodes, 12 resulted in complete or almost complete remission of functional symptoms (i.e. fully independent). To qualify that statement further, ten episodes of care concluded with complete symptom remission, one participant was discharged with a slight limp that was probably linked to chronic pain following previous discectomy, and one self-discharged with a slight limp, but was walking normally after a week at home. The other participant dropped out of treatment, with no clear improvement.

The mean improvement in FIM scores was 28.1, achieved in an average of 14.3 days. Typically, an improvement of this magnitude reflects someone who was initially dependent to some extent on other people or aids (such as a walking frame or wheelchair) for mobility and/or personal care, but was discharged fully mobile and independent.

Using the definition of reliable and significant change as postulated by Jacobson and Truax (1991), seven of the twelve episodes of care measured by the FIM resulted in reliable and significant change, with all of the other five episodes being unable to reach significance as the admission scores were all within 20 points of a maximum FIM score (a 20 point gain being necessary for a reliable and significant change).

With regards symptomatic relapse, the average follow-up time was 17 months post-discharge. It was found that three participants (25%) had experienced no symptoms whatsoever, five (42%) had experienced either fleeting symptoms (e.g. for two days, with subsequent full remission) or clinically insignificant symptoms (e.g. a very slight twitch), three (25%) had experienced symptomatic relapse, but still had significantly improved functioning compared to first presentation (e.g. they could still walk, whereas they could not on admission), and one (8%) showed no improvement – this was the participant who dropped out of treatment.

It is interesting to note that two of the three participants who continued to experience significant symptoms had been historically labelled as having an intellectual disability or a “borderline intellectual disability”, as did the participant who needed to be readmitted for a second episode of care. One might predict this, given the likely difficulties that this subset of the sample might have with understanding concepts such as ‘nocebo effect’ and ‘subconscious’. It would therefore be difficult for them to retain understanding of the treatment’s hypothetical model, and consequently make helpful attributions in the face of any momentary relapse in symptomatology. In such instances, one might suspect that a maintenance (‘vicious’) cycle could more easily be re-established.

**Discussion**

It appears that the treatment protocol is highly effective, in that it reliably and quickly eliminated symptoms in the vast majority of participants. This treatment protocol involves a cognitive behavioural intervention, with a focus on a particular maladaptive belief (akin to “I am neurologically damaged”) with behavioural activation / exposure provided via interdisciplinary team input. The results were surprising given the current dearth of compelling evidence for treatment that leads to consistent full symptom remission in people with FNSD / Conversion Disorder. The improvements were well maintained for the majority of participants over a considerable period of time.

Aspects of the protocol have clear similarity to the “Hypothesis A / Hypothesis B” concept often used in treating health anxiety (Salkovskis & Bass, 1997), where Hypothesis A is that the person has a health condition, and Hypothesis B is that the person believes that they have a health condition. However, in the case of FNSD, one has to explain how such a belief can actually lead to symptoms, otherwise “Hypothesis B” will not be accepted by people with the condition. The success of this intervention is not proof that a nocebo-like mechanism is responsible for functional neurological symptoms, although this seems plausible. The explanatory theories put forward by Brown (2004), Brown and Reuber (2016a) or Edwards et al (2012) may have greater empirical grounding, but are not “user friendly”. Our experiences with delivering this treatment protocol suggested that the concept of a placebo effect is well known to most of the general public, and the related idea of a nocebo response therefore becomes a readily believable “Hypothesis B” when transparently shared.

Another key observation is the almost total absence of conversation about emotional factors during the treatment protocol, which did not appear to affect its success, and most likely led to substantially decreased episode of care duration. This raises questions about whether or not traditionally labelled “psychological” or “mental health” difficulties are key aspects of the aetiology of the disorder, with many researchers noting the sizeable percentage of people with functional symptoms who have no discernible or diagnosable mental health issues. Some of the theory and observations summarised by Edwards et al (2012) are supported by the results of this treatment study, i.e. certain events, associated with physical / neurological illness in self or others, lead to abnormal beliefs about illness, which can then precipitate changes in cortical hierarchies / neural networks, when then lead to symptoms. Psychological / emotional factors would appear to be often present but perhaps not necessary for causation.

When considering alternative explanations for the success of this study, one possibility is that transparent sharing of a narrative that cites a “nocebo effect” as the most likely cause of their symptoms is something that encouraged participants to engage in more vigorous physical therapy, as opposed to adopting a more passive or avoidant response to it. If this were true, then it would be likely that it was the physical therapy that was the most crucial ingredient in the treatment.

However, our unit’s previous approach to treating functional symptoms included a similar intensity of physiotherapy intervention, but with psychological input focused on reduction of emotional distress (predominantly under a generic cognitive behavioural therapy model), akin to a ‘treatment as usual’ model. Given the success of our new treatment approach, a crude comparison was made with the previous treatment approach, by reviewing the outcomes of patients admitted previously to the adoption of the treatment protocol. Although this is not a valid control group, it does illustrate the sort of outcomes previously achieved. Of 10 previous patients identified by staff, only three had complete or almost complete symptom remission (30%), the mean FIM gain was 13.7, and the mean length of stay was 19.6 days. According to this rough comparison, the new ‘nocebo’ treatment approach appears around three times better in terms of both a) rate of recovery as assessed by the FIM, and b) proportion of patients who become completely or almost completely symptom free after treatment.

Furthermore, one of the current study’s participants had been admitted to the rehabilitation unit three years previously with very similar symptoms, and had been treated using the ‘physiotherapy plus psychological treatment as usual’ approach. During that previous admission, that participant’s FIM improvement was 22 points in 35 days. Using the newer treatment approach, their FIM improvement was 31 points in 9 days. This represents a rate of FIM improvement that was 5.5 times faster than the previous approach.

Another crude comparison could be made with the findings of Jordbru et al (2014), who also used an inpatient multi-disciplinary rehabilitation approach, also used the Functional Independence Measure as an outcome measure, but appeared to use a straightforward behavioural intervention for the psychological component of their treatment (attending to good function, ignoring poor function). Their study only treated people with psychogenic gait disorder, and there were other exclusions applicable (including those with diagnosed organic neurological conditions). The mean FIM gain in their cohort was 8.4 in three weeks, compared to the 28.1 point gain in two weeks achieved by the current study, although it should be noted that the vast majority of participants in both studies achieved full functional independence by the end of the treatment period.

The use of a treadmill with those participants who had weakness, mobility or gait issues is something that might warrant further investigation, as to whether this is an important component of the treatment. Treadmill use was adopted because of the team’s perception that people with functional symptoms rarely fall over, and that maybe this is because they are able to move better in reaction to a loss of balance (i.e. a relatively intact, automatic righting reflex). The hypothesis was that a treadmill could consistently but safely take someone’s body out of a balanced, upright position, and that hopefully their relatively intact righting reflex might lead to their feet moving forwards more easily / automatically. Informal observation supported this hypothesis. However, it seems unlikely that this is the most crucial component of the overall treatment protocol, as a significant minority of the participants did not have mobility issues, yet still achieved full symptom remission.

There are numerous methodological limitations to this study, including the retrospective design, lack of control group, small sample size, lack of independent or blind assessment, as well as the inherent difficulties in diagnosing functional neurological symptoms.

**Conclusions:**

A novel treatment protocol, based on a transparently shared hypothesis that functional neurological symptoms are the result of a particular maladaptive belief (similar to “I am neurologically damaged”) acting via a nocebo-like mechanism, appeared to be highly effective in eliminating these symptoms. Maintenance of treatment effects appears good across a long follow-up period. Acknowledging the small sample and lack of control group, there are some reasons to conclude that this treatment protocol may be more effective than other treatments previously described. Given the extent of the positive outcomes (i.e. consistent complete or almost complete symptom remission), a more controlled study of the protocol appears warranted. It is also important to note that the intervention required little in the way of direct therapeutic intervention (for inpatients, sometimes as little as 10-20 hours total direct contact with the participant, spread between the different disciplines; for the outpatient, only 3 hours input).

**References**

Benedetti, F., Pollo, A., Lopiano, L., Lanotte, M., Vighetti, S. and Raniero I (2003) Conscious expectation and unconscious conditioning in analgesic, motor, and hormonal placebo/nocebo responses. Journal of Neuroscience, 23:4315– 4323.

Brown, R.J. (2004) Psychological mechanisms of medically unexplained symptoms: an integrative conceptual model. Psychological Bulletin, 130: 793-812.

Brown, R.J. and Reuber, M (2016a) Towards an integrative theory of psychogenic non- epileptic seizures (PNES). Clinical Psychology Review, 47:55-70.

Brown, R.J. and Reuber, M (2016b) Psychological and psychiatric aspects of psychogenic non-epileptic seizures (PNES): A systematic review. Clinical Psychology Review, 47:157-182.

Carson, A.J., Brown R., David, A.S., Duncan, R., Edwards, M.J., Goldstein, L.H., Grunewald, R., Howlett, S., Kanaan, R., Mellers, J., Nicholson, T.R., Reuber, M., Shrag, A., Stone, J. and Voon, V (2012) Functional (conversion) neurological symptoms: Research since the millennium. Journal of Neurology, Neurosurgery and Psychiatry, 83: 842-850.

Collocca, L. and Miller, F.G. (2011) The nocebo effect and its relevance for clinical practice. Psychosomatic Medicine, 73: 598-603.

Deeley, Q., Oakley, D.A., Toone, B., Bell, V., Walsh, E., Marquand, A.F., Giampietro, V., Brammer, M.J., Williams, S.C.R., Mehta, M.A. and Halligan, P.W. (2013) The functional anatomy of suggested limb paralysis. Cortex, 49: 411-422.

Edwards, M.J., Adams, R.A., Brown, H., Pareés, I., and Friston, K.J. (2012) A Bayesian account of ‘hysteria’. Brain, 135: 3495-3512.

Ghaffar, O., Staines, W.R. and Feinstein A (2006) Unexplained neurologic symptoms: An fMRI study of sensory conversion disorder. Neurology, 67: 2036-2038.

Jacobson, N.S. and Truax, P (1991) Clinical Significance: A statistical approach to defining meaningful change in psychotherapy research. Journal of Consulting and Clinical Psychology, 59: 12-19.

Jordbru, A.A., Smedstad, L.M., Klungsoyr, O. and Martinsen, E.W. (2014) Psychogenic gait disorder: a randomized controlled trial of physical rehabilitation with one-year follow-up. Journal of Rehabilitation Medicine, 46:181–187.

Kroenke, K. (2007) Efficacy of treatment for Somatoform Disorders: a review of randomized controlled trials. Psychosomatic Medicine, 69:881–888.

LaFrance Jr, W.C. and Friedman, J.H. (2009) Cognitive behavioural therapy for psychogenic movement disorder. Movement Disorders, 24:1856–1857.

Mackintosh, S. (2009) Functional Independence Measure. The Australian Journal of Physiotherapy, 55: 65

Moene, F.C., Spinhoven, P., Hoogduin, K.A. and van Dyck, R. (2003) A randomized controlled clinical trial of a hypnosis-based treatment for patients with conversion disorder, motor type. International Journal of Clinical Experimental Hypnotherapy, 51: 29–50.

Ruddy, R, and House, A. (2005) Psychosocial interventions for conversion disorder. Cochrane Database of Systematic Reviews, Issue 4. Art. No.: CD005331

Salkovskis, P.M. and Bass, C. (1997) Hypochondriasis. In D.M. Clark and C.G. Fairburn (Eds) Science and Practice of Cognitive Behavioural Therapy. Oxford, UK: Oxford University Press

Stone, J., Zeman, A., Simonotto, E., Meyer, M., Azuma, R., Flett, S., and Sharpe, M. (2007) fMRI in patients with motor conversion symptoms and controls with simulated weakness. Neurology, 67: 2036-2038.

Voon, V., Cavanna, A.E., Coburn, K., Sampson, S., Reeve, A., and LaFrance Jr., W.C. (2016) Functional Neuroanatomy and Neurophysiology of Functional Neurological Disorders (Conversion Disorder). Journal of Neuropsychiatry and Clinical Neurosciences, 28:168-190.

Ziv, I., Djaldetti, R., Zoldan, Y., Avraham, M., and Melamed, E. (1998) Diagnosis of “non-organic” limb paresis by a novel objective motor assessment: the quantitative Hoover’s test*.* Journal of Neurology, 245: 797-802

Acknowledgements

The authors would like to acknowledge the support of the staff team at the ISIS Rehabilitation Centre, Dunedin, New Zealand, and also Professor Graeme Hammond-Tooke for his help with this submission.

Ethical statement

The authors assert that all procedures contributing to this work comply with the ethical standards of the relevant national and institutional committees on human experimentation and with the Helsinki Declaration of 1975, and its most recent revision.

Approved by Human Ethics Committee, University of Otago, NZ – reference no: H13/070.

Fig 1: A simple hypothesized model for the creation and maintenance of functional neurological symptoms.

**Pre-disposing factors**, i.e. some persuasive reason to doubt the integrity of one’s neurological system, e.g. family history

**Belief** that one’s neurological system is vulnerable to damage, dysfunction or disease

**Trigger** – situation or bodily sensation consistent with above belief, strengthening that belief past a certain threshold

Changes in neurological functioning, creating physical symptoms

Further strengthening of belief regarding neurological damage, dysfunction or disease.